Electrical muscle stimulation for pressure variation at the seating interface

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Abstract—A new method is proposed for pressure sore prevention using electrical muscle stimulation (EMS). Potential mechanisms through which EMS may act for this purpose are discussed, including both short-term/dynamic and chronic effects. Measurements of maximum pressure variation in three able-bodied subjects using low levels of stimulation were performed. Pressure distribution changes were also measured. Fatigue effects on pressure redistribution were studied for four able-bodied subjects as well as for one C4, complete spinal cord injured individual. The results indicate that EMS produces sizeable pressure reduction under the ischial tuberosity, with redistribution occurring over other parts of the seating surface in able-bodied subjects. Fatigue effects were not observed in the four able-bodied subjects even after prolonged stimulation. Fatigue was observed with the spinal cord injured subject, but only after extensive stimulation. These studies demonstrate the feasibility of using EMS at relatively low intensity to vary seating interface pressure. The results warrant continued investigation of EMS to assist in pressure sore prevention.

Key words: electrical muscle stimulation, muscle fatigue, pressure sores, seating interface pressure, spinal cord injury.

INTRODUCTION

Impact and cost

Many physically disabled individuals are subjected to sustained external forces which cause skin ulceration. These ulcerations (e.g., decubitus ulcers, ischemic ulcers, bed sores, etc.) can be collectively referred to as “pressure sores.” Paralyzed individuals who are wheelchair-dependent and have sensory loss are especially at risk for pressure sores. The most common location of these sores is over the ischial tuberosities, where interface forces tend to be the greatest (7,8,12,27).

The medical, social, and financial costs of pressure sores are dramatic. In one study covering the period from 1973 to 1978, 60 percent of patients with complete cervical cord injuries, 40 percent of patients with incomplete cervical injuries, and more than 50 percent of patients with complete thoracic and lumbar injuries developed pressure sores (35). Another retrospective study covering 1975-1980 reported an incidence per year for pressure sores of all grades for motor and sensory quadriplegics of 40 to
45 percent (37). Krouskop estimated a cost of $20,000 to $30,000 per incident in 1982 dollars, and an annual price tag of two billion dollars for medical care costs associated with pressure sores in the USA (22).

Beyond these costs are the effects of pressure sores on the lives of handicapped people: time lost from work, school, and family; psychological impact; and loss of general independence and productivity that can contribute to lowered self-esteem and self-worth (22). The prevalence and cost of pressure sores warrant the investigation of new methods to prevent their formation.

Etiology and prevention

Tissue ischemia is considered by some to be the primary cause of pressure sores (22,28). Associated tissue responses induced by external forces and contributing to pressure sore formation include: restriction of lymph flow, changes in metabolic conditions, local tissue integrity and viability, and edema (12,22,31,32). Additional intrinsic and extrinsic factors relating to pressure sore formation include the magnitude of applied force (2,34), force direction (i.e., normal versus shear) (1,5), duration of applied force (2,20,34), friction (6), tissue hygiene, circulatory health, nutritional health, moisture, and temperature.

A number of investigators have tried to determine critical levels of pressure and duration for pressure sore formation (19,20,26,34). This information has led to the development of a variety of wheelchair cushions to help prevent pressure sores. These cushions attempt to lower pressures in areas that have bony prominences, redistribute interface pressures more evenly over a wider area, and reduce pressure gradients in the tissue (9,12). Many different kinds of wheelchair cushions have been evaluated for their effectiveness in reducing ischial pressures for pressure sore prevention (12,14,19,28). These studies have generally concluded that seating cushions alone do not permit adequate pressure relief for continuous sitting.

Rehabilitation professionals regularly teach wheelchair users (especially those with sensory loss) to perform wheelchair push-ups, side leans, and front-to-back rocking. Studies have shown that intermittent pressure relief of tissues allows increased pressure tolerance (9,20,21,22,26,34), although the effectiveness of pressure relief relies on subject compliance (22). An alternate approach utilizes mechanical or pneumatic alteration of the seating surface to provide periodic pressure relief (14). The limited effectiveness of the various approaches to pressure sore prevention suggests that alternative approaches need to be further investigated. A new method which we have proposed for pressure sore prevention (17,23) utilizes electrical muscle stimulation (EMS).

Rationale

EMS may prevent pressure sores through several different mechanisms. These mechanisms can be grouped into two major categories: short-term/dynamic effects and chronic effects.

Effects of short-term/dynamic use of EMS. Short-term/dynamic effects of EMS in preventing the development of pressure sores may include the following: 1) Pressure variation (tissue undulation) provides a dynamic method for “pressure relief.” In terms of an ischemia and reduced lymph flow model of pressure sore formation, such pressure relief could increase blood and lymph flow to tissues, allowing higher oxygen delivery rates and metabolite removal. 2) Both voluntary and evoked muscle contractions can actively increase blood (and lymph) flow with contraction levels below 30 percent of maximum (10,36). Increased blood and lymph flow result from muscle pump activity during exercise, while local vasodilation mechanisms can lead to increased blood flow both during and after exercise.

Effects of chronic use of EMS. Chronic use of EMS can increase: capillary density and vascularization in muscle (4,15); fatigue resistance and aerobic capacity of muscle (15,30); and muscle strength and bulk (13,29,30). It has been suggested that decreased tissue bulk alone may significantly contribute to pressure sore formation (11). These results support the hypothesis that chronic EMS can aid in pressure sore prevention both alone and in conjunction with the short-term/dynamic effects of EMS.

The development of stimulation parameters to realize the short-term/dynamic and chronic effects of EMS must reflect consideration of possible negative effects of EMS. These include increased muscle fatigue, intramuscular pressure, muscle oxygen requirements, vasoconstriction, metabolite production, heat, and sweat.
This paper introduces the concept of using EMS for pressure sore prevention. The studies described in this paper were undertaken in an attempt to investigate the feasibility of EMS to produce sizeable interface pressure variations, and to develop a general estimate of their magnitude. Able-bodied subjects were chosen for initial investigation to avoid the complication of having to condition paralyzed, atrophied muscle prior to testing. It is hypothesized that the results should carry over to paralyzed individuals who have undergone appropriate conditioning of paralyzed muscle (30), or have maintained adequate muscle strength and bulk due to residual spasticity.

METHODS

Experiment 1: Maximum pressure variation

Bilateral stimulation of the gluteus maximus was performed on three able-bodied subjects while seated in a wheelchair. Subjects wore sweat pants and were seated in a standard position defined as: 1) Backrest-to-seat angle of at least 80 degrees; 2) A minimum 2-inch clearance from the popliteal fossa to the forward edge of the wheelchair; and, 3) Footrest adjusted to keep the thighs parallel to the seat and floor.

Stimulation was provided through 2 x 4 inch silicone rubber surface electrodes using a dual channel neuromuscular stimulator. Controllable parameters of the stimulator include current amplitude, pulse rate, and duty cycle (“on” and “off” times). The stimulator was current-controlled with ramped pulses (300 µsec duration) and an asymmetric biphasic waveform for charge balance. Cathodes were positioned bilaterally over the gluteus maximus, each approximately one-third of the distance from the sacrum to the greater trochanter, cephalic to the sitting surface. Specific location was determined through monopolar stimulation and visual inspection for maximum contraction. A common anode was placed over the sacrum.

Stimulation intensity was set with the subject in the defined standard seating position. With the frequency set at 3 Hz, the intensity was adjusted to elicit approximately a 1-inch mediolateral movement of the knee. This level of stimulation was easily tolerated by all sensate subjects.

A Scimedics pressure evaluator pad was initially used to measure seating interface pressures. This system measures pressure based on the pneumatic pressure inside a 3-inch diameter plastic pad (balloon). When the external pressure exceeds the internal pad pressure, a light sensor is activated. The interface pressure is taken as the pressure reading at sensor activation. The pad pressure can be adjusted between 0 and 250 mm Hg.

(A) Maximum pressure variation with varying stimulation frequency. A series of trials was conducted to determine maximum pressure variations produced with EMS. For these trials, the Scimedics pad was secured beneath one ischial tuberosity. The interface pressure at that point was measured at rest, and then during the stimulation. The difference between the two pressures was recorded as the interface pressure change induced by EMS. These trials were performed at each of five different stimulation frequencies, as shown in Table 1 for two of the three subjects. Stimulation cycle time was reduced as stimulation frequency increased. During these trials, subjects were seated on a sling seat with stimulation cycled for a 15-second period, followed by two minutes of rest, during which pressure relief was performed. The entire set of trials was repeated twice more, with the maximum pressure change averaged over all three trials.

(B) Maximum pressure variation with different seating surfaces. The effects of different seating surfaces on EMS-induced pressure variation was investigated for all three subjects. Stimulation rate was set at 50 Hz with a cycle on time of 2 sec, and a cycle off time of 10 sec. The protocol was otherwise identical to that previously described. The four seating surfaces investigated were: sling seat, 1-inch

Table 1.

<table>
<thead>
<tr>
<th>Stimulation Rate (/sec)</th>
<th>Cycle On (sec)</th>
<th>Cycle Off (sec)</th>
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</thead>
<tbody>
<tr>
<td>3</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
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</tr>
<tr>
<td>30</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>50</td>
<td>2</td>
<td>10</td>
</tr>
</tbody>
</table>
gel pad, Temper foam, and a high-profile ROHO cushion.

**Experiment 2: Pressure distribution studies**

The short-term/dynamic effects of EMS were studied using the Texas Interface Pressure Evaluator (TIPE) pad. The TIPE pad is 15 inches square and consists of a 12 x 12 matrix of activation switches. Pad pressure can be adjusted between 0 and 100 mm Hg. Each switch can be opened or closed independently. A closed switch indicates that the external normal pressure at that point is greater than the internal pad pressure; an open switch indicates an external pressure less than the pad pressure. A computer interface for the TIPE pad has been developed within our laboratory (16). A two-dimensional array displays the status of each switch on a computer terminal. The interface allows TIPE pad output to be sampled at a rate of 30 times/sec and stored on disk for later analysis. Off-line analysis allows replay of the data over a range of speeds from 1/5 to 5 times real time and calculation of the number of switch transitions during any defined segment of time. This system was used to dynamically study the effects of stimulation on the seating interface pressure. Both pressure redistribution and fatigue measurements were performed as described below.

**Pressure redistribution.** Pressure distribution measurements were performed on the two able-bodied subjects who participated in Experiment 1A. Electrode placement and stimulation settings were as before. Subjects were seated in the standard position on a 1-inch medium density polyurethane foam pad over a hard seat. Stimulation of 4 sec on/4 sec off was given at 50 Hz to observe dynamic pressure changes with stimulation. TIPE pad data was collected for four different interface pressures during both rest and stimulation.

**Fatigue measurements.** Fatigue studies were performed on four able-bodied subjects (including the three subjects who participated in Experiment 1 and one additional subject) and one C4 sensory- and motor-complete quadriplegic (2 years post-injury) in separate trials. Stimulation at 50 Hz was continually delivered with a cycle time of 4 sec on/4 sec off over a 30-minute period. Fatigue was evidenced by a reduction in the number of switch transitions from closed to open, or vice versa, between duty cycles. (A reduced number of switch transitions signifies a smaller contraction level and, therefore, increased fatigue.)

**RESULTS**

**Experiment 1: Maximum pressure changes**

Table 2 summarizes the maximum interface pressure changes produced through EMS at the various stimulation parameters. The results show that increasing stimulation rates produced increased interface pressure changes, as would be expected. Table 3 summarizes maximum interface pressure variations for each of the three subjects on different seating surfaces.

**Experiment 2(A): Pressure redistribution**

Two samples of the results from Experiment 2 are shown in Figure 1a and Figure 1b. In general, the switches that were closed without stimulation were located beneath the ischial tuberosities (i.e., the highest pressures were under the ischial tuberosities). Stimulation caused many switch transitions in areas surrounding the ischial tuberosities, signifying a redistribution of interface pressure. An increased number of switch closures was seen during stimulation for all subjects. Since the total load was constant, this implies that pressure under the ischial tuberosities was reduced during stimulation as the pressure at surrounding points increased. Follow-up pressure measurements made while varying air pressures in the pad verified that there was a reduction of interface pressure directly under the ischial tuberosities, with an increase in pressure over the

<table>
<thead>
<tr>
<th>Rate (/sec)</th>
<th>Mean Maximum Pressure Change (mm Hg)</th>
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<tbody>
<tr>
<td></td>
<td>Subject A</td>
</tr>
<tr>
<td>3</td>
<td>2.40 ± 1.09</td>
</tr>
<tr>
<td>5</td>
<td>2.64 ± 1.22</td>
</tr>
<tr>
<td>10</td>
<td>3.52 ± 1.36</td>
</tr>
<tr>
<td>30</td>
<td>5.68 ± 2.07</td>
</tr>
<tr>
<td>50</td>
<td>9.44 ± 1.42</td>
</tr>
</tbody>
</table>
Table 3.
Maximum pressure changes with various seating surfaces.

<table>
<thead>
<tr>
<th>Seating Surface</th>
<th>Subject A</th>
<th>Subject B</th>
<th>Subject C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sling Seat</td>
<td>9.44 ± 1.42</td>
<td>19.22 ± 4.26</td>
<td>15.78 ± 3.67</td>
</tr>
<tr>
<td>1&quot; Gel Pad</td>
<td>5.00 ± 1.10</td>
<td>15.63 ± 4.90</td>
<td>8.17 ± 2.93</td>
</tr>
<tr>
<td>Temper Foam</td>
<td>2.67 ± 1.15</td>
<td>10.67 ± 2.31</td>
<td>6.33 ± 1.75</td>
</tr>
<tr>
<td>High Profile ROHO</td>
<td>4.17 ± 0.98</td>
<td>11.67 ± 6.22</td>
<td>10.17 ± 3.37</td>
</tr>
</tbody>
</table>

surrounding area. This result is consistent with Experiment 1 results.

Experiment 2(B): Fatigue measurements

Constant stimulation for 30 minutes did not measurably affect the number of switch closures or openings with stimulation for able-bodied subjects. An example of a plot of the number of switch transitions with stimulation versus time is shown in Figure 2a. The number of transitions showed no significant (p<0.05) statistical correlations with time for any of the four able-bodied subjects. A plot of the pad switch transitions versus time for the quadriplegic subject is shown in Figure 2b. Fatigue was clearly evident in that the number of switch transitions gradually reduced over twenty minutes, after which no measurable changes were produced at the pre-set stimulation intensity. Electrode positioning was evaluated and discounted as the cause of the reduced response.

DISCUSSION

The pressure changes measured in these studies show that EMS to the gluteus maximus is able to alter the seating surface pressure in able-bodied subjects. Results from the maximum pressure measurement under the ischial tuberosities consistently showed reduced pressures during stimulation using the Scimedics pad. The magnitude of the pressure change under the ischial tuberosities varied, depending on the compliance of the seating surface. Pressure changes were maximized with the standard sling seat, and minimized with the ROHO cushion (the most compliant of the seating surfaces tested).

It is interesting that Subject A, who showed the smallest magnitude pressure changes during testing, had a substantially greater percentage of buttock adipose tissue. The increased compliance of such tissue may, in part, be responsible for the reduced magnitude of the observed pressure change as compared to the other two subjects.

The results using the TIPE pad are even more revealing. For example, at 50 mm Hg, 11 additional switches were activated during stimulation. This showed that the forces at the seating interface were being redistributed. Measurements made while varying the pad inflation pressure showed that this redistribution included a reduction of pressure directly under the ischial tuberosity and increased pressure in the surrounding region.

![Figure 1.](image)

Representative pad switch maps showing closed switches (darkened squares) with and without electrical stimulation. (a) Subject A, pad pressure 50 mm Hg. (b) Subject B, pad pressure 45 mm Hg.
Able-bodied subjects showed no measurable fatigue at stimulation levels and duration many times greater than would be applied clinically for pressure sore prevention. The quadriplegic subject showed total fatigue only after 20 minutes of 4 sec on/4 sec off, 50 Hz stimulation. Significant reduction of the stimulation frequency and duty cycle compared to that used in the fatigue study would be expected in any clinical EMS protocol for pressure sore prevention, where only intermittent cycled stimulation with relatively long periods of rest in between would be used. Thus, the endurance displayed by the one quadriplegic subject tested might be adequate for a clinical EMS regimen. Additionally, for individuals with atrophied muscles, the chronic application of EMS for muscle conditioning should help improve force output and fatigue resistance.

Pressure measurements have appreciable inherent error in quantifying absolute pressure (33). The Scimedics pad was particularly difficult to use because small shifts in the subject’s seating position could produce large changes in the pressure measurement. Additionally, it only provided averaged information about one area on the seating surface. The TIPE pad was an improvement in that it provided information regarding pressure distribution changes over the entire seating surface. This also allowed for easier recognition and correction of shifts in seating position. While the absolute magni-

![Figure 2](image_url)

Figure 2.
Number of pad switch transitions over time with 50 Hz, 4 sec on/4 sec off stimulation of the gluteus maximus. (a) Able-bodied subject. (b) Subject with C4 sensory- and motor-complete quadriplegia.
tude of pressure (which was not used in this study) may be inaccurately measured using these pads, such measurements provide valuable information regarding magnitude and direction of pressure changes produced using EMS within single trials sessions.

The measurement of pressure magnitude and distribution changes reported here are limited to able-bodied subjects. It has been postulated in the rationale that the results should extend to paralyzed individuals with appropriately conditioned muscle. Some paralyzed individuals (such as the one subject studied in Experiment 2) may have enough residual muscle function to produce adequate pressure variation for an EMS regimen employed to help prevent pressure sores. Others, however, would require chronic stimulation to improve muscle contraction properties to the point of usefulness (30). It is worth noting that the stimulation intensity had to be set considerably higher for the paralyzed subject, as compared to able-bodied subjects, in order to obtain approximately equal contraction levels. This is a probable indicator of existing muscle deconditioning and atrophy.

There are additional differences in tissue properties and circulatory function between able-bodied and paralyzed individuals (11,22) which may affect the capability of EMS to produce pressure variation, tissue undulation, and increased blood flow. Ongoing work includes larger scale studies with paralyzed individuals to further investigate the efficacy of EMS for pressure sore prevention. Future reports of trials with paralyzed subjects will include the effects of EMS on tissue shape variations (25) as well as muscle (26) and skin blood flow.

CONCLUSION

These studies demonstrate the feasibility of using EMS to vary seating interface pressures at relatively low stimulation levels (causing no discomfort and producing only small movements of the seated individual) in able-bodied subjects. The results provide a basis for further investigation of EMS for pressure sore prevention.

REFERENCES