

## Effect of functional neuromuscular stimulation on postural related orthostatic stress in individuals with acute spinal cord injury

Ahmed S. Elokda, MA, PT; David H. Nielsen, PhD, PT; Richard K. Shields, PhD, PT

The University of Iowa, Physical Therapy Graduate Program, 2600 Steindler Building, Iowa City, IA 52242-1008

**Abstract**—The purpose of the study was to evaluate the cardiovascular responses of functional neuromuscular stimulation (FNS) on postural-related orthostatic stress in individuals with acute spinal cord injury. Two tetraplegics and three paraplegics participated in this study. A repeated measure design was used in which subjects underwent two graded-tilt table (0°, 15°, 30°, 45°, 60°) orthostatic stress tests with and without FNS (randomized treatment order) of the knee extensors and foot plantar flexors. Successive one-minute digital readings of heart rate (HR) and blood pressure were obtained with an electronic pulse oximeter and automated blood pressure monitor, respectively. Analysis of covariance (ANCOVA) revealed significant test position (tilt angle) and treatment condition (with or without FNS) main effects, but no significant interactions. Between-treatment pairwise contrasts showed that systolic blood pressure was lower without FNS for 15°, 30°, 45°, and 60° of tilt, while diastolic blood pressure was lower without FNS at 30° and 45° of tilt. In contrast, HR showed a progressive rise with increasing tilt angle. Functional neuromuscular stimulation may be an important treatment adjunct to minimize cardiovascular changes during postural orthostatic stress in individuals with acute spinal cord injury.

This material is based on work supported in part by a grant from the Paralyzed Veterans of America Spinal Cord Research Foundation awarded to Richard K. Shields.

Address all correspondence and requests for reprints to: Richard K. Shields, PhD, PT, The University of Iowa, Physical Therapy Graduate Program, 2600 Steindler Building, Iowa City, IA 52242-1008; email: richard-shields@uiowa.edu

**Key words:** *blood pressure, functional neuromuscular stimulation, spinal cord injury, tilt table.*

### INTRODUCTION

Approximately 200,000 individuals with spinal cord injury live in the United States, with the addition of 7,000 new cases each year (1,2). Approximately half of the individuals with acute spinal cord injury have complete injuries of the spinal cord accompanied by loss of voluntary motor and sensory function below the level of the lesion (1). Numerous cardiovascular and hemodynamic effects constitute drastic sequelae after acute spinal cord injury. The magnitude of these effects is directly related to the level and severity of the injury, with the largest changes occurring in complete cervical injury (3). In this population, impairment of the sympathetic nervous system, in addition to compromised respiratory and venous muscle pumps, usually leads to severe venous pooling in the lower extremities.

Secondary effects include posttraumatic hypotension and diminished cardiac output (4,5), which are both significant contributing factors for the development of deep venous thrombosis and pulmonary embolism (3). A complete loss of autonomic responses results in orthosta-

tic hypotension when the individual with acute spinal cord injury sits up or passively stands. Reconditioning individuals with acute spinal cord injury to tolerate the upright position constitutes an initial part of the rehabilitation program. The use of a tilt table or gradually elevating a reclining wheelchair is usually the common methods to achieve this goal (6). A variety of external compression techniques, such as elastic stockings, pressure boots, and antigravity suits, have been used with varied success in decreasing orthostatic hypotension (7,8). In the last decade, functional neuromuscular stimulation (FNS) has been suggested to maintain muscle girth and bone integrity after spinal cord injury. The extent to which FNS can facilitate upright postures by maintaining blood pressure has not been systematically studied in individuals with acute spinal cord injury (9).

Davis et al. (10) presented the effects of arm crank exercise with and without FNS of the lower extremity in individuals with chronic spinal cord injury. The FNS group demonstrated a 10–19 percent augmentation in stroke volume and a 4–16 bpm decrease in heart rate (HR) with a net effect of a mild enhancement (7–8 percent increase) in cardiac output compared to the group not receiving FNS. Glaser et al. (11) evaluated the cardiovascular responses to FNS in able-bodied individuals and individuals with chronic spinal cord injury. Both groups showed between a 12–30 percent increase of stroke volume and cardiac output. The proposed mechanism for this increase during FNS was thought to be the mechanical shunting of blood due to muscle contraction (11). Phillips et al. (12) investigated the effects of FNS intensity on lower limb blood flow during multilevel-intensity arm crank exercise in 8 individuals with chronic spinal cord injury. Blood flow was increased incrementally with increases in intensity of FNS across all exercise levels. The authors proposed that FNS is effective in increasing blood flow and reducing venous pooling in paralyzed muscle in individuals with chronic spinal cord injury.

Figoni et al. (13) studied the cardiovascular effects of FNS concurrent with arm crank exercises during tilting between 30° and 70° in individuals with chronic spinal cord injury. The stroke volume increased 16 percent and the cardiac output increased 18 percent at the 30° tilt position. Conversely, FNS had no effect on the stroke volume and cardiac output at the 70° tilt position, and no effect on HR and systolic blood pressure (SBP) at the 30° or 70° tilt position. The effect of tilting upright with or without FNS was presented for four individuals with

chronic spinal cord injury. Stroke volume decreased with increasing tilt angles while cardiac output decreased only at 10°. Although no blood pressure data were reported, the authors indicated that a general response to tilting was an increase in SBP by 7–16 mmHg with FNS.

Several of the studies on cardiovascular responses to FNS have been presented only in abstract form (10,11,13,14). Moreover, all previous studies examining cardiovascular responses to FNS were carried out in individuals with chronic spinal cord injury. However, postural-related orthostatic stress most often occurs clinically when placing individuals with acute spinal cord injury in a sitting or standing position. Accordingly, we examined the cardiovascular responses (HR, SBP, and DBP [diastolic blood pressure]) during tilting, with or without electrical stimulation of the lower leg in individuals with acute spinal cord injury. We hypothesized that electrical stimulation would attenuate the posturally induced orthostatic hypotension.

## METHODS

### Individuals

Five individuals with acute spinal cord injury selected from the inpatient population at the University of Iowa Hospitals and Clinics were invited to participate in the study. Medical/orthopedic clearance and informed written consent were obtained prior to enrollment. All individuals had acute and complete spinal cord lesions (one week to six weeks postinjury). The completeness of the spinal cord injury was determined by clinical neurological assessment to indicate loss of motor function, loss of sharp-dull distinction, loss of light touch, and no joint position sense below the level of lesion. Exclusion criteria included secondary medical complications involving the kidney, bladder, heart, lungs, skin, or peripheral vascular system. Individuals on cardiovascular medications (anti-hypertension and/or inotropic or chronotropic drugs) were excluded. Two individuals with tetraplegia and three individuals with paraplegia were included in the study. Descriptive data are presented in **Table 1**.

### Instruments

Subject's HR was monitored with a Nellcor pulse oximeter (Nellcor Pulse Oximeter, N3000, Nellcor, Inc., 2391 Fenton Street, Chula Vista, CA 91914). A finger sensor was used. The system provided a continuous digital HR readout with an accuracy of  $\pm 2$  beats (15,16). The

**Table 1.**

Descriptive subject data (n=5 males).

| No          | Diag  | Week  | Age    | Height    | Weight    |
|-------------|-------|-------|--------|-----------|-----------|
| 1           | T8    | 4     | 36     | 172.7     | 102.3     |
| 2           | C6,C7 | 3     | 32     | 181.0     | 81.8      |
| 3           | T8    | 2     | 27     | 182.9     | 63.6      |
| 4           | C8,T1 | 3     | 27     | 177.8     | 62.7      |
| 5           | C6    | 3     | 26     | 177.8     | 63.6      |
| Mean and SD |       | 3±0.7 | 29±4.3 | 178.4±3.8 | 74.8±17.3 |

No=subject number; Diag=clinical diagnosis; Week=weeks post injury; Age in years; Height in cm; Weight in kg.

BP was obtained using a digitally displayed Nellcor Symphony Blood Pressure Monitor, N-3100 (Nellcor, Inc., 2391 Fenton Street, Chula Vista, CA 91914). An automatic pressure source inflated the rubber bladder of the pressure cuff at a controlled variable rate of inflation and deflation. The system could be manually triggered or set in an automatic mode. The automatic mode (one-minute intervals) was adopted for the current study. The pressure cuff was positioned on the arm such that the arrow in the cuff was placed over the brachial artery. Although recovery periods were provided between trials, baseline measurements were not always identical. Data were subsequently analyzed by ANCOVA with the baseline measurements as the covariate. The investigator recorded pressure measurements. The measurement accuracy was  $\pm 2$  mmHg (15,16). A visual analogue scale was used to ensure patient safety through monitoring the severity of symptoms, fainting and/or headache, that the subject may experience. Description of symptoms (headache, neck/shoulder pain, dimming or loss of vision, weakness, syncope, hot sweating, upset stomach) was noted at the bottom of the data record sheet.

### Procedure

Descriptive subject data (gender, age, height, weight) were obtained from the medical record. Testing was conducted in the Human Performance Laboratory at the Multidisciplinary Rehabilitation Center of the University of Iowa Hospitals and Clinics. Individuals were transported to and from the research laboratory by hospital staff. Upon arrival to the research laboratory, the individuals were immediately transferred to the tilt table (0° tilt) to facilitate acclimation to the research environment and to insure good baseline resting measurements. The individuals were positioned on the tilt table with the feet in slight contact with the footboard at the end of the table. The footboard accommodated partial weight bear-

ing during the tilting procedures. Adjustable restraining straps were secured at the chest and knee levels to provide subject support and safety. The arms were kept free to allow accessibility for the blood pressure measurements. Angle of inclination was monitored by a reference goniometer affixed to the tilt table. Inclination of the tilt table was manually regulated with the toggle control switch for the tilt table electrical motor. The test protocol involved a minimum of six minutes of resting 0°g baseline measurements followed by six four-minute stages for each of the tilt table angles (0°, 15°, 30°, 45°, 60°g) followed by four minutes of recovery. The physiological measurements (HR, SBP, and DBP) were taken at one-minute intervals during the resting baseline period and the respective graded test positions. Subject perception of orthostatic tolerance was also assessed at one-minute intervals during the tilting procedures. Criteria for terminating the test were based on acute low levels of hypotension (SBP < 60 mmHg or DBP < 40 mmHg) to avoid kidney filtration problems (17), or severe orthostatic symptoms demonstrated by the subjects' reports of fainting and/or headache. The above test protocol was conducted with and without the application of FNS to the lower extremities (bilateral stimulation of the knee extensors and ankle plantar flexors). The with *versus* without order was counter balanced.

The electrical stimulation procedure first involved rubbing the skin with alcohol where stimulating electrodes were placed (motor points of the quadriceps and the ankle plantar flexors). For the quadriceps the distal electrode was placed over the vastus medialis muscle while the proximal electrode was placed over the vastus lateralis muscle near its insertion on the upper femur. For the plantar flexors, one electrode was placed proximally over the belly of the gastrocnemius muscle while the distal electrode was placed 4 inches above the malleolus over the soleus muscle. Large rectangular (8 cm by 13

cm), flexible, self-adhesive pad electrodes (Verso-Stimi Con Med. Corp., Utica, NY) were used in conjunction with a custom-designed computer-interfaced electrical stimulation unit (18). The stimulator had a range of 0–400 volts with a constant current from 0 to 200 milliamps. The stimulator was triggered by digital pulses from a board housed in a microcomputer under custom software control. The stimulator emits biphasic square waves with pulse widths ranging from 200 to 800 microseconds. A 20-Hz stimulation frequency was used without any ramping of the intensity. Alternating 2-seconds-on and 4-seconds-off periods were used to minimize muscle fatigue (18,19,20,21) and to optimize the movement of blood through the muscle during the short 2-second contraction. The intensity of the stimulation was adjusted to achieve a strong visible contraction of the quadriceps and plantar ankle flexor muscles.

### Data Analysis

The study was based on two factors: 1) posture (tilt table angle, 0°, 15°, 30°, 45°, 60°), and 2) functional neuromuscular stimulation (A with, B without). The order for stimulation was counter balanced so that some individuals received no FNS first while others received no FNS second.

Statistical analysis was performed using the Statistical Analysis System (SAS), Statistical Analysis System Institute, Inc., Cary, NC. Descriptive statistics (means and standard deviations) were calculated on the demographic data (age, height, weight). A  $p$  value of  $<0.05$  was adopted for statistical significance in this study. Although recovery periods were provided, between trials baseline measurements were not always identical. Data were subsequently analyzed by ANCOVA with baseline measurements as covariates. To enhance reproducibility and optimize statistical power, the ANCOVA utilized all observations and performed the analysis on the computed means of each tilt stage. Based on the adjusted mean values, the ANCOVA tested the interaction and the main effects for the treatment conditions (with FNS *versus* without FNS), and test positions (angles of tilt). On an *a priori* basis, in order to provide more definite information regarding the treatment effect of FNS, Tukey pairwise comparisons were made at each test position (angles of tilt).

To investigate the accumulative effect of the tilting procedure, paired  $t$ -tests were used to analyze the overall tilt effect (0°-maximum tilt) for each test condition with FNS and without FNS. Paired  $t$ -tests were also used to investigate the between-treatment effects (0°-maximum tilt differences with FNS *versus* without FNS). Subsequent to visual

analysis of the raw data, there appeared to be a possible rebound effect associated with the tilting procedure. This question was more thoroughly investigated through paired  $t$ -tests of the base line per tilt *versus* recovery measurements for each test condition, with FNS *versus* without FNS. Paired  $t$ -tests were also used to compare the magnitude of the with FNS *versus* without FNS difference scores.

## RESULTS

The SBP showed a progressive decrease with increasing tilt angle. The FNS treatment condition appeared to attenuate the rate of SBP decrease. The ANCOVA revealed significant test position (tilt angle) and treatment (with or without FNS) main effects ( $p<0.05$ ), but the test of interaction was not significant ( $p>0.05$ ). The mean ANCOVA-adjusted SBP values are presented in **Table 2**. Based on the *a priori* between-treatment pairwise contrasts the difference in SBP for the 0° test position was not significant ( $p>0.05$ ). However, for all other test positions, SBP without FNS was lower than with FNS. The SBP decreased for both the with-FNS and the without-FNS conditions, with the magnitude of the decrease being more pronounced without FNS. The paired  $t$ -test indicated a significant overall tilt effect in both conditions, with and without FNS ( $p<0.05$ ). The magnitude of the FNS overall tilt effect was smaller than the without-FNS effect ( $p<0.05$ ). The magnitude of the rebound (recovery minus baseline difference values) for the condition with FNS was relatively larger than the without-FNS conditions. The recovery values for both with and without FNS were slightly elevated, with the magnitude of the increase with FNS being slightly higher. The paired  $t$ -test indicated a baseline *versus* recovery difference for the with-FNS condition ( $p<0.05$ ), and no difference without FNS ( $p<0.05$ ). The magnitude of the rebound with FNS was greater than that without FNS ( $p<0.05$ ).

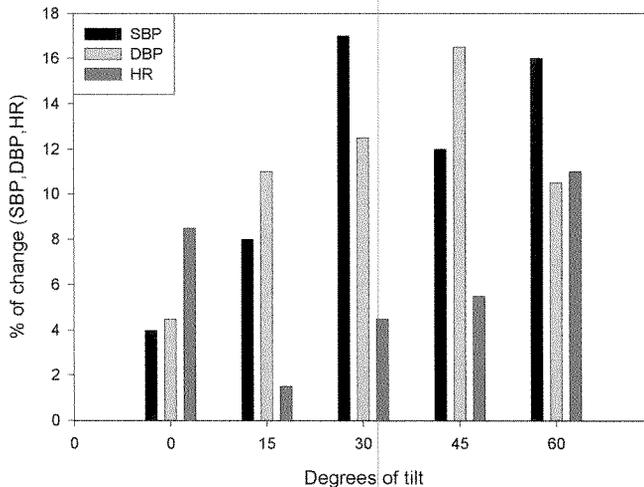
**Table 2.**

Systolic blood pressure in mmHg\*: means, standard deviations, and between-treatment Tukey mean contrasts.

| Tilt | With FNS  | Without FNS | P      |
|------|-----------|-------------|--------|
| 0    | 118.9±1.9 | 118.0±2.3   | 0.7478 |
| 15   | 116.3±1.9 | 110.6±2.1   | 0.0480 |
| 30   | 110.6±1.9 | 099.0±2.2   | 0.0001 |
| 45   | 105.0±2.4 | 098.1±2.4   | 0.0392 |
| 60   | 098.9±2.3 | 090.7±2.2   | 0.0071 |

\*=all values ANCOVA adjusted; Tilt=tilt sequence, in degrees.

The mean percentage change for SBP at each degree of tilt with FNS as compared to that without FNS is graphically presented in **Figure 1**. Indeed, at all tilt angles the SBP was higher with FNS. Moreover, the magnitude of the effect increased at greater tilt angles. Hence, at a 60° tilt angle there was a 15 percent increase in SBP with FNS as compared to not using FNS.



**Figure 1.**

Mean percentage change between with-FNS and without-FNS conditions for systolic blood pressure (SBP), diastolic blood pressure (DPB), and heart rate (HR), at each degree of tilt.

The DBP showed a progressive decrease with increasing tilt angle from 0° to 30°, then tended to plateau between the 30° and 60° angle of tilt. For all test angle positions, the without-FNS DBP was lower than for the with-FNS DBP. The ANCOVA revealed significant test position (tilt angle) and treatment (with or without FNS) main effects ( $p < 0.05$ ). The test of interaction was not significant ( $p = 0.34$ ). The mean ANCOVA-adjusted DBP values are presented in **Table 3**. Based on between-treatment pairwise contrasts, the differences in DBP for 0°, 15°, and 60° were not significant. However, for the 30° and 45° test positions, the DBP without FNS was lower than that with FNS. The DBP decreased for both with-FNS and without-FNS conditions, with the magnitude of the decrease more pronounced for without FNS. The paired *t*-test indicated a significant overall tilt effect in both conditions; with and without FNS ( $p < 0.05$ ). The magnitude of the with-FNS overall effect was found to be significantly smaller than the without-FNS ( $p < 0.05$ ). The

magnitude of the rebound (recovery minus baseline differences values) for the condition with FNS was relatively larger than the without-FNS condition. The recovery values for both the with-FNS and without-FNS conditions were slightly elevated with the magnitude of the increase, with the with-FNS condition slightly higher. The paired *t*-test indicated a significant baseline *versus* recovery difference for with FNS ( $p < 0.05$ ), and a non-significant difference without FNS ( $p < 0.05$ ). The rebound with FNS was not significantly different from without FNS ( $p > 0.05$ ).

**Table 3.**

Diastolic blood pressure in mmHg\*: means, standard deviations, and between-treatment Tukey mean contrasts.

| Tilt | With FNS  | Without FNS | P      |
|------|-----------|-------------|--------|
| 0    | 69.9±1.4  | 70.0±1.7    | 0.9900 |
| 15   | 67.1±1.4  | 64.6±1.7    | 0.1438 |
| 30   | 63.6±1.4  | 59.5±1.5    | 0.0236 |
| 45   | 63.7±1.8  | 58.3±1.7    | 0.0128 |
| 60   | 61.89±1.7 | 60.1±1.6    | 0.3900 |

\*=all values ANCOVA adjusted; Tilt=tilt sequence, in degrees.

The mean percentage change for DBP at each degree of tilt with FNS as compared to without FNS is presented in **Figure 1**. The magnitude of the effect of FNS on DBP becomes greater at higher tilt angles. Indeed, with FNS there was, on average, a 10 to 15 percent increase in DBP.

There was a rise in HR with increasing tilt angle. The with-FNS and without-FNS HR response lines appeared to coincide through the 45° tilt angle. This was followed by an abrupt increase in HR for the with-FNS condition at the 60° tilt angle. The ANCOVA revealed significant test position (tilt angle) and treatment (with or without FNS) main effects ( $p < 0.05$ ). However, the test of interaction was also statistically significant ( $p < 0.05$ ). The mean ANCOVA-adjusted HR values are presented in **Table 4**. Based on the between-treatment pairwise contrasts, there was only one significant difference [at 60° tilt HR was significantly higher with FNS than without FNS ( $p < 0.05$ )]. The HR increased for both with-FNS and without-FNS conditions. The paired *t*-test indicated a significant overall tilt effect in both conditions; with and without FNS ( $p < 0.05$ ). The magnitude of the HR increases for the with *versus* without conditions was not significantly different ( $p < 0.05$ ). The recovery HR was lower

than baseline for both the with- and without-FNS conditions. However, the without-FNS decrease was more pronounced. The paired *t*-tests indicated a significant baseline *versus* recovery difference for without FNS ( $p < 0.05$ ), but a nonsignificant difference with FNS ( $p < 0.05$ ). The magnitude of the rebound without FNS was significantly greater than the without-FNS rebound ( $p < 0.05$ ).

**Table 4.**

Heart rate (beats per minute)\*: means, standard deviations, and between-treatment Tukey mean contrasts.

| Tilt | With FNS  | Without FNS | p      |
|------|-----------|-------------|--------|
| 0    | 77.0±1.2  | 74.2±1.4    | 0.1443 |
| 15   | 82.8±1.2  | 85.0±1.2    | 0.2100 |
| 30   | 92.3±1.2  | 92.1±1.3    | 0.9200 |
| 45   | 98.5±1.5  | 97.1±1.6    | 0.5100 |
| 60   | 112.7±1.4 | 104.9±1.4   | 0.0001 |

\*=all values ANCOVA adjusted; Tilt=tilt sequence, in degrees.

The mean percentage change for HR at each degree of tilt with FNS as compared to not using FNS is presented in **Figure 1**. The effect of FNS on HR was more variable at all angles; however, the HR was always greater with FNS.

## DISCUSSION

Rehabilitation specialists are challenged to design and implement patient rehabilitation programs for acute spinal cord injury that will be safe while still adequately addressing the sequelae of prolonged recumbency and bedrest. These sequelae may include but are not restricted to orthostatic hypotension (19), lower limb edema (22), deep venous thrombosis (23), pulmonary embolism (22), hypercalciuria (24), osteoporosis (25,26), and metabolic and physiological deteriorations (27). Orthostatic training is a cornerstone to rehabilitating an individual with acute spinal cord injury because it encourages early weight bearing on the lower extremities and facilitates early transition to wheelchair mobilization. Tilt table conditioning is an accepted procedure for the clinical treatment of orthostatic hypotension (6,7,8). With regard to individuals with spinal cord injury, FNS has been used primarily with arm crank exercise, and in one report the combination of arm crank exercise, tilting, and FNS was

used in individuals with chronic spinal cord injury. One study investigated the effect of tilting and FNS without arm crank exercise. This study represents the first controlled study that systematically investigates the combined effects of multiple angles of tilting and FNS in individuals with acute spinal cord injury.

The outcome variables of SBP, DBP, and HR were used to assess the treatment efficacy. The results of this study showed a progressive decrease in SBP and DBP with and without FNS at 15°, 30°, 45K and 60K of tilting. The accumulative tilting effects (0° *versus* maximum tilt) showed similar findings. In all cases the decreases were less pronounced with FNS compared to without FNS (**Figure 1**). Impaired vasomotor tone leading to venous pooling with decreased venous return and subsequent decreased Starling effect (decreased presystolic ventricular stretch) is offered as one physiological explanation for the observed decreases in SBP. Impaired arterial vasomotor tone leading to decreased peripheral vascular resistance is attributable to the observed decreases in DBP. The application of FNS activates the muscle pump via intermittent muscle contractions that produce a milking effect of the superficial and deep veins of the legs, and the presence of venous valves allows unidirectional blood flow towards the heart. The enhanced venous return increases ventricular filling and End Diastolic Volume. The net effect of this increased preload stretch is an increase in ventricular contractility with concomitant increases in stroke volume and SBP.

The findings of this study showed an increase of SBP that is consistent with those reported by Davis et al. (14), who studied the effect of FNS in reversing venous pooling induced by progressive orthostatic challenge (tilting 0°, 30°, 70°) in men with paraplegia. In the present study, the observed mean decrease of 11 mmHg appears to be comparable to that presented by Davis et al. (14). This is in contrast with the findings of Figoni et al. (13), who studied the effect of FNS with and without arm crank exercise at 0°, 30°, and 70°g tilt angles and reported no significant change in SBP. Although Davis et al. (14) and Figoni et al. (13) used the same FNS, they did not achieve a consistent result on SBP, which may reflect the differences between arm crank and tilting. In the present study, DBP showed a progressive drop during tilting, and FNS appeared to attenuate this drop in DBP. The results of Davis et al. (14) showed no significant difference in DBP between FNS and non-FNS conditions. The ability of FNS to attenuate the drop in DBP may be explained as a passive mechanical effect. The isometric

contractions induced with FNS compress muscular arteries, reducing blood flow, and thus creating a higher total peripheral resistance, which would raise the DBP. In contrast to the decreased responses seen with blood pressure, HR increased subsequent to tilting. Increased sympathetic reflex activity to maintain an acceptable cardiac output is thought to explain the inverted HR response; however, this does not seem plausible because this was observed in our acute and complete tetraplegic subjects who should have limited sympathetic drive. Increased catecholamines secretion in blood by the adrenal medulla stimulating the adrenergic receptors in the heart may be an alternative factor that contributes to the observed increase in HR (28).

The exaggerated HR response at the 60° tilt position is somewhat difficult to interpret. A possible explanation may be the accumulative metabolic effect of repeated isometric muscle contraction associated with FNS. Although hemoglobin oxygen saturation was not analyzed in this study, percent saturation was monitored for patient safety. Of interest was the observation that in all individuals, desaturation was noted at the maximum tilt position with FNS conditions. This finding suggests potential anaerobic muscle metabolism. The end products of hypoxemia (decreased PaO<sub>2</sub>), and hypercapnia (increased PaCO<sub>2</sub>) may have had led to a chronotropic increase in HR via stimulation of the central (cardiac acceleratory center of the medulla oblongata) and peripheral (carotid and aortic bodies) chemoreceptors (17).

The results of this study suggested a rebound effect supported by the significant rise in SBP and DBP, and a reflex inhibition of HR, associated with returning the body from maximum tilt to 0° of recovery. The observed rise in SBP may be due to an abolished effect of gravity. The abrupt return to the horizontal position would have augmented venous return, eliciting an immediate Starling effect with subsequent increases in blood pressure and activation of the baroreceptor reflex to decrease HR. This rebound phenomenon was also noticed by Cobert and Frankel during their study of individuals with tetraplegia when they returned them to the horizontal position from 45° tilting (29). The therapeutic benefit of this rebound effect is unknown, but it may have physiological benefits.

## CONCLUSION

In summary, the present study represents a controlled investigation of FNS and upright tilting in indi-

viduals with acute spinal cord injury. The results support the hypothesis that FNS is effective at maintaining SBP, DBP, and HR during upright test maneuvers after acute paralysis. The findings suggest a reduced venous pooling subsequent to observed attenuation of induced decreases in SBP and DBP with lessened compensatory increases in HR. These findings suggest that FNS can be used effectively to reduce orthostatic hypotension in the clinical setting for patients with acute spinal cord injury. Further research is recommended to examine the precise mechanisms contributing to cardiovascular adaptations when using FNS and to establish the long-term clinical efficacy of using FNS to reduce orthostatic hypotension in individuals with spinal cord injury.

## REFERENCES

1. Blumer C, Quine S. Prevalence of spinal cord injury: An international comparison. *Neuroepidemiology* 1995;14:258–68.
2. Anonymous. Spinal cord injury: the facts and figures. Birmingham, AL: Spinal Cord Injury Statistical Center, University of Alabama; 1986.
3. Charles H. Review of experimental spinal cord injury with emphasis on the local and systemic circulatory effects. *Neurochirurgie* 1991;37:291–302.
4. Alexander S, Kerr FWL. Blood pressure responses in acute compression of the spinal cord. *J Neurosurg* 1964;21:485–91.
5. Tibbs PA, Young G, Todd EP, McAllister RG Jr, Hubbard S. Studies of experimental cervical spinal cord transection. Part IV. Effects of cervical spinal cord transection on myocardial blood flow in anesthetized dogs. *J Neurosurg* 1980;52:197–202.
6. Figoni SF. Cardiovascular and hemodynamic responses of tetraplegic individuals to tilting and to standing: A review. *Paraplegia* 1984;22:99–109.
7. Thomas JE, Schirger A. Orthostatic hypotension: etiologic considerations, diagnosis and treatment. *Med Clin North Am* 1968;52:809–16.
8. Stumpf JL, Mitrzyk B. Management of orthostatic hypotension. *Am J Hosp Pharm* 1994;51:648–60.
9. Glaser RM. Physiologic aspects of spinal cord injury and neuromuscular stimulation. *Central Nervous System Trauma* 1986;3:49–62.
10. Davis G, Servedio F, Glaser R, et al. Hemodynamic responses during electrically-induced leg and voluntary arm crank exercise in lower-limb disabled males. Proceedings of 10th annual RESNA Conference; 1987; San Jose, California.
11. Glaser RM, Rattan SN, Davis GM. Central hemodynamic responses to lower-limb FNS. Proceedings of the 9th annual Conference of the IEEE Engineering in Medicine and Biology Society; 1987. p. 615–7.
12. Phillips W, Burkett LM, Munro R, Davis M, Pomeroy K. Relative changes in blood flow with functional electrical stimulation during exercise of paralyzed lower limbs. *Paraplegia* 1995;33:90–3.
13. Figoni SF, et al. FNS-assisted venous return in exercising SCI men. Proceedings of the International Conference of the

- Association of Advanced Rehabilitation Technology; 1988. p. 328–9.
14. Davis GM, et al. Cardiovascular responses to FNS-induced isometric leg exercise during orthostatic stress in paraplegics. Proceedings of the International Conference of the Association for Advancement of Rehabilitation Technology; 1988; Montreal, Canada. p. 326–7.
  15. Orenstein DM, et al. Accuracy of three pulse oximeters during exercise and hypoxemia in individuals with cystic fibrosis. *Chest* 104(4):1993;1187–90.
  16. Palve H. Reflection and transmission pulse oximetry during compromised peripheral perfusion. *J Clin Monit* 1992;8(1):12–5.
  17. Gyton. *Basic human physiology: normal function and mechanisms of disease*. New York: Saunders; 1995.
  18. Shields RK. Fatigability, relaxation properties, and electromyographic responses of the human paralyzed soleus muscle. *J Neurophysiol* 1995;73:2195–205.
  19. Shields RK, Law LF, Reiling B, Sass K, Wilwert J. Effects of electrically induced fatigue on the twitch and tetanus of the soleus muscle in individuals with paralysis. *J Applied Physiol* 1997;87:46–55.
  20. Shields RK, Chang YJ. Effects of fatigue on the torque frequency curve in the human paralyzed soleus muscle. *J Electromyography Kinesiol* 1997;1:1–12.
  21. Shields RK, Chang YJ, Ross M. Neuromuscular propagation after fatiguing contractions of the paralyzed soleus muscle in humans. *Muscle Nerve* 1998;21:776–87.
  22. Rowell LD. *Human circulation during physical stress*. London: Oxford University Press; 1986.
  23. Tator CH. Review of experimental spinal cord injury with emphases on the local and systemic circulatory effects. *Neurochirurgie* 1991;37:291–302.
  24. Kaplan PE, Roden W, Gilbert E, Richards L, Goldschmidt JW. Reduction of hypercalciuria in tetraplegia after weight-bearing and strengthening exercises. *Paraplegia* 1981;19:289–93.
  25. Abramson AS. Bone disturbances in injuries to the spinal cord and cauda equina (paraplegia). *J Bone Joint Surg* 1948;30A:982–7.
  26. Odeen L, Knutsson E. Evaluation of the effects of muscle stretch and weight load in individuals with spastic paralysis. *Scand Jour Rehabil Med* 1981;13:117–21.
  27. Vallbona C, Lipscomb HS, Carter RE. Endocrine responses to orthostatic hypotension in quadriplegia. *Arch Phys Med Rehabil* 1966;47:412–21.
  28. Mathias CJ, Frankel HL, Christensen NJ, Spalding JM. Enhanced pressor response to noradrenalin in individuals with cervical spinal cord transection. *Brain* 1976;99:757–70.
  29. Corbett JL, Frankel HL, Harris PJ. Cardiovascular responses to tilting in tetraplegic man. *J Physiol* 1971;215:411–31.

Submitted for publication November 18, 1999.  
Accepted in revised form January 31, 2000.