

## Effects of regular use of neuromuscular electrical stimulation on tissue health

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**Abstract**—Changes in tissue health were monitored in a group of spinal cord injury (SCI) individuals with the use of an implanted neuromuscular electrical stimulation (NMES) system to provide standing and to facilitate standing transfers. Tissue health was evaluated through monitoring tissue oxygen levels in the ischial region along with measuring interface pressures at the seating support interface. Baseline assessments were done at study enrollment and repeated on completion of a conditioning exercise program. Serial assessments of tissue health were performed on eight NMES implant recipients. Unloaded tissue oxygen levels in the ischial region tended to increase after following the NMES exercise program for 8 weeks. Concurrently, pressure distributions at the seating support interface tended to change such that although the total pressure acting at the interface did not change, ischial region pressures showed a significant decrease. These changes indicate that chronic use of NMES has a quantifiable benefit on tissue health.

**Key words:** neuromuscular electrical stimulation, spinal cord injury, tissue health.

### INTRODUCTION

One of the major consequences of complete spinal cord injury (SCI) is motor paralysis below the level of the lesion. Loss of motor control and the consequent immobility rapidly lead to disuse muscle atrophy with loss of muscle bulk, which affects pressure distributions at the support surfaces. Specifically, reduced gluteal muscle bulk will

tend to produce increased interface pressures in the region of the ischial tuberosities when individuals are sitting.

Regional blood flow will be also affected by motor paralysis. In normal functional muscle, regular muscular contractions act to facilitate regional blood flow by “pumping” blood through the vascular network. This blood pump mechanism is no longer effective below the level of the lesion in individuals with SCI. Following SCI, sympathetic nervous activity below the level of the lesion decreases, leading to decreased blood pressure [1,2]. Vascular patency is adversely affected such that the vessels are less capable of withstanding normal loading conditions and maintaining blood flow [3]. Concurrent with loss of muscle bulk, a loss of capillary network will occur, thus reducing the volume of blood that can be delivered to the tissues even under optimal conditions.

**Abbreviations:** ASIA = American Spinal Injuries Association, NMES = neuromuscular electrical stimulation,  $P^I$  = ischial interface pressure,  $P^O$  = overall interface pressure, SCI = spinal cord injury, STS = standing transfer study,  $T^O$  = unloaded tissue oxygen.

**This material was based on work supported by grants from the Rehabilitation Research and Development Service of the Department of Veterans Affairs and the Food and Drug Administration Office of Orphan Products Development.**

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Thus, SCI leads to changes in both vascular capability and capacity. These factors, along with more global considerations, such as reduced respiratory function seen in individuals with tetraplegia [6], will reduce the supply of oxygen and other nutrients to paralyzed soft tissue. The overall effect is degeneration in tissue health below the level of SCI, leading to increased risk of tissue breakdown and pressure sore development.

All individuals with SCI, and particularly those with complete lesions, are considered to be at high risk of pressure sore development throughout their lifetime. This significant secondary complication is the major cause for readmission to the hospital following primary rehabilitation. Treating pressure sores in the United States has been estimated to cost over \$1.33 billion per annum [5], primarily because of the need for prolonged periods of bed rest associated with many methods of treatment. Pressure sores tend to reduce independence and affect many aspects of daily life, such as physiological well-being, social interactions, work or college attendance, and need for caregiver time. Further medical complications may also arise, in particular, systemic infections leading to fatality. Approaches to the prevention of pressure sores in high-risk populations can generally be classified as education-focused or device-focused. However, despite the development of many support devices and the introduction of skin care training within many rehabilitation programs, the incidence of pressure sores remains unacceptably high [6]. Furthermore, there remains a significant proportion of the SCI population who exhibit chronic recurrence of tissue breakdown [7].

All device-orientated prevention techniques focus only on improving the support characteristics at the user/support interface with the seating system. Thus far, the intrinsic changes in muscle characteristics that occur because of SCI have been considered irreversible and have not been considered addressable. Neuromuscular electrical stimulation (NMES) offers a unique means to produce beneficial changes at the user/support system interface by altering the intrinsic characteristics of the user's paralyzed tissue itself.

For rehabilitation purposes, the effects of NMES on paralyzed muscle can be considered in terms of the activation of paralyzed neuromuscular units [8,9]. SCI interrupts the normal control of muscles below the lesion, leading to paralysis. Partially innervated muscles below the level of the lesion will become weak. Thus, muscles controlled by nerves at or below the lesion will be unable

to sustain prolonged contractions. A NMES exercise program can be designed to increase both the strength and the fatigue resistance of paralyzed muscles using stimulation patterns that provide repetitive maximal contractions to select muscle groups. Concurrently, muscular vascularisation will start to increase as early as 4 days after initiating low-frequency electrical stimulation. It has been shown that capillary density can triple in paralyzed muscles after 2 weeks of regular moderately intensive stimulation [10]. These changes in stimulated muscle characteristics may also improve fatigue resistance.

This study investigated the hypothesis that chronic use of NMES improves pressure distribution at the seating support area, specifically reduction of peak pressures over bony prominences because of increased muscle mass area. In addition, chronic NMES will increase vascularity, leading to improved tissue blood flow and resulting in improved regional tissue health in individuals with SCI.

## METHODS

### Subjects

All subjects followed in this study were also participating in a study using surgically implanted epimysial electrodes and a multichannel implanted stimulator for standing and transfers [11]. Gluteal muscle electrodes were implanted bilaterally as part of this system. Primary exclusion and inclusion criteria for this study are as follows:

- Inclusion Criteria
  - Low cervical or thoracic level SCI (C6–T12).
  - More than 6 months postinjury.
  - Skeletal and cognitive maturity.
  - Upper motor neuron injury.
  - ASIA (American Spinal Injuries Association) Impairment Scale: Low-cervical/high-thoracic injuries (C6–T4): A, B, or C and mid- to low-thoracic injuries (T4–T12): A or B.
- Exclusion Criteria
  - Cardiac arrhythmia or pacemaker fitted.
  - Acute orthopedic problems, e.g., scoliosis, hip dislocation.
  - Acute medical complications, e.g., cardiac abnormalities, uncontrolled seizures, systemic compromise.
  - Frequent urinary tract infections.
  - Current open pressure sores.

- Immunodeficiency.
- Acute chronic psychological problems or chemical dependency.
- Seizure disorder.
- Pregnancy.

### Interventions and Stimulation Regimes

Following implantation of the standing and transfer neuroprosthesis, subjects were required to remain on bed rest for approximately 1 week. We reexamined subjects at 6 weeks postimplantation to obtain a profile of the neuroprosthesis electrode properties. They then commenced an exercise regime for a minimum of 8 weeks for muscle strengthening before using the system for standing. The exercise regime included three different stimulation patterns as described in **Table 1**. The duration and modes of exercise were varied over the 8-week training period as the muscles became conditioned. The training protocol is summarized in **Table 2**.

### Data Collection

The study design was based on pre- and posttreatment measures with the subjects acting as their own controls. Baseline assessment was done after the subject had been enrolled in the standing transfer study (STS) before implantation of the stimulation system and/or before

commencing any routine stimulation regime. Once the subject commenced the 8-week conditioning exercise regime, he or she was asked to maintain a log of the stimulation patterns used each day and the times for which they exercised. At the end of the exercise regime, the assessment procedure was repeated. The assessment methodology characterized tissue health variables using objective measurement techniques.

We assessed the effects of NMES exercise on tissue blood flow in the gluteal region through measurements of local transcutaneous oxygen levels ( $T_cPO_2$ ) using a Radiometer TCM3-2 monitor (Radiometer America Inc., Westlake, Ohio). The oxygen electrode (Radiometer, model E5280-8) was calibrated with a gas containing 20.9 percent oxygen and 5 percent carbon dioxide in nitrogen. The temperature control of the system was set at 43 °C, to produce maximal local vasodilation. The variation in room temperature over the course of each assessment was maintained at  $25 \pm 2$  °C throughout each assessment. The transcutaneous gas electrode was placed with the subject in a side-lying position on a bed or plinth, with hips and knees flexed to 90° to approximate the angulation of the lower limbs in sitting. The left or right ischial tuberosity was randomly selected at baseline assessment for regional tissue gas monitoring and was also selected at repeat assessment. The ischial tuberosity

**Table 1.**  
Conditioning stimulation patterns.

Pattern	Muscles Stimulated	Frequency (Hz)	Duty Cycle				Sequence	User Posture	Objective
			Ramp Up (s)	On (s)	Ramp Down (s)	Off (s)			
A: Leg Lifts	Vastus lateralis (bilateral)	30	2	5	4	10	Alternating	Supine	Strengthening of knee extensors
B: Hips and Backs	Phase I: gluteus maximus and hamstrings (bilateral) Phase II: erector spinae (bilateral)	30	2	5	2	10	Alternating: Phase I/ Phase II	Supine	Strengthening of hip and trunk muscles
C: Full Extension Pattern	Vastus lateralis, hamstrings, gluteus maximus and erector spinae (bilateral)	16	2	20	2	10	Simultaneous	Supine (legs straight)	Endurance training

Ramp up = Increase in stimulation from baseline to saturation.

Ramp down = Decrease in stimulation from saturation to baseline.

Saturation = Point at which no further increase in force is seen with further increase in pulse duration (width). Typically occurs at a pulse width of 150 s to 200 s in muscles with no spillover.

was then palpated and a fixation ring was located over the

**Table 2.**

Training protocol for functional electrical standing system.

Week	Exercise Pattern	Mode	User Posture	Duration
1	A	Unresisted isotonic	Supine, legs straight	1 set of 10
	B	Unresisted isometric	Supine, legs straight	1 set of 10
	C	Unresisted isometric	Supine, legs straight	15 min–30 min
2	A	Unresisted isometric	Supine, knees flexed over bolster	2 sets of 10
	B	Unresisted isometric	Supine, legs straight	2 sets of 10
	C	Unresisted isometric	Supine, legs straight	45 min–60 min
3, 4	A	Resisted isotonic (weights)	Supine, knees flexed over bolster	3 sets of 10 (5 min intervals)
	B	Resisted isotonic (body weight)	Supine, knees flexed over bolster	3 sets of 10 (5 min intervals)
	C	Unresisted isometric	Supine, legs straight	60 min
5, 6	A	Resisted isotonic (increased weights)	Supine, knees flexed over bolster	3 sets of 10 (5 min intervals)
	B	Resisted isotonic (body weight)	Supine, knees flexed over bolster	3 sets of 10 (5 min intervals)
	C	Unresisted isometric	Supine, legs straight	60 min
7, 8	A	Resisted isotonic (increased weights)	Supine, knees flexed over bolster	3 sets of 10 (5 min intervals)
	B	Resisted isotonic (body weight)	Supine, knees flexed over bolster	3 sets of 10 (5 min intervals)
	C	Unresisted isometric	Supine, legs straight	120 min

bony landmark. The fixation ring consists of a 20 mm diameter adhesive ring surrounding a central plastic screw mounting. The electrode mounting is approximately 10 mm in diameter and 6 mm high. The central region of the ring was filled with contact fluid and the electrode securely attached. With the electrode and underlying soft tissue, a 20 min equilibration period unloaded then followed so that local vasodilation could stabilize before the monitoring of tissue gas levels commenced. Following equilibration, tissue oxygen ( $T_cPO_2$ ) levels in unloaded tissue were monitored for a 5 min period.

We monitored the effects of NMES exercise on interface pressure distributions at the subject/support interface using an Advanced Clinseat System (Tekscan Inc., Boston, Massachusetts). The interface pressure mat uses conductive and semiconductive inks in a thin, flexible grid-based sensor array. The array contains more than 2,000 sensors and operates within the range 1 to 250 mmHg (accuracy +10%) at the scanning rate of 125 Hz. Real-time three-dimensional (3-D) images of pressure distribution at the seating interface are produced with the use of graphical display software. The system analysis can also determine peak regional pressures, center of pressure, and contact area.

After we completed the unloaded tissue gas measurement, the subjects were transferred carefully to their wheelchairs to begin the seated assessment. All seated assessments were completed with the subjects sitting in their usual seating system, i.e., using their own wheelchair and support cushion.

Before transfer, the interface pressure sensor array was placed over the subject's cushion in the wheelchair. We monitored interface pressures by scanning the subject/support interface at 2 frames/s (each frame is a complete scan of the seating interface) for 200 s periods. An initial data set was recorded once the patient had stabilized in the seated position and the regional soft tissues had acclimated to the applied loads, after 3 to 5 min (prestimulation). A dynamic stimulation pattern was then applied to provide active side-to-side weight shifting for 5 min. Following dynamic stimulation, a poststimulation interface pressure data set with no stimulation was recorded.

## RESULTS

To date, repeat tissue health assessments have been completed for eight subjects participating in the STS.

Clinical and personal details for this subject group are summarized in **Table 3**.

### Electrical Stimulation Conditioning of Gluteal Muscles

Electrical stimulation of the gluteal muscles was achieved during conditioning exercise patterns B (hips and backs) and C (endurance training). Keeping an exercise log was presented as being optional to study participants. Exercise data were recorded for 37 weeks by the four study participants who returned any exercise log. We compared the stimulation patterns and times recorded in the exercise logs with the prescribed stimulation protocol to establish a measure of exercise compliance as reported by the subjects. Successful attainment of the exercise protocol for the week was defined as 95 percent or greater of the required stimulation pattern usage. We found that study participants attained 86 percent compliance for stimulation pattern B (hips and backs) but only 27 percent compliance for stimulation pattern C (endurance training). Median total gluteal stimulation (with the use of stimulation patterns B and C) was 68.3 hr (range: 24.0 to 75.5 hr), with an average ratio of 1:2 B:C stimulation pattern usage.

### Changes in Tissue Health Variables Following Conditioning Protocol

Baseline mean unloaded tissue oxygen levels ( $T^{\text{O}}$ ) were found to have increased by 1 to 36 percent at post-exercise assessment for five subjects; however, three subjects showed some decrease. Therefore, the difference between baseline and postexercise  $T^{\text{O}}$  did not show any statistical significance (**Table 4**).

We determined mean interface pressure values by averaging over all frames for pre- and poststimulation data sets taken at the same assessment. The mean overall seating interface pressure ( $P^{\text{O}}$ ) was determined as the mean pressure over all active sensors (i.e., pressure greater than 0 mmHg). To determine mean ischial interface pressures, we identified the peak pressures under each ischial tuberosity and defined a surrounding area approximately 12 cells  $\times$  12 cells as the ischial pressure region. The reference points for each ischial pressure region were determined for the prestimulation data set and then applied to the poststimulation data set collected at the same assessment. Mean ischial interface pressure ( $P^{\text{I}}$ ) was then determined as the mean interface pressure within each ischial pressure region for each frame within a data set, averaged over all frames. Initial data analysis showed no significant statistical differences between left and right ischial pressure and therefore bilateral values were averaged for each subject.

We found that overall mean interface pressure showed no significant differences between baseline and postexercise levels (**Table 5**). However, mean ischial region interface pressures had a uniform tendency to decrease at postexercise assessment (**Table 5**), leading to a statistically significant difference at  $p < 0.01$ .

## DISCUSSION

The current study seeks to evaluate the effects of electrical stimulation on tissue health through measures that indirectly indicate the likelihood of tissue breakdown. For

**Table 3.**  
Clinical and personal subject profiles.

Subject	Gender	Age at Entrance to Study	Months Postinjury	Injury Level	ASIA* score	Body Mass (kg)	Height (m)	Body Mass Index
1	M	46	93	T6	A	91.63	1.75	29.83
2	M	37	46	T4	A	113.40	1.88	32.10
3	M	47	15	T6	A	86.18	1.73	28.89
4	F	28	20	C7	B	56.70	1.68	20.18
5	M	35	204	T5	B	89.81	1.75	29.24
6	M	28	27	T9	A	49.89	1.65	18.30
7	M	41	106	C5/6	A	68.04	1.75	22.15
8	M	27	13	T8	A	99.79	1.85	29.03

\*ASIA score = American Spinal Injuries Association score

**Table 4.**

Changes in mean tissue oxygen level in unloaded ischial tissue following conditioning exercise with NMES.

Subject	Baseline (mmHg)	Postconditioning (mmHg)	$\Delta$ (%) <sup>*</sup>
1	46.3	63.0	36
2	44.5	57.4	29
3	52.5	55.0	5
4	98.6	92.9	-6
5	55.9	43.7	-22
6	80.2	88.5	10
7	78.9	79.8	1
8	78.2	55.8	-29

<sup>\*</sup>Differences in values are nonsignificant.

example, a low regional tissue oxygen level implies that soft tissues are receiving an inadequate supply of blood, thus placing them in a compromised state and at an increased risk of local cell death, a precursor of tissue breakdown.

Long-term use of electrical stimulation has been reported anecdotally to have a positive effect on tissue health. Levine et al. reported that the effects of stimulating the gluteal muscle using surface stimulation in able-bodied subjects included a redistribution of interface pressures away from the ischial region while the muscle was being actively stimulated, together with an increase in local blood flow [12,13]. However, these feasibility studies focused on using surface stimulation for able-bodied subjects because the researchers wished to avoid the complication of conditioning atrophied paralyzed

muscle before testing. The focus of the current study was to evaluate the effects of conditioning paralyzed muscle in a group of SCI subjects by long-term use of NMES. The results obtained indicate that positive changes in tissue health tend to occur over time with regular chronic stimulation.

Regional tissue oxygen ( $T_cPO_2$ ) levels tended to increase for most study participants, which implies that regional vascularisation was increasing with regular application of NMES. It is noted, however, that a direct correlation between percentage increase in  $T_cPO_2$  and self-reported exercise time could not be demonstrated.

The total pressure acting through the seating support area did not appear to alter with regular use of NMES; however, the levels in the high-pressure areas around the ischial tuberosities did decrease significantly. Taken together, these results imply that the conditioning NMES protocol does not cause an alteration in the total force acting through the seating support area, but it does affect the distribution profile at the seating support area. We propose that these changes occur because of hypertrophy in the stimulated muscle, leading to improved "cushioning" over the ischia and thus a more even overall pressure distribution.

## CONCLUSIONS

These early findings appear to support the hypothesis that long-term use of NMES using implanted systems can improve the regional health of paralyzed muscle by increasing regional blood flow and improving interface

**Table 5.**

Changes in mean interface pressures following conditioning exercise with NMES.

Subject	Overall (mmHg)			Ischial (mmHg)		
	Baseline	Postconditioning	$\Delta$ (%) <sup>*</sup>	Baseline	Postconditioning	$\Delta$ (%) <sup>†</sup>
1	38.55	37.77	-2	68.27	67.26	-1
2	51.93	45.43	-13	92.73	79.89	-14
3	34.37	36.91	7	60.33	59.22	-2
4	26.62	27.37	3	68.34	53.83	-21
5	46.23	40.71	-12	94.62	78.47	-17
6	26.40	25.64	-3	55.03	53.61	-3
7	28.20	29.46	4	68.00	66.57	-2
8	41.78	42.79	2	76.51	66.26	-13

<sup>\*</sup>Differences in values are nonsignificant.

<sup>†</sup>All values were significant at  $p < 0.01$ .

pressure distribution. These changes can reduce the risk of pressure sore development. The participants in the current study who received the implanted standing system were required to have no open pressure sores to be eligible for the primary study. These individuals exhibited reduced tissue health because of their spinal cord but could be considered to be at the lower end of the tissue compromise risk spectrum for this patient group. To make the effects of NMES more consistent, further study of additional subjects is required to (1) determine the natural variability of tissue health within the SCI population and (2) determine the factors that contribute to intersubject variability.

## ACKNOWLEDGMENT

NeuroControl Corporation provided implanted stimulation systems.

## REFERENCES

1. Mathias CJ, Frankel HL. Autonomic disturbances in spinal cord lesions. In: Bannister R, Mathias CJ, editors. *Autonomic failure*. 3rd ed. Oxford: Oxford University Press; 1992. p. 839–81.
2. Mathias CJ, Frankel HL. The cardiovascular system in tetraplegia and paraplegia. In: Frankel HL, editor. *Spinal cord trauma, Handbook of clinical neurology*. Amsterdam: Elsevier Science Publishers; 1992. p. 435–56.
3. Schubert V, Fagrell B. Postocclusive reactive hyperemia and thermal response in the skin microcirculation of subjects with spinal cord injury. *Scan J Rehabil Med* 1991; 23:33–40.
4. Linn WS, Adkins RH, Gong H, Waters RL. Pulmonary function in chronic spinal cord injury: a cross-sectional survey of 222 southern California adult outpatients. *Arch Phys Med Rehabil* 2000;81:757–63.
5. Graitcer PL, Maynard FM, editors. *Proceeding of the First Colloquium on preventing secondary disabilities among people with spinal cord injury*. Atlanta, Georgia: U.S. Department Health and Human Services, Centers for Disease Control; 1990. p. 119.
6. Yarkony GM, Heinemann AW. Pressure ulcers. In: Stover SL, DeLisa JA, Whiteneck GG, editors. *Spinal cord injury: Clinical outcomes from the model systems*. Gaithersburg, Maryland; 1995. p. 100–119.
7. Niazi ZB, Salzberg CA, Byrne DW, Viehbeck M. Recurrence of initial pressure ulcer in persons with spinal cord injuries. *Adv Wound Care* 1997;10:38–42.
8. Kralj A, Vodovnik L. Functional electrical stimulation of the extremities. *J Med Eng Tech* 1977;1(Pt 1):12–15.
9. Benton LA, Baker LL, Bowman BR, Waters RL. *Functional electrical stimulation—a practical clinical guide*. Rancho Los Amigos, Rehabilitation Engineering Center; 1980.
10. Hudlicka O, Brown M, Cotter M, Smith M, Vrbova G. The effect of long-term stimulation of fast muscles on their blood flow, metabolism, and ability to withstand fatigue. *Pflugers Arch* 1977;369:141–49.
11. Davis JA, Triolo RJ, Uhlir JP, Bhadra N, Lissy DA, Nandurkar S, Marsolais EB. Surgical technique for installing an eight-channel neuroprosthesis for standing. *Clin Orthop* 2001;385:237–52.
12. Levine SP, Kett RL, Cederna PS, Bowers LD, Brooks SV. Electrical muscle stimulation for pressure variation at the seating interface. *J Rehabil Res Dev* 1989;26:1–8.
13. Levine SP, Kett RL, Gross MD, Wilson BA, Cederna PS, Juni JE. Blood flow in the gluteus maximus of seated individuals during electrical muscle stimulation. *Arch Phys Med Rehabil* 1990;71:682–86.

Submitted for publication March 5, 2002. Accepted in revised form June 23, 2003.

