Carotid baroreflex control of heart rate and blood pressure during ES leg cycling in paraplegics

Jacqui Raymond, Glen M. Davis, Martinus N. van der Plas, Herbert Groeller, and Scott Simcox. Carotid baroreflex control of heart rate and blood pressure during ES leg cycling in paraplegics. J. Appl. Physiol. 88: 957–965, 2000.—This study investigated control of heart rate (HR) and mean arterial pressure (MAP) at rest and during electrical stimulation (ES) leg cycling exercise (LCE) in paraplegics (Para). Seven men with complete spinal lesions (T5–T11) and six able-bodied (AB) men participated in this study. Beat-to-beat changes in HR and MAP were recorded during carotid sinus perturbation. Carotid baroreflex function curves were derived at rest and during ES-LCE for Para and during voluntary cycling (Vol) for AB. From rest to ES-LCE, oxygen uptake (VO2) increased (by 0.43 l/min) and HR rose (by 11 beats/min), yet MAP remained unchanged. In AB, Vol increased VO2 (by 0.53 l/min), HR (by 22 beats/min), and MAP (by 8 mmHg). ES-LCE did not alter the carotid sinus pressure (CSP)-MAP relationship, but it displaced the CSP-HR relationship upward relative to rest. No rightward shift was observed during ES-LCE. Vol by AB produced an upward and rightward displacement of the CSP-MAP and CSP-HR relationships relative to rest. These findings suggested that the carotid sinus baroreflex was not reset during ES-LCE in Para.

IN ABLE-BODIED (AB) individuals, heart rate (HR) and blood pressure rise with increasing dynamic leg exercise intensity (23). The autonomic modulation of these alterations is mediated by central command and reflex feedback via group III and IV skeletal muscle afferent nerves (16, 25). In addition to these neural mechanisms, the arterial baroreflex may also play a role in the regulation of the cardiovascular responses during exercise. In intact dogs (30), rabbits (7), and humans (20, 22), the carotid baroreflex stimulus-response curve is “reset” to higher operating pressures than at rest during graded intensities of dynamic exercise. Thus the baroreflex contributes to elevating arterial pressure rather than operating to oppose it (25). When exercise intensity is constant, the baroreflex continuously regulates arterial pressure at its new point, acting as a negative feedback system controlling fluctuations above and below the operating point (25). The mechanism responsible for resetting the arterial baroreflex during exercise has yet to be isolated (23). However, it has been suggested to originate either as a centrally generated signal (25) or from the mechanically sensitive skeletal muscle afferent fibers (21).

Electrical stimulation (ES)-induced leg cycling exercise (LCE) performed by people with spinal cord injuries produces inconsistent HR and blood pressure responses. Whereas some studies demonstrated a decrease in HR during ES exercise (1, 3), others have observed an increase (2, 8, 11, 14) or no change (4, 29). Similarly, blood pressure either was increased during ES-LCE (1, 8) or remained unchanged from resting values (11, 14, 29).

In people with paraplegia (Para) who undertake ES-LCE, the spinal lesion interrupts the transmission of afferent neural signals from the exercising muscles. Furthermore, the nonvoluntary nature of ES-induced muscle contractions allows such exercise to be performed in the absence of central command (1, 4, 29). Therefore, this exercise paradigm abolishes the action of central command and skeletal muscle afferent feedback on the baroreflex while allowing the baroreceptors to remain operational. On the basis of this paradigm, we hypothesized that ES-LCE in Para would not elicit arterial baroreflex resetting. Therefore, the purpose of this study was to investigate the carotid baroreflex control of HR and mean arterial pressure (MAP) in Para at rest and during ES-LCE. The implications of the altered control of the cardiovascular responses during ES-LCE may assist in explaining the inconsistent HR and blood pressure responses previously observed in Para.

METHODS

Subjects. Seven men (37 ± 6 yr old) with clinically complete spinal lesions between T5 and T11 (11 ± 7 yr since injury) took part in this study. Before their participation, an independent medical practitioner examined each subject for any contraindications to exercise. The subjects also underwent carotid duplex ultrasound scans to screen for stenoses of the carotid arteries as interpreted by a cardiologist. Subjects with stenosis >20% diameter of the carotid artery or color Doppler
evidence of hemodynamically significant plaque were excluded. Six AB men of similar age (38 ± 5 yr) and physical activity levels participated in this study as a cohort group. All subjects abstained from caffeine and food intake for at least 3 h before the experiment and avoided vigorous exercise in the previous 24 h.

Physiological measurements. HR was monitored via a three-lead electrocardiogram (ECG; Portascope CR 55, Cardiac Recorders). Beat-to-beat changes in blood pressure were assessed via finger photoplethysmography (Finapres 2300, Ohmeda, Louisville, CO). The monitoring cuff was placed around the middle finger of the right hand, and the forearm and hand were supported so that the cuff was aligned at heart level. The analog signals of the ECG and blood pressure waveforms were synchronized via computer software (Labview 4.0, National Instruments, Austin, TX). The ECG was sampled at 1,000 Hz, and a value for systolic, diastolic, and mean arterial pressures was collected at each QRS complex of the ECG. Finger photoplethysmography measurements accurately track the beat-to-beat changes in blood pressure both at rest and during exercise (9). However, the absolute value for blood pressure from the Finapres may not be as accurate (9), possibly because of cutaneous vasodilation occurring with temperature regulation (19). Therefore, blood pressure was also monitored on three occasions throughout each trial via auscultation. The blood pressure recorded via the Finapres was corrected for the blood pressure determined via auscultation.

During all trials, steady-state oxygen uptake (Vo2) was averaged over 20-s intervals via open-circuit spirometry (Sensormedics 2900 Metabolic Cart, Sensormedics, Yorba Linda, CA). The mean value over 2 min of collection was taken to represent the steady-state Vo2 for each trial.

Cycle ergometry. Gel-backed surface electrodes (Empi, St. Paul, MN) were placed over the neuromuscular motor points of the gluteal, quadriceps, and hamstring muscle groups of Para subjects prior to their commencing ES-LCE (ERGYS 2, Therapeutic Alliances, Dayton, OH). During ES-LCE, monophasic rectangular pulses were delivered at a frequency of 35 Hz and pulse duration of 0.375 ms. The amplitude and timing of the stimulation were preset by a microprocessor that received feedback based on crank position and velocity. ES current output to the leg muscles was varied by the microprocessor to maintain a cycling cadence of 50 revolutions/min. The maximum current output delivered to the muscle was 140 mA, and the minimum cycling cadence maintained was 35 revolutions/min. AB subjects performed leg cycling on the same ergometer but under voluntary control at 50 revolutions/min.

Neck chamber. A Silastic neck chamber (Engineering Development Laboratory), previously described by Sprenkle et al. (28), was used to initiate carotid sinus perturbation. The chamber encased the front half of the neck, providing an airtight seal between the mandible and the clavicles and sternum. Positive and negative pressures were delivered to the carotid sinuses via a bellows system that was controlled by computer software.

The neck pressure suction procedure used in this experiment was based on that employed by Potts and co-workers (22) involving a randomly selected sequence of −70, −60, −40, −20, 0, 20, 30, and 40 mmHg pressures. The procedure involved delivering each positive or negative pressure to the carotid sinus region of the neck 50 ms after the R spike of the QRS complex. For the next 5 s, the stimulus was renewed with each succeeding R spike, to maintain the desired pressure in the face of any leakage from around the neck chamber. After 5 s of carotid sinus perturbation, the pressure returned to zero for 30 s, at which time the next pressure was selected in the sequence. To ensure that the pressures were accurately delivered, the level of neck pressure being generated was displayed on an oscilloscope (Tektronix, model TDS 420, Wilsonville, OR).

Just before the onset, and for 5 s after each level of neck pressure, the subject was instructed to hold his breath at end inspiration. This technique, used by Potts and colleagues (22), mitigated any influence of respiration on HR and MAP. All subjects reported that, at rest and during exercise, the breath-hold was not stressful and that no undue strain was involved.

Procedures. Subjects reported to the laboratory for a familiarization session and two test sessions. The familiarization session involved habituation to the procedures to be used on the subsequent test days. At this stage, two Para and three AB subjects were excluded from the study because the silastic neck chamber was unable to achieve an airtight seal.

The two test sessions followed the same procedures and were performed at the same time of day, separated by at least 48 h. Each subject was positioned on the leg cycle ergometer and instrumented for the measurement of HR and blood pressure. Just before data collection commenced, the neck chamber was applied.

Both Para and AB subjects participated in three trials on each day. The three Para trials comprised 1) rest; 2) passive leg cycling (passive), whereby an assistant turned the pedals of the cycle ergometer; and 3) ES-LCE at 0 W. The three trials were always performed in the same order with rest preceding passive so that the latter would not be influenced by prior exercise. The AB trials included 1) rest, 2) voluntary leg cycling at 18 W (Vol 18 W), and 3) voluntary leg cycling at 42 W (Vol 42 W). The two exercise trials were performed in a randomized order but were always preceded by the rest trial.

During each trial, three sequences of neck pressure and suction were delivered to the carotid sinuses of the subject. The carotid sinus perturbations were performed only when the subject had reached a steady state (within 4–5 min of the start of the trial). Sequences were separated by 3 min.

Beat-to-beat changes in HR and MAP were collected throughout each trial and stored to a personal computer for later analysis. Vo2 was measured for a 2-min period at the completion of the carotid sinus perturbations.

Data analysis. During each carotid sinus perturbation, the peak HR and MAP responses within 10 s of the onset of the stimulus were recorded (22). An example of the HR and MAP responses during carotid sinus perturbation for a typical Para subject at rest and during ES-LCE is shown in Fig. 1. The responses at each level of pressure for the three sequences performed during the trial were averaged for each subject.

The carotid sinus pressure (CSP) was calculated as MAP minus the neck chamber pressure (100% transmission of pressure to the carotid sinus was assumed). The HR or MAP response for each subject was then fitted to a sigmoidal four-parameter logistic function equation (12) by nonlinear least-squares regression

\[ HR \text{ or MAP} = A_1 \left(1 + e^{[A_2(CSP-A_3)]^{1/A_4}}\right)^{-1} + A_4 \]

where HR or MAP was the calculated variable and A1 was the response range (maximum – minimum), A2 was the gain coefficient, A3 was the centering point or the CSP at which equal increases or decreases in HR or MAP occurred, and A4 was the minimum HR or MAP response.

In addition to the four parameters derived from the nonlinear regression, other landmarks were also identified on the HR/MAP stimulus-response curves. These included the
RESULTS

Voluntary leg cycling. In AB, HR was higher during Vol 18 W (73.6 ± 4.0 beats/min) and Vol 42 W (82.8 ± 7.7 beats/min) than at rest (61.3 ± 4.2 beats/min). MAP was also higher during exercise (Vol 18 W: 95.5 ± 8.5 mmHg; Vol 42 W: 97.8 ± 8.6 mmHg) compared with at rest (89.7 ± 7.6 mmHg). VO2 was greater during Vol 18 W (0.51 ± 0.07 l/min) and Vol 42 W (0.80 ± 0.09 l/min) than at rest (0.26 ± 0.05 l/min). During Vol 42 W, HR and VO2 were higher than during Vol 18 W.

The four parameters calculated from the nonlinear regression of the CSP-HR and CSP-MAP relationships for AB are presented in Table 1. Voluntary leg cycling increased the centering point and the minimum HR or MAP response. However, the response range and the slope coefficient were not altered from rest to exercise.

Table 1. Parameters derived from stimulus-response curves for able-bodied group

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>18 W</th>
<th>42 W</th>
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<tbody>
<tr>
<td>CSP-HR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Response range</td>
<td>14.3</td>
<td>13.8</td>
<td>14.6</td>
</tr>
<tr>
<td>Slope coefficient</td>
<td>0.16</td>
<td>0.15</td>
<td>0.11</td>
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<tr>
<td>Centering point</td>
<td>84.5</td>
<td>97.5</td>
<td>102.2</td>
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<td>Minimum HR or MAP response</td>
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<td>63.7</td>
<td>73.3</td>
</tr>
<tr>
<td>Maximal gain</td>
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<td>-0.34</td>
<td>-0.31</td>
</tr>
<tr>
<td>r²</td>
<td>0.95</td>
<td>0.95</td>
<td>0.95</td>
</tr>
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<tr>
<td>CSP-MAP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Response range</td>
<td>15.2</td>
<td>15.8</td>
<td>17.8</td>
</tr>
<tr>
<td>Slope coefficient</td>
<td>0.18</td>
<td>0.26</td>
<td>0.15</td>
</tr>
<tr>
<td>Centering point</td>
<td>82.9</td>
<td>97.1</td>
<td>100.6</td>
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<tr>
<td>Minimum HR or MAP response</td>
<td>80.7</td>
<td>84.0</td>
<td>88.5</td>
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<tr>
<td>Maximal gain</td>
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<td>-0.45</td>
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<tr>
<td>r²</td>
<td>0.94</td>
<td>0.97</td>
<td>0.94</td>
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Values are means ± SD and median (interquartile range) for maximal gain. r², r-Squared of the stimulus-response curve fitted to the carotid sinus pressure (CSP) and heart rate (HR) response of each subject. MAP, mean arterial pressure; Vol, voluntary leg cycling. *Significantly different from rest (P < 0.05).
in AB. There was also no significant difference in maximal gain of the CSP-HR and CSP-MAP stimulus-response curves among the three trials.

Voluntary leg cycling elicited an upward and rightward shift of the CSP-HR and CSP-MAP relationships relative to rest (Fig. 2). Although the AB subjects performed two levels of exercise in this study, clear evidence for resetting of the carotid sinus baroreflex was only present at Vol 42 W. Figure 3 illustrates the sigmoidal curves fitted to the mean CSP, HR, and MAP at rest and Vol 42 W. The four landmarks used as criteria of carotid baroreflex resetting are also shown on the curves. For both the CSP-HR and CSP-MAP relationships, the centering and operating points occurred at significantly higher levels of CSP during Vol 42 W than at rest. There was also a significant change in the threshold and saturation points for the CSP-HR and CSP-MAP relationships, respectively.

Passive leg cycling. In Para, passive decreased HR (69.3 ± 11.4 beats/min at rest vs. 62.7 ± 9.7 beats/min at passive) relative to rest, but did not alter MAP (91.8 ± 6.2 mmHg at rest vs. 92.7 ± 7.0 mmHg at passive) or VO2 (0.22 ± 0.02 l/min at rest vs. 0.24 ± 0.03 l/min at passive). There was no change in the four parameters calculated from the nonlinear regression of either relationship except for an increase in the CSP at the centering point and a decrease in the minimum response for the CSP-HR relationship (Table 2). The maximal gain of the stimulus-response curves was also unchanged from rest. Passive led to a downward displacement of the CSP-HR relationship but did not produce any alteration in the CSP-MAP relationship compared with rest (Fig. 4).

ES-induced leg cycling. VO2 (0.65 ± 0.15 l/min) and HR (80.1 ± 11.1 beats/min) were higher during ES-LCE than at rest or during passive. However, MAP (92.0 ± 7.8 mmHg) was not altered during ES-LCE from its resting value.

Compared with results obtained at rest, ES-LCE did not elicit any change in either maximal gain or the four nonlinear regression parameters for either the CSP-HR...
or the CSP-MAP relationships (Table 2). The CSP-HR relationship was displaced upward relative to rest; however, the CSP-MAP relationship was not altered (Fig. 4). Figure 5 presents the sigmoidal curves fitted to the mean CSP, HR, and MAP at rest and during ES-LCE. The threshold, saturation, centering, and operating points are also displayed. ES-LCE did not alter the CSP at which these points occurred for either the CSP-HR or CSP-MAP relationships. With the use of the same criteria for baroreflex resetting that were applied to AB subjects, these data indicated that ES-LCE did not elicit carotid baroreflex resetting in Para subjects.

Para vs. AB differences. Significant interaction (trial-by-group) effects were observed for steady-state MAP and the centering, operating, and saturation points of the CSP-MAP relationship. For the CSP-HR relationship, only the centering point demonstrated a significant interaction effect. The operating, threshold, and saturation points approached statistical significance for a between-group interaction effect (P = 0.056, P = 0.059, P = 0.079, respectively).

DISCUSSION

Carotid baroreflex control during voluntary leg cycling. The importance of the carotid baroreflex in regulating blood pressure during exercise has been well established in animals (7, 30) and humans (20, 22). Evidence that the baroreflex is reset to operate around a higher CSP implies that this reflex contributes to an increase in blood pressure during exercise rather than opposes it. The results of AB subjects performing leg cycling in the present study supported the concept of carotid sinus baroreflex resetting during voluntary exercise. With an increase in V\textsubscript{O\textsuperscript{2}}, the carotid baroreflex reset to a higher sinus distending pressure as demonstrated by the increase in CSP at which the centering, operating, threshold, and saturation points occurred (Fig. 3).

The observations from AB data in this study agreed with previous investigations (20, 22). Potts and colleagues (22) observed increases in threshold, saturation, and centering point pressures of the carotid-cardiac (CSP-HR) and carotid-vasomotor (CSP-MAP) function curves from rest to exercise at power outputs eliciting 25% and 50% peak V\textsubscript{O\textsuperscript{2}} (V\textsubscript{O\textsuperscript{2}\textsubscript{peak}}). Similarly, Papelier and associates (20) observed a progressive rightward and upward displacement of the CSP-HR and CSP-MAP function curves during leg cycling power outputs ranging from 60 to 240 W. In both studies, the gain of the carotid sinus baroreflex remained unchanged from resting values, suggesting that, during

| Table 2. Parameters derived from stimulus-response curves for paraplegic group |
|-----------------------------------|--------|-------------|
| Parameter                        | Rest   | Passive     | ES-LCE 0 W |
| CSP-HR                           |        |             |            |
| Response range                   | 12.8 ± 4.0 | 14.1 ± 7.6 | 15.2 ± 3.0 |
| Slope coefficient                | 0.13 ± 0.11 | 0.09 ± 0.08 | 0.11 ± 0.04 |
| Centering point                  | 90.4 ± 12.4 | 101.4 ± 12.3 | 93.0 ± 8.2 |
| Maximum HR or MAP response       | 61.8 ± 15.7 | 53.1 ± 7.4 | 70.5 ± 13.6 |
| Maximal gain                     | −0.27 (0.46) | −0.19 (0.15) | −0.38 (0.30) |
| r\textsuperscript{2}             | 0.95 ± 0.03 | 0.96 ± 0.02 | 0.95 ± 0.03 |
| CSP-MAP                          |        |             |            |
| Response range                   | 15.5 ± 2.9 | 13.3 ± 3.0 | 11.3 ± 4.1 |
| Slope coefficient                | 0.10 ± 0.06 | 0.33 ± 0.48 | 0.20 ± 0.10 |
| Centering point                  | 85.9 ± 7.5 | 96.0 ± 17.1 | 95.0 ± 16.2 |
| Maximum HR or MAP response       | 84.0 ± 6.4 | 85.0 ± 3.9 | 86.0 ± 6.5 |
| Maximal gain                     | −0.39 (0.35) | −0.39 (0.71) | −0.43 (0.26) |
| r\textsuperscript{2}             | 0.95 ± 0.02 | 0.95 ± 0.04 | 0.87 ± 0.10 |

Values are means ± SD and median (interquartile range) for maximal gain. *Significant difference between passive and electrical stimulation leg cycling (ES-LCE) (P < 0.05); †Significantly different from rest (P < 0.05).
exercise, the baroreflex maintained appropriate sensitivity to control fluctuations in blood pressure. In the present study, although the workloads performed by AB were relatively low, there was clear evidence of resetting of the CSP-HR and CSP-MAP relationships during Vol 42 W (Fig. 3). During exercise, the CSPs at the operating, centering, threshold, and saturation points were higher than at rest. The shift of these points is consistent with the notion that the entire reflex has been centrally modified to operate around a new prevailing blood pressure (24).

Carotid baroreflex control during ES-LCE. ES-LCE performed by the Para group did not displace the CSP-HR or CSP-MAP stimulus-response relationships rightward (Fig. 5), despite an increase of $V_{O_2}$ similar in magnitude to their AB cohorts. However, similar to results in AB, baroreflex sensitivity was unchanged in Para during exercise. These observations suggested that the carotid baroreceptor reflex was not reset during ES-LCE but remained functional near the resting level. In one Para subject who could perform steady-state ES-LCE at 6 W ($HR = 97$ beats/min), the higher power output did not elicit a rightward shift of the stimulus-response curves relative to rest or ES-LCE at 0 W (Fig. 6). This was in contrast to the AB group of the present study and to earlier reports (20, 22), whereby increasing exercise intensities led to progressive rightward displacement of the CSP-HR and CSP-MAP relationships.

The precise stimulus initiating carotid baroreflex resetting is unknown; however, both central command and skeletal muscle afferent neural feedback have been implicated (21, 25). The lack of resetting in Para is consistent with the evidence that signals arising from central command and/or skeletal muscle afferent feedback alter the reflex’s interpretation of afferent information from the baroreceptors. In Para with sensorimotor complete injuries, the spinal lesion interrupts the ascending and descending neural traffic. Therefore, in
the absence of this neural traffic, and without central command, there was no stimulus for carotid baroreceptor resetting during ES-LCE and the reflex thus remained operational near resting levels.

Mechanisms for cardioacceleration in Para. In the absence of central command or skeletal muscle afferent feedback, our observations suggest that nonneural mechanism(s) mediate cardioacceleration during ES-LCE in Para. Although it is unclear precisely what this mechanism may be, Kjær and co-workers (14) have suggested that humoral feedback may be an important stimulus for cardioacceleration in the absence of central command and skeletal muscle afferent feedback.

A second characteristic of the mechanism contributing to cardioacceleration may be that it is time dependent. Although HR responses during ES-induced leg exercise have been inconsistent, those studies that have detected cardioacceleration (2, 8, 11, 14, and the present study) noted it after at least 10 min of exercise. Shorter exercise trials were used in those investigations that observed no change (4, 29) or a decrease (1, 3) in HR. The apparent time course of cardioacceleration is consistent with the hypothesis of a blood-borne stimulus, whereby this type of mechanism might have a minimal immediate effect but would accumulate with time or intensity of exercise.

On the basis of the findings of the present investigation and those from previous studies (3, 14), it is possible to reflect on why there has been inconsistency in the HR response during ES-induced exercise in Para. Inconsistent observations may be explained by the absence of arterial baroreflex resetting coincident with nonneural mechanism(s) mediating cardioacceleration during ES-LCE. We suggest that, at the onset of ES-induced exercise blood pressure increases, and, in the absence of arterial baroreflex resetting, this increase is interpreted as hypertension. In turn, this elicits a reflex decrease in HR. Notably, Brice and colleagues (3) observed an increase in arterial blood pressure immediately followed by a decrease in HR in the transition from rest to ES-induced leg exercise and again in the transition to a higher work rate. As ES-induced leg exercise progresses, we suggest that HR rises further not via increased central command, afferent neural activity, or via the baroreflex (because steady-state MAP remains unchanged) but rather is mediated by some nonneural mechanism(s) that accumulates over time.

Another possible factor that may contribute to cardioacceleration in Para subjects performing ES-LCE is an increase in blood temperature. During a 30-min ES-LCE trial, Hjeltnes and co-workers (10) demonstrated increases in rectal temperature after 12 min of cycling in subjects with tetraplegia. However, whether the rise in temperature contributed to cardioacceleration in their experiment was unclear, as the HR had plateaued by 9 min in all but one subject.

MAP during ES-LCE. MAP did not increase above rest during ES-LCE. This observation is consistent with previous studies of Para subjects undertaking ES-induced leg exercise (1, 11, 14, 29). Moreover, the absence of a pressor response supports the viewpoint that arterial pressure is elevated during exercise via reflex changes arising from stimulation of the chemosensitive muscle afferents in the active muscle (16, 25).

Kjær and co-workers (13) also found that MAP remained unchanged from rest during ES-LCE in AB subjects whose legs were paralyzed following epidural anesthesia at L3–L4. Conversely, when the same subjects performed voluntary leg cycling at an equivalent VO2, MAP rose from 93 ± 4 mmHg at rest to 119 ± 4 mmHg during exercise. Further evidence for the pressor reflex being mediated by afferent feedback from the exercising muscles can be derived from experiments on AB subjects performing ES-induced leg exercise (1, 29). Thomas and colleagues (29) demonstrated a 39- to 44-mmHg increase in systolic blood pressure in AB subjects undertaking ES-induced knee extension. This increase in blood pressure was attributed to activation of the muscle afferents based on the assumption that the muscle contractions were elicited without an increase in central command.

Carotid baroreflex control during passive. Passive was included in this study to determine whether limb movements in the absence of muscle contractions might alter HR or MAP in Para individuals. Such “sham exercise” was employed to delineate between the effects of leg cycling motion vs. dynamic ES-induced muscle contractions and also to eliminate the effect of knowledge of leg movements. The lack of increase in HR during passive suggested that the higher HR observed during ES-LCE was entirely due to neuromuscular recruitment.

Interestingly, passive elicited a decrease in HR of 7 beats/min. This decrease is difficult to interpret on the basis of the measurements made in this study; however, we can conclude that it was not a baroreflex response, as MAP was unaltered. Muraki and colleagues (17) noted that, in Para subjects, an increase in stroke volume occurred during passive without a concurrent change in HR. This may suggest that the decrease in HR observed in our study was due to an increase in stroke volume. However, unpublished results from our laboratory demonstrate that passive does not elevate stroke volume yet still reduces HR. Similar observations have been made in people with quadriplegia (18). Therefore, the mechanism explaining the decrease in HR during passive has yet to be elucidated.

Potential limitations of the study. The inherent limitations of the neck pressure/suction method have been detailed elsewhere (20, 22). Briefly, these limitations include 1) the possibility that the reflex HR and MAP responses are affected by the extracarotid baroreceptors (22) and 2) the incomplete transmission of positive and negative pressures to the carotid sinuses (15). In addition to these factors, it is possible that performing an end-expiratory breath-hold technique alters chemoreceptor activity or provokes a Valsalva maneuver to influence HR and MAP during the time of carotid sinus perturbation.

For all subjects, the peak change in HR generally occurred within 5 s following either neck pressure or
neck suction (e.g., Fig. 1). The peak change in MAP typically occurred after the carotid sinus perturbation had finished, representing the contribution of the slower, sympathetically mediated changes in vascular resistance. However, the recording of responses after the 5-s perturbation may allow for increased counter effects from the extracarotid baroreceptors or perhaps for a reduction in the magnitude of the response because the stimulus was no longer being applied. However, the gains of our stimulus-response curves were similar to those studies that employed longer carotid sinus perturbation times. Papelier and colleagues (22) demonstrated significant baroreflex resetting at a similar VO\textsubscript{2} to the Para groups. In addition, as resetting appears to be a function of exercise intensity (20), it could be argued that, with a higher sinus pressure during ES-LCE in Para subjects, the lack of carotid sinus baroreflex was not reset to operate around a similar CSP. The lack of carotid sinus baroreflex was not reset to operate around a similar CSP. Unfortunately, in this study, VO\textsubscript{2}peak was not measured in either the Para or AB groups, precluding us from expressing leg cycling workloads as a relative intensity of effort.

In conclusion, this study demonstrated that the baroreflex was not reset to operate around a higher sinus pressure during ES-LCE in Para subjects. Instead, the carotid baroreflex responded to fluctuations in blood pressure with similar sensitivity to rest and around a similar CSP. The lack of carotid baroreflex resetting may assist in explaining the inconsistent HR responses previously observed in these individuals during ES-induced leg exercise. Furthermore, the lack of resetting in subjects with complete paraplegia confirmed the importance of central command and/or skeletal muscle afferent feedback in modifying baroreflex control of cardiovascular responses during voluntary dynamic exercise, as both these neural pathways were absent during ES-LCE. A secondary conclusion of this study was that the increase in HR observed during ES-LCE could be the result of a nonneural mechanism mediating cardioacceleration in Para. Finally, this study demonstrated the potential of the spinal cord-injured individual to provide a human model of cardiovascular control during exercise.

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