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Acute hemodynamic responses of spinal cord injured individuals to functional neuromuscular stimulation-induced knee extension exercise

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Abstract—The purpose of this study was to determine and compare acute hemodynamic responses of spinal cord injured (SCI) quadriplegics (quads), and paraplegics (paras) during a graded-intensity knee extension (KE) exercise test utilizing functional neuromuscular stimulation (FNS) of paralyzed quadriceps muscles. Seven quads and seven paras (N=14) performed a series of 4-minute stages of bilateral alternating FNS-KE exercise (approximately zero to 70 degree range of motion at the knee and 6 KE/min/leg) at ankle loads of 0, 5, 10, and 15 kg/leg. Physiologic responses were determined with open-circuit spirometry, impedance cardiography, and auscultation. Comparing rest with peak FNS-KE for both groups combined, FNS-KE exercise elicited significant ($p<0.05$) increases in oxygen uptake (130 percent), pulmonary ventilation (120 percent), respiratory exchange ratio (37 percent), arteriovenous oxygen difference (57 percent), cardiac output (32 percent), stroke volume (41 percent), mean arterial pressure (18 percent), and rate-pressure product (23 percent). Heart rate increased significantly by 11 percent from the 5- to the 15-kg/leg stages. Physiologic responses of quads and paras were very similar, except for lower ($p<0.05$) arterial pressures, rate-pressure product, and peripheral vascular resistance in quads. This graded FNS-KE exercise up to the 15-kg/leg load induced relatively small but appropriate increases in aerobic metabolism and cardiopulmonary responses that appear to be safe and easily tolerated by quads and paras. Arterial pressure needs to be monitored carefully in quads to prevent excessive hypertension or hypotension. Although FNS-KE exercise has been shown to elicit peripheral adaptations to improve muscle strength and endurance, it is probably not an effective central cardiovascular training tool for all but the least fit SCI individuals. This information is important for understanding the effects of FNS use during more complex activities such as cycling and ambulation.

Key words: *aerobic metabolism, cardiopulmonary response, cardiovascular training tool, functional neuromuscular stimulation (FNS), knee extension exercise, muscle performance, paralyzed quadriceps muscle, paraplegia, quadriplegia, spinal cord injury (SCI), therapeutic exercise.*

INTRODUCTION

Knee extension (KE)* exercise via functional neuromuscular stimulation (FNS) has been used for strength and endurance training of the paralyzed quadriceps muscles in spinal cord injured (SCI) individuals (6,14,15,16,19,20,22,34). Previous studies have reported acute responses of oxygen uptake ($\dot{V}O_2$), pulmonary ventilation ($\dot{V}_E$), blood pressure (BP) and heart rate (HR) dur-

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*See the **Appendix** for a complete list of abbreviations.

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Journal of Rehabilitation Research and Development Vol. 28 No. 4 Fall 1991

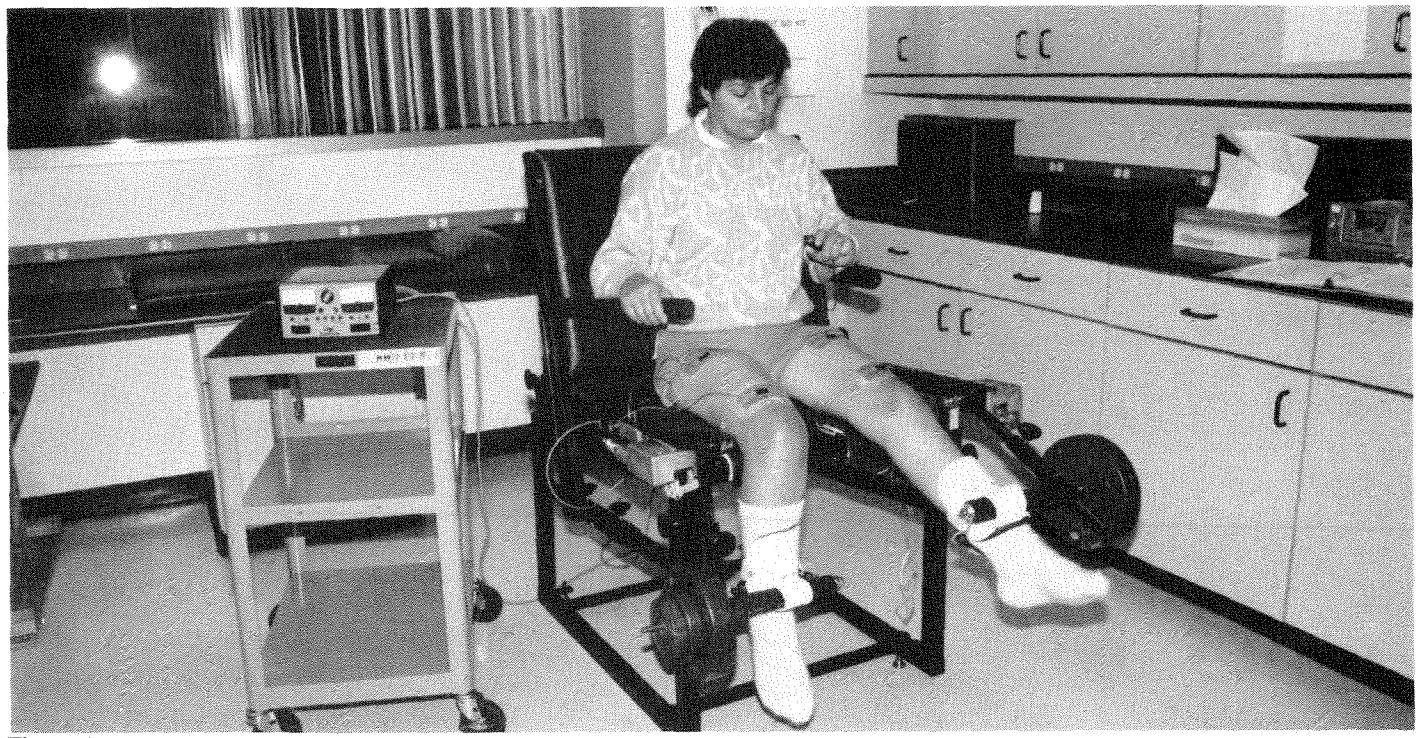

Figure 1.
Subject positioned on the FNS knee extension exercise system.

ing FNS-KE exercise (2,17,20,22,36), but have excluded arteriovenous oxygen difference (a-$\bar{v}O_2$) and hemodynamic variables such as cardiac output ($\dot{Q}$), stroke volume (SV), and total peripheral vascular resistance (TPR). Thus, the hemodynamic responses to this exercise have not been well characterized. Voluntary knee extension exercise by able-bodied individuals is known to induce moderate increases in $\dot{V}O_2$, HR, and $\dot{Q}$, but this is accompanied by extensive sympathetic stimulation producing marked increases in MAP and TPR (30). While previous studies (2,17,20,22,35) suggest that FNS-KE exercise by SCI subjects elicits a moderate pressure load on the heart, the small increase in HR in relationship to $\dot{V}O_2$ indicates little or no systemic sympathetic nervous system activation. These studies have provided limited understanding of the central and peripheral hemodynamic adjustments that facilitate O_2 transport and aerobic metabolism. This information is important in evaluating the potential cardiovascular benefits and risks associated with this form of exercise. Therefore, the purpose of this study was to determine and compare acute hemodynamic responses of SCI quadriplegics (quads) and paraplegics (paras) during FNS-KE exercise using instrumentation and a protocol developed in our laboratory (5). These responses would form the physiologic basis for evaluating the cardiovascular safety and risk of this mode of exercise.

METHODS

Subjects

Fourteen SCI men volunteered to participate in this study. Their mean ($\pm$ SD) age, height, and weight were 32 $\pm$ 8 yr, 178 $\pm$ 8 cm, and 76 $\pm$ 18 kg, respectively. Functional levels of SCI ranged from C_6 to C_7 for the seven quads, and from T_5 to T_{11} for the seven paras. All paras and two of the seven quads had neurologically complete SCIs. All quads demonstrated sympathetic dysfunction as determined by clinical autonomic nervous system tests consisting of BP responses to isometric exercise and head-up tilt; only three of the paras responded abnormally (4). No subjects wore support hose or abdominal binders during testing. Subjects averaged 7 $\pm$ 4 yr since injury, and all had passed medical screening tests. They completed the informed consent procedures in accordance with regulations of the Institutional Review Board of Wright State University.

All subjects had completed at least 3 months of training with FNS-KE exercise and/or FNS leg cycle ergometry prior to completing the FNS-KE testing protocol in this study. They had also previously reached the maximum allowable FNS-KE load of 15 kg/leg without complications. Selection of subjects was not randomized, as they were chosen for their demonstrated responsiveness to FNS

Table 1.
Medications taken by subjects.

Medication	Purpose	Possible Side Effects Affecting Exercise Responses	Paraplegics n	Paraplegics Dosage mg/day	Quadriplegics n	Quadriplegics Dosage mg/day
Valium (diazepam)	centrally acting tranquilizer, anti-spasticity agent	Transient cardiovascular depression	2	5,20	3	5,10,30
Ditropan (oxybutynin chloride)	smooth muscle anti-spasmotic to facilitate bladder emptying	tachycardia, hypertension	4	5,10,10,20	1	5
Lioresal (baclofen)	anti-spastic skeletal muscle relaxant	CNS depression, hypotension	2	20,40	2	40,40
Dibenzaline (phenoxybenzamine chloride)	long-acting adrenergic alpha-receptor blocking agent	hypotension, tachycardia	0	—	1	40

Source: Physicians' Desk Reference. 45th ed., Oradell, NJ: Medical Economics Company, Inc., 1991.

without discomfort, and their ability to complete the FNS-KE testing protocol through the 15-kg/leg stage. Therefore, all subjects could be described as well-trained with above average strength and endurance in FNS-KE exercise.

For safety reasons, medication schedules of the subjects were not altered during this study. **Table 1** summarizes these medications, the number of subjects taking each medication, daily dosages taken, as well as their general purpose and possible side-effects affecting exercise responses (37).

FNS-KE instrumentation and physiologic testing procedures

After completion of medical screening and informed consent procedures, subjects were oriented to the instrumentation and experimental protocol. All testing took place with the subjects sitting upright on an adjustable padded chair (**Figure 1**) that was part of a specially constructed FNS-KE exercise system (5). The resting hip and knee angles were set at 105 degrees and 80 degrees of flexion, respectively. Subjects were strapped securely around the chest, waist, and distal thigh to stabilize the trunk, hips, and thighs. The system allowed a zero to 70 degree range of motion at the knee during FNS-KE exercise. The bilateral left-right cycle rate varied from 3 to 12 KE/leg/min, averaging approximately 6 KE/min/leg. The cycle rate decreased with increased external resistive load and degree of fatigue. Peak stimulation current and the number of muscle fibers stimulated to contract gradually increased automatically to compensate for progressive fatigue that occurred with repetitive contractions. The threshold current and stimulating current ramp outputs were adaptively and automatically controlled via feedback mechanisms to reduce delay between contractions and to accomplish the KE maneuver in a smooth and effective manner (5). Stimulation parameters consisted of biphasic square-wave pulses of 0.3 msec pulse width at 35 Hz frequency, and up to 150 mA current output. Stimulation was delivered via rubberized carbon surface electrodes (4.5 × 10.0 cm, Model 3793, Medtronic, Inc., Minneapolis, MN) taped to the skin, one over a motor point in the proximal lateral quadriceps, and the other in the distal medial quadriceps. Conductive paste ("Liqui-cor," Burdick Corp., Milton, WI) was used between the electrodes and the skin to insure low-resistance contact.

The testing protocol illustrated in **Figure 2** was used to assess physiologic responses during graded FNS-KE exercise. After 5 minutes of rest, subjects performed a continuous progressive-intensity FNS-KE exercise test during which steady-rate physiologic responses were determined. Resistive loads began with 0 kg/leg and progressed by 5-kg/leg increments every 4 minutes to a maximum of 15 kg/leg. The maximal FNS-KE exercise load and stimulating current were limited to 15 kg/leg and 150 mA, respectively,

Journal of Rehabilitation Research and Development Vol. 28 No. 4 Fall 1991

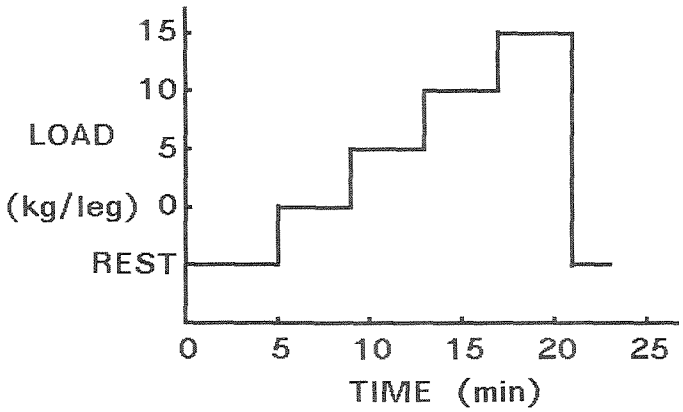

Figure 2.
Testing protocol used during FNS knee extension exercise.

due to the questionable strength of the osteoporotic femoral and tibial bones of SCI subjects and to avoid skin burns (6,20). It is our opinion that abrupt maximal contraction strength tests of rested non-fatigued muscles should not be performed on SCI subjects due to the risk of fracture.

Physiologic determinations

Aerobic metabolic energy expenditure and cardiopulmonary responses were determined noninvasively during the fourth minute of each stage of the test. $\dot{V}O_2$ (STPD), carbon dioxide output ($\dot{V}CO_2$, STPD), and $\dot{V}_E$ (BTPS) were determined with computerized open-circuit spirometry ("System 2001," Medical Graphics Corp., St. Paul, MN). The respiratory exchange ratio (RER) was calculated as $\dot{V}CO_2/\dot{V}O_2$.

Central hemodynamic responses such as HR (bpm), SV (mL/beat), and $\dot{Q}$ (L/min) were assessed with impedance cardiography (26,29,30), utilizing a Minnesota Impedance Cardiograph (Model 304B, Surcom, Inc., Minneapolis, MN). Analog recordings of dz/dt, phonocardiogram, and ECG waveforms were digitized and analyzed via a microcomputer (12).

Other variables monitored at the end of each exercise stage included: (*a*) arterial systolic blood pressure (SBP, mmHg), and diastolic blood pressure (DBP, mmHg) by auscultation; and, (*b*) central, peripheral, and integrated subjective ratings of perceived exertion (RPE) using Borg's 0–10 scale (1). Rate-pressure product (RPP, bpm·mmHg/10^3), representing myocardial O_2 demand or "stress" on the heart (32), was calculated as HR·SBP/10^3. Mean arterial blood pressure (MAP, mmHg), representing the average arterial pressure during the cardiac cycle, was calculated as DBP+[(SBP−DBP)/3]. The a-$\bar{v}O_2$ (mL/100 mL), representing the rate of extraction of O_2 from the blood by peripheral tissues, was calculated as $\dot{V}O_2/\dot{Q}$, and TPR (mmHg/L/min) was calculated as MAP/$\dot{Q}$.

Data analysis

Each physiologic variable was analyzed with a two-way split-plot analysis of variance (ANOVA) between groups and across exercise stages from rest to the 15-kg/leg load. Significant ANOVAs were further analyzed with *post hoc* Tukey tests among exercise stages within each group, separate one-way repeated measures ANOVAs for each group across stages, and *post hoc* independent *t*-tests between groups at each stage. The 5 percent significance level was used in all statistical hypothesis testing.

RESULTS

All subjects completed the graded FNS-KE exercise test to the 15 kg/leg stage. Several physiologic variables ($\dot{V}O_2$, a-$\bar{v}O_2$, $\dot{Q}$, SV, HR, $\dot{V}_E$, RER, RPE) for quad and para groups were not significantly different ($p>0.05$) at each test stage. Therefore, these data were averaged together at each stage and summarized ($\bar{X} \pm SD$) in the lower portion of **Table 2**. When comparing rest with the peak (15-kg/leg) stage for both groups combined, FNS-KE exercise elicited significant ($p<0.05$) increases in $\dot{V}O_2$ by 130 percent (2.3 METS; 0.30 L/min), $\dot{V}_E$ by 120 percent (11.8 L/min), RER by 37 percent (0.31), a-$\bar{v}O_2$ by 57 percent (3.1 mL/100 mL), $\dot{Q}$ by 32 percent (1.51 L/min), SV by 41 percent (26 mL/beat), and HR by 6 percent (4 bpm).

Central, peripheral, and integrated RPEs were not significantly different from each other within each exercise stage; therefore, only the integrated RPE was used in subsequent statistical analyses. RPE increased significantly across exercise stages, but remained at low levels (0.2–2.2 during FNS-KE) throughout the test.

Mean MAP, TPR, and RPP were significantly lower for quads than for paras at each exercise stage. These data are summarized in the upper portions of **Table 2**. For the paras, MAP and RPP increased significantly by 15 percent and 14 percent (13 mmHg and 1.2 bpm·mmHg/10^3), respectively, while TPR decreased significantly by 28 percent (−6.4 mmHg/L/min). For the quads, MAP and RPP increased significantly by 26 percent and 15 percent (17 mmHg and 13 bpm·mmHg/10^3), respectively, while TPR did not change.

DISCUSSION

Previous investigators have reported $\dot{V}O_2$, $\dot{V}_E$, HR, and BP responses to various modes of FNS-KE exercise (2,17,20,22,36), but no data have been available concerning the central and peripheral hemodynamic adjustments

Table 2.
Physiologic responses of subjects during FNS-induced knee extension exercise.

	Rest	0 kg/leg	5 kg/leg	10 kg/leg	15 kg/leg	
Paraplegics (n=7)						
Oxygen Uptake (L/min)	0.24 ± 0.05	0.36 ± 0.08	0.44 ± 0.08	0.50 ± 0.10	0.58 ± 0.11	
Pulmonary Ventilation (L/min)	10.0 ± 1.7	13.7 ± 1.1	17.1 ± 2.4	19.9 ± 3.4	22.6 ± 4.5	
Arteriovenous O_2 Difference (ml/100 ml)	6.1 ± 1.8	7.8 ± 1.8	8.4 ± 1.8	8.8 ± 2.0	8.9 ± 1.8	
Cardiac Output (L/min)	4.04 ± 0.63	4.72 ± 0.47	5.30 ± 0.50	5.79 ± 0.67	6.56 ± 0.31	
Stroke Volume (ml/beat)	59 ± 12	71 ± 10	81 ± 9	83 ± 10	95 ± 10	
Heart Rate (bpm)	70 ± 9	67 ± 8	66 ± 7	70 ± 5	71 ± 5	
Mean Arterial Pressure (mmHg)	89 ± 9	93 ± 10	95 ± 7	101 ± 11	102 ± 16	*
Total Peripheral Resistance (mmHg/L/min)	22.6 ± 5.2	19.9 ± 4.1	18.0 ± 3.0	17.9 ± 3.2	16.3 ± 1.8	*
Rate-Pressure Product (bpm·mmHg/10^3)	8.5 ± 0.8	8.6 ± 1.4	7.7 ± 0.9	9.4 ± 0.7	9.7 ± 1.1	*
Rating of Perceived Exertion (Integrated)	0 ± 0	0.4 ± 0.4	0.8 ± 0.6	1.8 ± 1.0	2.6 ± 1.5	
Quadriplegics (n=7)						
Oxygen Uptake (L/min)	0.21 ± 0.03	0.31 ± 0.05	0.37 ± 0.05	0.42 ± 0.04	0.49 ± 0.05	
Pulmonary Ventilation (L/min)	9.6 ± 1.3	12.9 ± 1.8	15.1 ± 2.2	18.2 ± 2.2	20.8 ± 3.4	
Respiratory Exchange Ratio	0.84 ± 0.06	0.94 ± 0.05	1.02 ± 0.06	1.10 ± 0.07	1.13 ± 0.07	
Arteriovenous O_2 Difference (mL/100 mL)	4.8 ± 0.91	5.8 ± 1.5	7.0 ± 0.6	8.0 ± 1.2	8.3 ± 1.1	
Cardiac Output (L/min)	4.50 ± 0.73	4.80 ± 0.72	5.21 ± 0.52	5.73 ± 0.77	6.01 ± 0.79	
Stroke Volume (mL beat)	68 ± 17	74 ± 19	80 ± 20	80 ± 22	84 ± 22	
Heart Rate (bpm)	68 ± 12	67 ± 14	69 ± 14	71 ± 15	76 ± 17	
Mean Arterial Pressure (mmHg)	66 ± 15	71 ± 9	76 ± 11	81 ± 15	83 ± 18	*
Total Peripheral Resistance (mmHg/L/min)	14.7 ± 3.4	15.7 ± 1.9	14.7 ± 1.0	15.4 ± 3.7	13.9 ± 3.8	*
Rate-Pressure Product (bpm·mmHg/10^3)	5.5 ± 0.9	5.9 ± 1.0	6.5 ± 0.8	7.0 ± 1.3	7.9 ± 1.7	*
Rating of Perceived Exertion (Integrated)	0 ± 0	0.1 ± 0.2	0.9 ± 0.2	1.1 ± 0.6	1.8 ± 0.8	
All Subjects (N=14)						
Oxygen Uptake (L/min)	0.23 ± 0.04	0.34 ± 0.07	0.40 ± 0.07	0.46 ± 0.08	0.53 ± 0.09	
Pulmonary Ventilation (L/min)	9.8 ± 1.4	13.3 ± 1.5	16.1 ± 2.4	19.1 ± 2.9	21.6 ± 3.9	
Respiratory Exchange Ratio	0.84 ± 0.04	0.94 ± 0.07	1.03 ± 0.6	1.10 ± 0.06	1.15 ± 0.09	
Arteriovenous O_2 Difference (mL/100 mL)	5.4 ± 1.5	6.8 ± 1.9	7.7 ± 1.5	8.4 ± 1.6	8.6 ± 1.4	
Cardiac Output (L/min)	4.27 ± 0.69	4.80 ± 0.59	5.26 ± 0.49	5.58 ± 0.73	6.27 ± 0.66	
Stroke Volume (mL/beat)	63 ± 15	73 ± 15	80 ± 15	81 ± 17	89 ± 18	
Heart Rate (bpm)	69 ± 10	67 ± 11	67 ± 11	71 ± 11	73 ± 13	
Mean Arterial Pressure (mmHg)	78 ± 17	82 ± 15	86 ± 13	91 ± 16	92 ± 19	*
Total Peripheral Resistance (mmHg/L/min)	18.6 ± 5.8	17.8 ± 3.7	16.4 ± 2.7	16.6 ± 3.6	15.0 ± 3.2	*
Rate-Pressure Product (bpm·mmHg/10^3)	7.0 ± 1.7	7.2 ± 1.8	7.1 ± 1.0	8.2 ± 1.0	8.7 ± 1.7	*
Rating of Perceived Exertion (Integrated)	0 ± 0	0.2 ± 0.3	0.8 ± 0.5	1.5 ± 0.9	2.2 ± 1.2	

Values are $\bar{X} \pm$ SD.
*Denotes statistically significant difference between paraplegics and quadriplegics.

during FNS-KE. Due to their different testing instrumentation and protocols (2,17,22,36), physiologic responses are difficult to compare in these studies.

Aerobic metabolism and pulmonary ventilation

$\dot{V}O_2$ increased linearly with load, probably reflecting local metabolic control within skeletal muscle fibers. Similar to previous studies, $\dot{V}O_2$ during FNS-KE exercise in the present study approximately doubled (2.3 METS) the resting level in both quads and paras. The peak $\dot{V}O_2$ of 0.53 L/min during FNS-KE in this study is low compared to the peak values previously reported for SCI subjects during voluntary arm-crank ergometry: 0.8 L/min for quads, 1.5 L/min for paras (40). Similarly, the peak $\dot{V}O_2$ during FNS-KE in this study is low compared to those of approximately 1.0 L/min reported for trained SCI subjects during FNS leg cycle ergometry (8,38). Thus, FNS-KE elicited a peak $\dot{V}O_2$ approximately one-half of that reported for FNS leg cycle ergometry which utilizes more muscles and stimulates them to contract at a higher frequency (11,18,23). Judging from the small muscle mass that was activated, the low frequency of contractions, and low peak $\dot{V}O_2$, FNS-KE is probably not an effective central cardiovascular training tool for all but the least fit SCI individuals.

$\dot{V}_E$ increased linearly with load and $\dot{V}O_2$ during progressive-intensity FNS-KE exercise. The precise nature of this control is currently speculative. However, considering the peripheral nature of FNS and the interrupted autonomic pathways, the control of $\dot{V}_E$ appears primarily humoral in nature (23,24). The linear increase in RER over unity at loads exceeding 5 kg/leg strongly suggests heavy CO_2 production from anaerobic metabolism and lactic acid buffering by the bicarbonate system. The resulting hypercapnea and metabolic acidosis perhaps drives ventilation via chemoreceptor mechanisms. Similar to peak $\dot{V}O_2$, peak $\dot{V}_E$ was also relatively low compared to typical aerobic exercise modes (11,18,23), suggesting that FNS-KE will not stress the pulmonary system to the extent required for substantial training effects.

O_2 transport: Cardiovascular and hemodynamic responses

According to the Fick relationship ($\dot{V}O_2 = \dot{Q} \cdot a\text{-}\bar{v}O_2$), elevations in $\dot{V}O_2$ must be accompanied by increases in $\dot{Q}$ and/or $a\text{-}\bar{v}O_2$. In this study, the 136 percent increase in $\dot{V}O_2$ during FNS-KE exercise was achieved through 32 percent and 57 percent increases in $\dot{Q}$ and $a\text{-}\bar{v}O_2$, respectively. This indicated that the increase in systemic aerobic metabolism was accomplished both by increases in blood and O_2 delivery through the central circulation and by increases in the rate of O_2 extraction in exercising muscles. However, peripheral extraction of O_2 appears to be the predominant of these two factors (7). This suggests that the peak $\dot{V}O_2$ achieved during FNS-KE exercise (2.3 METS) required minimal support from the central cardiovascular system and/or that little or no activation of the sympathetic nervous system is necessary to support this low level of aerobic metabolism.

$\dot{Q}$ is controlled by the systemic circulation which returns blood to the heart and by the pumping activity of the heart. $\dot{Q}$ is directly proportional to the pressure gradient that drives blood through the systemic circulation (i.e., MAP*). $\dot{Q}$ is also inversely proportional to the resistance to arterial blood flow (TPR), normally controlled by the sympathetic activation of arteriolar diameter and local accumulation of metabolites which cause vasodilation in active muscle (21). Thus, $\dot{Q}$ = MAP/TPR, and $\dot{Q}$ can increase from either increases in MAP and/or reductions in TPR. During FNS-KE exercise, the heart is not likely to be the dominant factor determining $\dot{Q}$ (in the absence of heart disease). Since the venous return is likely to be impaired in SCI subjects with sympathetic dysfunction and peripheral vascular insufficiency, the systemic circulation can be viewed as the dominant factor. Similar to $\dot{Q}$, venous return is determined by the ratio of the pressure gradient for venous return and the resistance to venous return influencing $\dot{Q}$. Venous return is usually elevated by increasing the pressure gradient via mechanisms which decrease venous pooling, such as: (*a*) quadriceps contractions which activate the thigh venous muscle pump; (*b*) alternate lower-leg elevation which reduces the vertical column of the venous pool from the heart; and, (*c*) increasing activity of the thoracic/respiratory pump.

The arterial pressure responses in this study differed somewhat from those reported by previous investigators. Whereas a marked pressor response in quads, but not in paras, had been reported in previous studies (22,36), a small increase of MAP in both groups was observed in this study. The MAP of both groups increased, but quads averaged 20 mmHg below paras. These findings support the hypothesis that spinal reflexes, independent of the brain, may exert control on BP in SCI subjects (22,35). According to this hypothesis, accumulation of metabolites from exercising muscles might elicit splanchnic vasoconstriction, counteracting some of the vasodilation in exercising muscles and resulting in increased MAP (28). In this study, quads displayed relatively low MAPs, suggesting profound peripheral vasomotor dysfunction (27). Despite possible exercise-induced splanchnic vasoconstriction, TPR of quads was lower than that of paras at all test stages. In quads, a relatively normal $\dot{Q}$ response was achieved through both low afterload pressure and low resistance in the circulatory system.

While $\dot{Q}$ is not directly determined by SV and HR, increases in HR and/or SV accompany increases in venous return and $\dot{Q}$ ($\dot{Q}$=SV·HR). Since HR increased only 6 percent, the 32 percent increase in $\dot{Q}$ during FNS-KE appeared to be due primarily to the 41 percent increase in SV. This strongly suggests that this FNS-KE exercise system enhances return of venous blood to the heart via mechanical mechanisms that reduce venous pooling and assist venous return from the lower body back to the right atrium of the heart (8). Increased cardiac filling and preload may allow enhanced myocardial performance and SV via the Frank-Starling mechanism. Consequently, the increased SV and MAP during FNS-KE may elicit the parasympathetically mediated baroreflex through the intact efferent vagal arc. This may explain the depressed HR at the lower (0–5 kg/leg) FNS-KE loads and only slight cardioacceleration at the higher (10–15 kg/leg) loads. Vagal with-

*$\dot{Q}$ = Pressure Gradient / Total Peripheral Resistance = ΔP / TPR = (MAP – Right Atrial Pressure) / TPR. Since Right Atrial Pressure ≅ 0 mmHg, ΔP ≅ MAP, and $\dot{Q}$ ≅ MAP / TPR.

drawal, rather than sympathetic stimulation, may be the mechanism for the small increase in HR at the higher loads.

Effects of prior exercise training

All subjects selected for this study were previously trained in FNS exercise and their quadriceps performance was superior to those who were excluded. Although this previous training improved the quadriceps strength and endurance for FNS exercise, it is not currently known if the magnitude of metabolic and cardiopulmonary responses was altered. More fit able-bodied individuals performing a given voluntary exercise task would be expected to have lower cardiopulmonary response levels in comparison to their less fit counterparts. However, the effects of this prior training on physiologic (i.e., cardiopulmonary) responses to FNS-KE at the 0–15 kg/leg loads are believed to be minimal because FNS-KE exercise is peripherally induced, SCI subjects have sympathetic impairments, and FNS exercise training promotes mostly peripheral (muscular) adaptations (25).

No evidence exists to document FNS training-induced increases in sympathetic activity. The low magnitude of the sympathetically mediated cardiovascular responses induced by FNS-KE in both paras and quads suggests that little sympathetic activity is stimulated. Therefore, the level and completeness of SCI are likely to be the primary factors determining the cardiopulmonary response to FNS exercise at any stage of training.

Effects of medications

Many medications and drugs can affect cardiovascular responses during exercise in humans. In this study, subjects were requested to refrain from caffeine, nicotine, alcohol, heavy meals, and strenuous physical activity for at least 3 hours before testing. **Table 1** lists several medications taken by subjects which were prescribed to reduce spasticity of skeletal and smooth (bladder) muscles. Other medications taken by subjects (antibiotics, stomach antacid, stool softener) have no neurological or cardiovascular side effects. One incomplete quadriplegic subject took 40 mg/day of Dibenzyline which can produce and maintain "chemical sympathectomy" (37). Because it increases blood flow to the skin, mucosa, and abdominal viscera, arterial BP usually falls, resulting in orthostatic hypotension and tachycardia. Paradoxically, during rest and FNS-KE exercise, this quadriplegic subject on Dibenzyline was neither hypotensive (BP range: 110/80–100/68) nor tachycardic (HR range: 50–71 bpm). In summary, the medications taken by subjects in this study did not appear to have strong cardiovascular side effects. Therefore, they probably had minimal effects on the collected cardiovascular data.

Practical implications

Safety. Safety is always a concern with involuntary electrically induced exercise in the SCI population. From a cardiovascular perspective, the low absolute levels of peak responses to FNS-KE observed in this study probably pose little risk to otherwise healthy paras or quads. Even during FNS-KE exercise at the 15-kg/leg stage, the RPP (an index of myocardial $\dot{V}O_2$ or cardiac stress) remained within a relatively safe range (31). These responses were also lower than have been observed for SCI subjects during other forms of exercise such as FNS leg cycle ergometry and voluntary arm exercise (9,10,13,18). Although the RPP in paras was significantly higher at all stages than in quads, this difference has no clinical significance due to the low absolute levels in both groups. Low RPP values during FNS-KE are attributed to both low HR and SBP responses. The small increase in RPP across loads was due primarily to the 19 percent increase in SBP and, to a lesser extent, to the 6 percent increase in HR.

Other subjective and physiologic indicators of the cardiovascular safety of FNS-KE exercise as used in this study included: (*a*) mean RPE of 2.2 ("weak" perceived exertion); (*b*) absence of ECG abnormalities that would signify myocardial ischemia; (*c*) mean peak HRs less than 20 percent predicted HR reserve; and, (*d*) absence of hypertensive responses or autonomic dysreflexia. Overall, the cardiovascular responses observed in this study were appropriate for the low level of aerobic metabolism induced by FNS-KE and appeared to be well within the capability of healthy SCI subjects to tolerate with little cardiovascular risk.

Orthostatic hypotension was evident in three quads during FNS-KE exercise in this study. Although completely without hypotensive symptoms or known heart disease, arterial BPs near 70/40 mmHg were recorded at rest and during FNS-KE exercise in these subjects, two of whom were neurologically incomplete. These subjects displayed orthostatic hypotension while resting in the upright sitting posture before the exercise test began. During FNS-KE exercise, their BPs generally increased toward normal resting levels; thus, FNS-KE exercise served to counteract orthostatic hypotension in quads. Readers are cautioned to monitor BP in upright quads, regardless of completeness of SCI, and especially if a history of peripheral vascular insufficiency or orthostatic hypotension is present (3). Additionally, no orthopedic or skin complications were encountered during the FNS-KE exercise testing.

Training. Due to the low levels of aerobic metabolism, central circulation, and cardiac volume and pressure loading elicited by FNS-KE exercise in this study, we predict little central cardiovascular adaptation to FNS-KE training. However, FNS-KE training would be expected to produce local adaptations in muscles such as muscular hypertrophy, increased strength and endurance, increased vascularization and enhanced ability to extract and utilize O_2. It is probable that local anaerobic metabolic capability would also be improved, since submaximal FNS recruits predominantly fast-twitch muscle fibers (30). Previous training studies have already documented FNS-KE training-induced increases in muscular strength and endurance and/or thigh girth (6,33,39).

Since FNS-KE does not substantially activate sympathetic responses in SCI subjects, acute physiologic responses and chronic trainability may be quite limited relative to able-bodied individuals. Future research is needed to document changes in sympathetic and parasympathetic activity during exercise in SCI subjects, whether voluntary or electrically stimulated, and to relate these findings to the degree of acute cardiovascular response and aerobic trainability.

CONCLUSIONS

The acute hemodynamic responses of these SCI subjects indicate that graded FNS-KE exercise stresses the O_2 transport system to a relatively low degree. FNS-KE induces mild cardiac volume and pressure loads that are easily tolerated by, and appear to be safe for, paras and quads. Physiologic responses of quads and paras are similar, except for the relative hypotension and lower peripheral vascular resistance in quads. These differences are probably associated with impaired peripheral sympathetic nervous system activity and limited vasoconstriction in quads. Hypothetically, FNS-KE training would be expected to produce greater peripheral adaptations in muscles than central cardiovascular changes due to the low levels of aerobic metabolism and central circulatory responses generated by this mode of exercise.

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Journal of Rehabilitation Research and Development Vol. 28 No. 4 Fall 1991

APPENDIX

List of Abbreviations

ANOVA	Analysis of variance
a-$\bar{v}O_2$	Arteriovenous oxygen difference (mL/100 mL)
BP	Blood pressure (mmHg)
BTPS	Body temperature and pressure, saturated
C	Cervical
CO_2	Carbon dioxide
DBP	Diastolic blood pressure (mmHg)
dz/dt	First derivative of the impedance change with time, rate of change of impedance
ECG	Electrocardiogram
FNS	Functional neuromuscular stimulation
HR	Heart rate (bpm)
KE	Knee extension
MAP	Mean arterial pressure (mmHg)
METS	Multiples of the resting metabolic rate
O_2	Oxygen
para	Paraplegic
quad	Quadriplegic
$\dot{Q}$	Cardiac output (L/min)
RER	Respiratory exchange ratio
RPE	Rating of perceived exertion
RPP	Rate-pressure product (bpm·mmHg/10^3)
SBP	Systolic blood pressure (mmHg)
SCI	Spinal cord injury, spinal cord injured
SD	Standard deviation
STPD	Standard temperature and pressure, dry
SV	Stroke volume (mL/beat)
T	Thoracic
TPR	Total peripheral resistance (mmHg/L/min)
$\dot{V}CO_2$	Carbon dioxide output (L/min)
$\dot{V}_E$	Pulmonary ventilation (L/min)
$\dot{V}O_2$	Oxygen uptake (L/min)
$\bar{X}$	Mean