Pressure ulcers are typically associated with individuals of compromised mobility, the elderly, and people with spinal cord injuries. A pressure ulcer is any lesion caused by unrelieved pressure resulting in damage of underlying tissue involving the skin, fat, fascia, muscle, and bone.1,2

Pressure ulcers develop following a prolonged period of compression of the tissue between a bony prominence and a surface, which causes the occlusion of capillaries and leads to ischemia. According to Grey et al,3 an external pressure of 50 mmHg may rise to 200 mmHg at a bony prominence. The described mechanical forces are much higher than the physiological (32 mmHg) capillary pressure. Continuous compression (more than 2-3 hours) can reduce blood flow, which causes ischemia. The ischemic state causes tissue necrosis, which ultimately leads to pressure ulcer formation.4,5

Significant etiological factors in pressure ulcers include paralysis and sen-

Effect of High Voltage Monophasic Stimulation on Pressure Ulcer Healing: Results From a Randomized Controlled Trial

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Abstract: Objective. To investigate the effect of high voltage monophasic stimulation (HVMS) on pressure ulcer healing. Methods. Fifty-eight patients with pressure ulcers were included and ultimately allocated into two comparative groups. Patients in group A and B were treated pharmacologically (a local bath of potassium permanganate, compresses of fibrolan, colistin, iruxol, and wet dressings containing 10% sodium chloride). Patients in group A were additionally treated with HVMS (100 µs, 100 Hz, 100 V) once daily, five times a week for 6 weeks. Results. The relative changes in total surface area were (85.38% in group A versus 40.08% in group B); length (71.22% in group A versus 30.38% in group B); width (76.09% in group A versus 32.48% in group B); and volume (20.69% in group A versus 9.39% in group B). The Gilman Index (0.64 cm in group A versus 0.28 cm in group B) indicated a difference in favor of group A (P ≤ 0.001). More efficient decrease of pus and greater granulation growth were observed in group A. Conclusion. HVMS appears to be a promising and useful treatment modality for pressure ulcers.
sory loss, soft tissue atrophy, and skin maceration attributable to incontinence. Other contributing factors include advanced age, poor diet, smoking, anemia, vitamin deficiency, and hypoproteinemia. Depending on the depth of tissue injury, pressure ulcers can lead to serious complications, such as osteomyelitis, septicemia and even death.\(^6\)

Pressure ulcers present a massive financial burden not only for the healthcare system, but also for patients. Hirshberg et al\(^{11}\) reported an average hospital charge of $48,934 per patient for the management of ulcers, which did not include the cost of treatment before hospitalization or the cost of pharmaceuticals. Bennett et al\(^{7}\) noticed that the cost of treating a pressure ulcer varies in the United Kingdom from £1.064 (Stage I) to £10.551 (Stage IV). Cost increases coincide with ulcer stage because the time to heal is longer and due to the fact that the incidence of complications is higher in more severe cases. The total cost in the United Kingdom is £1.4 to £2.1 billion annually (4% of total National Health Service [NHS] expenditure). Thus, a need exists for an effective therapy that decreases the healing time and severity of ulcers, is easy to use, and is cost efficient.

The application of various forms of electrical current to augment wound healing has been reported. As early as 1668, electrically charged gold leaf was applied to smallpox lesions to prevent scar formation. Charged gold leaf has also been used for its hemostatic effect in vascular surgery and to heal diabetic and ischemic skin ulcers.\(^8\)

Although the mechanism responsible for the apparent facilitation of the wound healing process is not readily apparent, Becker\(^9\) has suggested the existence within the body of a direct current electrical system that is responsible for controlling tissue healing. Becker theorized that when the body is injured, the inherent electrical balance of the body is disturbed, resulting in a shift in current flow within this system. Becker referred to this shift in current flow as the “current of injury,” which he believes is generated by the injured tissue and is responsible for initiating the healing process. Becker suggested that the ability of anodal electrical current to facilitate healing is based on the current amplifying the magnitude of the body’s current of injury, which acts as a signal to initiate and maintain the healing process.

A recent review article reports that electrical fields may stimulate and direct epithelial cell proliferation and cell migration at the wound edge, and thus promote wound healing.\(^10\)

Another possible benefit of electrical stimulation of an open wound is apparent bactericidal effect that has been reported.\(^11\)

The present study investigates the effect of high voltage monophasic stimulation (HVMS) on pressure ulcer healing. Study endpoints were the Gilman Index and other measured parameters as predictors of healing.

**Keypoints**
- Electrical fields may stimulate and direct epithelial cell proliferation and cell migration at the wound edge, and thus promote wound healing

**Methods**

**Patients.** The methods, plan, scope of therapy, inclusion of patients into groups, and other procedures of scientific research were reviewed, approved, and accepted by the Bioethical Commission of the Medical University of Silesia (Katowice, Poland).

A prospective, randomized, controlled clinical trial was conducted from March 2004 to July 2008. Fifty-eight (58) patients with Stage I, II, and III pressure ulcers were included in this study; and were ultimately allocated into two comparative groups. Computer-generated randomized numbers were sealed in sequentially numbered envelopes and group allocation was independent of place and person delivering the treatment.

**Key points** were: Stage I, (erythema of intact skin—darker skin, dislocation of the skin, warmth, edema, hardness); Stage II (partial-thickness, skin loss, involving the epidermis, dermis or both; the injury is superficial and clinically presents as an abrasion, blister, or shallow crater); or Stage III (total-thickness skin loss, involving damage to or necrosis of subcutaneous tissue that may extend down to fascia or muscle; pressure ulcer appears clinically as a deep crater).\(^12\) The exclusion criteria were: spinal cord injuries or other loss of sensitivity (paresis or paralysis), chronic venous insufficiency, arteriosclerosis (ABI < 0.9), diabetes, ventricular arrhythmia, cardiac pacemakers, metal implants, pregnancy, and post-steroid therapy.

All patients were treated at the Traumatic Surgery Hospital (Piekary Slaskie, Poland).

**Key points**
- A prospective, randomized, controlled clinical trial was conducted from March 2004 to July 2008
- Fifty-eight (58) patients with Stage I, II, and III pressure ulcers were included in this study and were allocated into two comparative groups
- Patients in both groups were treated with pharmacological agents, which included local wound cleansing with potassium permanganate
Group A consisted of 29 patients (10 women, 19 men), whose wounds were treated with HVMS and pharmacologic agents. Group B consisted of 29 patients (18 women, 11 men). They were treated only with pharmacologic agents, administered identically as in group A. Other details of the examined patients and ulcers are shown in Table 1.

In group A, three patients had pressure ulcers from poorly fitting footwear and three others acquired ulcers from poorly fitted artificial limbs (prosthesis). Six patients had ulcers as a result of plaster cast usage after a bone fracture, and two due to complication of unhealed post-operative wounds. Three patients developed ulcers after internal pressure from surgical metal plates and screws following orthopedic operation while four patients developed ulcers after prolonged immobilization. Other patients’ ulcers were a result of mechanical soft tissue injuries (abrasion, scratch, etc.). In group B, one patient had pressure ulcers from poorly fitting footwear; three patients had pressure ulcers from a poorly fitted artificial limb (prosthesis). Two patients had ulcers as a result of plaster cast usage after a bone fracture and three as a result of complications of unhealed postoperative wounds. Three patients had ulcers related to internal pressure from surgical metal plates and screws after an orthopedic operation. Seven patients had ulcers as a result of prolonged immobilization. The remaining patients had ulcers following mechanical soft tissue injuries ($P > 0.05$; chi-square independence test).

In group A, 7 patients had a Stage I pressure ulcer, 13 patients had a Stage II, and 9 had a Stage III ulcer. In group B, 8 patients had a Stage I pressure ulcer, 13 had a Stage II, and 8 had Stage III ulcers ($P > 0.05$).

Body mass index (BMI) was calculated for all patients using the following equation:

$$\text{BMI} = \frac{\text{real body mass in kilograms}}{(\text{height in meters})^2}$$

According to international reference values, a BMI > 30 kg/m² indicates obesity. The number of smokers was recorded as well.

**Treatment.** Patients in both groups were treated with pharmacological agents, which included local wound cleansing with potassium permanganate. The ulcer base was covered with compresses of fibrolan, colistin, and iruxol, and wet dressings of 10% sodium chloride. The dressings were changed daily (in experimental group local bath, compresses, and wet dressings were provided always after HVMS procedures). The therapy period lasted 6 weeks.

A monophasic pulsed current generator (Ionoson™, Physiomed Electromedizin AG, Germany) was used for electrical stimulation in group A. During the course of treatment, double-peaked monophasic impulses of 100 µs (microsecond) and frequency 100 Hz were applied at 100 V. Treatment was performed with a current amplitude, which produced sub-motor stimulation that caused a mild tingling sensation. Electrodes were made of silver or conductive carbon rubber. The active electrode size was matched to the wound size, and placed on saline soaked gauze directly into the wound. The return electrode was positioned on intact periwound skin (Figure 1). Each procedure lasted 50 minutes. Stimulation was repeated once daily for 5 days a week during the entire therapeutic series. Treatment always began with cathode stimulation to clean the wounds of nonviable tissue. Cathode stimulation time lasted for 2 weeks. This was followed by anode stimulation, performed for 4 weeks.

**Outcome parameters.** Treatment progress was evaluated by measuring the wound area that included nonviable and granulation tissue by planimetry of congruent projections of these wounds onto transparency paper, using a digitizing pallet. The depth of the ulceration was measured at various points by precision micrometry. Later, noticeable results were transferred to the software. The electronic equipment for the measurement of areas and volumes of the ulcers consisted of a digitizer (Kurta XGT, Norgwyn Montgomery Software Inc., North Wales, PA) wired to a personal computer with modified software (C-GEO), thus allowing for the calculation of these...
### Figure 2. Parameter calculation formulas.

**$S\%$: relative change of the total surface area (%)**

$$\varnothing S\% = \frac{(S_I - S_F) \cdot 100\%}{S_I}$$

$S_I, S_F$: initial and final total area (cm$^2$)

**$L\%$: relative change of the length (%)**

$$\varnothing L\% = \frac{(L_I - L_F) \cdot 100\%}{L_I}$$

$L_I, L_F$: initial and final length (cm)

**$W\%$: relative change of the width (%)**

$$\varnothing W\% = \frac{(W_I - W_F) \cdot 100\%}{W_I}$$

$W_I, W_F$: initial and final width (cm)

**$V\%$: relative change of the volume (%)**

$$\varnothing V\% = \frac{(V_I - V_F) \cdot 100\%}{V_I}$$

$V_I, V_F$: initial and final volume (cm$^3$)

**$R\%$: relative change of the non-viable tissue-covered area (%)**

The amount of non-viable tissue ($\varnothing R\%$) was calculated from the (%) relative change of non-viable tissue-covered areas ($R_I, R_F$):

$$\varnothing R\% = \frac{(R_I - R_F) \cdot 100\%}{R_I}$$

$$R_I = \frac{S_{inf1}}{S_I \cdot 100\%}$$

$R_I$: relative change of the non-viable tissue-covered area before therapy (%)

$S_{inf1}$: initial non-viable tissue-covered area (cm$^2$)

$S_I$: initial total area (cm$^2$)

**$Z\%$: relative change of the granulation area (%)**

The amount of granulation tissue ($\varnothing Z\%$) was calculated from (%) relative change of the granulation areas ($Z_I, Z_F$):

$$\varnothing Z\% = \frac{(Z_I - Z_F) \cdot 100\%}{Z_I}$$

$$Z_I = \frac{S_{gran1}}{S_I \cdot 100\%}$$

$Z_I$: relative change of the granulation area before therapy (%)

$S_{gran1}$: initial granulation area (cm$^2$)

$S_I$: initial total area (cm$^2$)

**$d$: Gilman Index (cm)**

$$d = \frac{\varnothing S}{p} = \frac{S_I - S_F}{(C_F + C_I) / 2} = \frac{2(S_I - S_F)}{(C_F + C_I)}$$

$S_I, S_F$: initial and final total area (cm$^2$)

$C_I, C_F$: initial and final circumference (cm)
parameters. Measurements of area (the total surface area and isolated areas covered with pus or granulation) and volume were performed in each person before therapy and every week during treatment, in addition to time post therapy. The areas and volume of tissue deficiency in the ulcerations were calculated from these data. Length and perpendicular width dimension measurements (for observation of the correlation between surface area and linear dimension changes) were also recorded. The observation of the healing process was supported by precisely calculated parameters, such as the Gilman Index\(^1\) and relative changes\(^1\) (Figure 2).

**Statistical Analysis**

The chi-square independence test (greatest reliability level) was used for analysis of the indicators, which characterized patients in all comparative groups. Mean values of the Gilman Index, total area (isolated non-viable tissue-covered and granulation areas), length, width and volume of the ulcers before and after therapy were compared in both groups by Wilcoxon matched-pairs signed-rank test. The Mann-Whitney U-test was used to evaluate differences in relative changes between the groups. Two-sided results \((P < 0.05)\) were considered to be statistically significant. To define relationships between the change of wound area and volume with changes of linear dimensions the Spearman correlation index was used.

**Results**

The patients’ characteristics in the examined groups were all homogeneous, except gender. Treatment proved effective in both groups (Table 2).

In group A, 8/29 ulcers healed, while in group B 4/29 ulcers healed. A comparison of relative change of the total surface area (85.38% in group A versus 40.08% in group B), length (71.22% in group A versus 30.38% in group B), width (76.09% in group A versus 32.48% in group B), volume (20.69% in group A versus 9.39% in group B) and the Gilman Index (0.64 cm in group A versus 0.28 cm in group B) revealed a difference in favor of group A \((P \leq 0.001)\).

More efficient decrease of non-viable tissue and greater promotion of granulation tissue were observed in group A \((\overline{R} = 26.21\%, \overline{Z} = 40.04\%)\) than in group B \((\overline{R} = 18.84\%, \overline{Z} = 33.19\%)\). However, the changes noted did not demonstrate a statistically significant difference \((P > 0.05)\), but were nearly significant \((P = 0.07)\) between the groups.

In both comparative groups the change of wound area

<table>
<thead>
<tr>
<th>Table 1. Characteristics of patients and ulcers.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
</tr>
<tr>
<td>No. patients</td>
</tr>
<tr>
<td>Age (years)</td>
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<tr>
<td></td>
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<tr>
<td></td>
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<tr>
<td>Sex</td>
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<td>Weight (kg)</td>
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<td>BMI</td>
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<td>Smokers</td>
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<tr>
<td>Ulcer location</td>
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<td></td>
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<td></td>
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<tr>
<td>Duration of disorder (months)</td>
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<tr>
<td></td>
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<tr>
<td>Initial wound area (cm²)</td>
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<td></td>
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<td></td>
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<tr>
<td>Initial wound volume (cm³)</td>
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</tr>
</tbody>
</table>

Chi-square independence test
and volume occurred simultaneously. In group A the correlation between change of total area and length of ulcers was 0.85 \( (P = 0.002) \), total area and width was 0.84 \( (P = 0.002) \), and total area and volume was 0.66 \( (P = 0.01) \). In group B the correlation between change of total area and length of ulcers was 0.55 \( (P = 0.02) \), total area and width was 0.54 \( (P = 0.02) \), total area and volume was 0.49 \( (P = 0.04) \). Thus, the wound healing progressed steadily; however, the process was more steady and significant in patients treated with electrical stimulation.

An example of the healing progress of one patient is shown in Figures 3–5.

### Discussion

The present study was conducted over a 4-year period. One weakness of this lengthy study time was the wide variety of patients and ulcers. The study’s strength was that both groups were compared in terms of several wound-healing outcomes by which a steady healing process was demonstrated.

This prospective, randomized, controlled clinical trial demonstrated that HVMS applied to pressure ulcers 5 times per week, reduced the wound surface area over the 6-week study period by 85.38%. This rate of wound closure was twice that observed in wounds treated in the control group.

According to the present results, HVMS appeared to be quite effective. All factors that could affect wound healing were monitored and recorded in the present study. No difference (except gender) among these factors was detected among the subjects who received electrical stimulation and the subjects in the control group.

The authors were only able to locate one well conducted, randomized, controlled, clinical trial in the literature.\(^{15}\) The study consisted of 34 patients who had a spinal cord injury and a Stage II or Stage IV pressure ulcer. Subjects were stratified based on wound severity and duration and were randomly assigned to receive either a customized, community-based standard wound care (SWC) program that included pressure management or the wound care program plus high voltage monophasic stimulation applied to the wound bed (HVMS + SWC). The percentage decrease in wound surface area at the end of the 3-month intervention period was significantly greater in the HVMS + SWC group (70%) than in the SWC group (36%, \( P = .048 \)). These results demonstrated that HVMS can stimulate pressure ulcer healing.

### Table 2. Results in group A and B using the Wilcoxon test.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Before therapy</th>
<th>After therapy</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total area (cm(^2))</td>
<td>A</td>
<td>4.45 ± 3.39</td>
<td>0.81 ± 1.20</td>
<td>≤ 0.001</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>4.93 ± 4.95</td>
<td>3.00 ± 4.22</td>
<td>= 0.002</td>
</tr>
<tr>
<td>Length (cm)</td>
<td>A</td>
<td>3.31 ± 1.35</td>
<td>1.20 ± 1.51</td>
<td>≤ 0.001</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>3.11 ± 1.74</td>
<td>2.20 ± 1.81</td>
<td>≤ 0.001</td>
</tr>
<tr>
<td>Width (cm)</td>
<td>A</td>
<td>1.84 ± 0.97</td>
<td>0.49 ± 0.68</td>
<td>≤ 0.001</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>1.84 ± 0.93</td>
<td>1.23 ± 1.01</td>
<td>= 0.002</td>
</tr>
<tr>
<td>Volume (cm(^3))</td>
<td>A</td>
<td>0.04 ± 0.12</td>
<td>0.00 ± 0.00</td>
<td>= 0.03</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>0.04 ± 0.11</td>
<td>0.03 ± 0.08</td>
<td>= 0.05</td>
</tr>
<tr>
<td>Pus-covered area (%)</td>
<td>A</td>
<td>12.05 ± 24.79</td>
<td>0.58 ± 3.12</td>
<td>= 0.01</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>16.59 ± 26.62</td>
<td>9.05 ± 25.85</td>
<td>= 0.04</td>
</tr>
<tr>
<td>Granulation area (%)</td>
<td>A</td>
<td>85.64 ± 26.68</td>
<td>49.05 ± 49.18</td>
<td>= 0.004</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>76.37 ± 36.11</td>
<td>71.98 ± 44.45</td>
<td>= 0.05</td>
</tr>
<tr>
<td>Gilman Index (cm)</td>
<td>A</td>
<td>0.22 ± 0.18</td>
<td>0.86 ± 0.45</td>
<td>≤ 0.001</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>0.18 ± 0.15</td>
<td>0.42 ± 0.51</td>
<td>≤ 0.001</td>
</tr>
</tbody>
</table>

### Key Points

- The changes noted did not demonstrate a statistically significant difference \( (P > 0.05) \), but were nearly significant \( (P = 0.07) \) between the groups.
The HVMS can be incorporated successfully into an interdisciplinary wound care program in the community.

Adunsky and Ohry\textsuperscript{16} focused on direct current in pressure ulcer and chronic wound healing.\textsuperscript{16} The authors studied the decubitus direct current treatment (DDCT) electrostimulation treatment of pressure ulcers (Stage III) with respect to rates of ulcer closure and wound area reduction. This multicenter, double blind, randomized, placebo-controlled study included 63 patients from 11 geriatric and rehabilitation medicine departments. The authors compared a placebo treated group (PG) to an active treatment group (TG). Treatment lasted for 8 consecutive weeks with a 12-week follow up period. At day 57 (end of treatment) and day 147 (end of follow-up), a difference was not found between the groups regarding complete wound closure rates ($P = 0.28$ and 0.39, respectively), nor was a difference found regarding mean time needed to achieve complete wound closure ($P = 0.16$). Absolute ulcer area reduction and speed rate of wound area reduction (reflected by change from baseline ulcer area, percentage) were better in participants allocated in the treatment group only until day 45 (standardized estimate for trend of healing speed -0.44 and -0.14 for TG and PG, respectively). Afterward, differences were not found between the two groups. A logistic regression analysis favored complete healing in TG compared to PG (odds ratio 1.6, CI: 0.4–4.73). Analysis of per protocol patients revealed that time needed for wound closure was 52\% longer in PG ($P = 0.03$, compared with TG). The results suggested that DDCT treatment for Stage III pressure ulcers, in addition to the conservative wound care, may be useful in accelerating the healing process during the first period of care.

Lee et al\textsuperscript{17} investigated the efficacy of ultra low microcurrent delivered by the Electro Pressure Regeneration Therapy (EPRT) device for managing chronic wounds. In this study, 23 patients with chronic skin ulcers and 2 with abdominal dehiscence that was present for an average of 16.5 months were treated with the EPRT device who were previously unresponsive to standard conservative treatment in a hospital setting. Wounds were treated with direct current (maximum of 3 mA) of 1 polarity for 11.5 minutes and then with a current of the opposite polarity for another 11.5 minutes. Treatment was applied through ultra low microcurrents (in the mA to nA range) conducted through special wraps applied above and below the wound. The results revealed that 34.8\% of cases achieved complete wound healing after an average of 45.6 treatment hours and
39.1% achieved ≥ 50% healing after an average of 39.7 treatment hours. Several patients achieved significant results after 1 to 2 treatments. In their opinion, the EPRT device not only accelerated healing but also appeared to negate the effect of a person's age on wound healing.

Salzberg et al. conducted a randomized, double-blind study to determine if non-thermal, pulsed, electromagnetic energy treatment significantly increases the healing rate of pressure ulcers in patients with spinal cord injuries. Subjects included 30 male volunteers with a spinal cord injury admitted to a Veterans Administration Hospital in New York over a 2-year period. Twenty subjects had a Stage II pressure ulcer and 10 had Stage III. Subjects were given non-thermal pulsed high-frequency electromagnetic energy treatment for 30 minutes (twice daily) for 12 weeks or until healing was achieved. The percentage of healed pressure ulcers was measured after 1 week. Of the 20 patients with Stage II pressure ulcers, the active group had a significantly increased healing rate with a greater percentage of the ulcer healed at 1 week compared to the control group. After controlling for the baseline status of the pressure ulcer, active treatment was independently associated with a significantly shorter median time to complete healing of the ulcer. Stage III pressure ulcers healed faster in the treatment group, but the sample size was limiting. Active, non-thermal, pulsed, electromagnetic energy treatment significantly improved healing for men with spinal cord injuries and Stage II pressure ulcers.

The theoretical interpretation of the effects of HVMS is complicated. One phenomenon that could explain wound healing related to HVMS is the so-called skin battery, which is driven by the sodium pump. The surface between the positively charged wound surface and the negatively charged undamaged skin around the wound generates an electrical current, which travels through the tissues of the moist environment of the wound. This is a precondition for correct tissue reconstruction. Absence or reduction of the potential difference may delay the regenerative process. By stimulating the wound with the positive pole the potential difference may be increased or restored, and the healing process stimulated again.

An in-vitro experiment demonstrated that HVMS inhibits the growth of Pseudomonas faeruginosa, Escherichia coli, and Staphylococcus aureus. The bactericidal effect is considered to be a result of a blockage of diffusion through the cell membranes.

Other researchers believe that the leading cause is electrotaxis—macrophages migrate toward the cathode and neutrophils migrate toward both electrodes. The bactericidal effect around the cathode may be the result of attraction of the phagocytizing macrophages and leukocytes to the infected tissue.

Granulation is also promoted by cathode stimulation, which attracts positively charged fibroblasts. When the wound base is filled with the granulation tissue, anode stimulation is applied, which facilitates the migration of negatively charged epidermal cells.

According to some authors, the acceleration of wound closure and strength may be the result of electrically induced synthesis of collagen and DNA in the fibroblasts, which was observed in an in-vivo study. In the event of particularly treatment-resistant wounds, electrical stimulation may restore the natural potential and facilitate the migration of cells to the wound when healing processes are inhibited due to tissue dryness, presence of some metals (eg, silver), use of non-conductive dressing materials (eg, petroleum jelly), or cleansing enzymes.

**Key Points**

- All factors that could affect wound healing were monitored and recorded in the present study. No difference (except gender) among these factors was detected among the subjects who received electrical stimulation and the subjects in the control group.

**Conclusion**

Based on these results, the authors believe that HVMS is a promising and useful treatment for pressure ulcers. Electrical stimulation coupled with basic wound care, if administered early on in the course of pressure ulcer treatment, might avoid lengthy hospitalizations.

**References**