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Review Paper

Electricity Can Renovate Even the Worst Type of Wounds

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ABSTRACT

Researchers from Chalmers Insitute of Technology (CTH) and the University of Freiburg have proposed an interesting technique that enables chronic wounds to heal faster than ever. Medical conditions like diabetes, cancer, disturbed blood circulation, and spinal injuries can sometimes impair our body's natural ability to heal wounds. Patients who live with such conditions often experience wounds that don't heal.These unrepaired chronic wounds become a source of infection and sometimes even lead to amputations, making patients' lives very difficult. In their latest study, the researchers claim to heal chronic wounds three times faster using electric current showing majority of improvement.

INTRODUCTION

Electricity has been used in so many applications in the past, but its use to heal wounds is an emerging field that is gaining people's interest in research and the health arena. Recently, a study made some discoveries that could validate that electricity can heal wounds to a more impressive extent by threefold on even the worst cases. This groundbreaking discovery not only illuminates the complex mechanisms of electrical healing but will eventually become the innovation in the field for new approaches in medical treatment and wound care. We explore here the transformative power of electricity in wound healing through our look into the key findings of the study, its implications for healthcare practices and the future prospects of integrating electrical stimulation in clinical settings.

How electricity actually heal wounds

Our skin is our body's largest external organ and it plays a crucial role in acting as the first line of defense against mechanical and pathogenic threats, and ideally, any injury to this barrier will be repaired rapidly [**1**]. A wound is any injury causing a break in the structure of living tissue, which may go beyond the skin's epithelial layer to affect the underlying subcutaneous structures depending on the extent of damage [**1**]. Wound healing is a complex yet well-orchestrated physiological process involving a variety of cells

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and chemical mediators. The series of events involved in wound healing can be broadly classified into three main phases: (a) The inflammatory phase, (b) the proliferative phase, and (c) the remodeling phase [**1**]. The events occurring in these phases involve hemostasis to control bleeding, migration of inflammatory cells to the wound site (chemotaxis), granulation tissue formation, collagen repair, vascularization, and re-epithelialization [**2**]. These important events work through a signaling system coordinated by a myriad of mediators such as growth factors and cytokines [**2**]. Examples of these include transforming growth factor (TGF), insulin-like growth factor (IGF), fibroblast growth factor (FGF), vascular endothelial growth factor (VEGF), keratinocyte growth factor (KGF), and platelet derived growth factor (PDGF), that collectively help induce differentiation of immune cells to clear debris and fight infection, stimulate growth, promote formation of new blood vessels, and release inflammatory mediators [**3**]. **Figure 1** depicts the stages of cutaneous wound healing (specifically **Figure 1**A depicts a wound during the inflammatory phase of healing and **Figure 1**B depicts a wound during the proliferative and remodeling phase of healing) and the respective growth factors released to stimulate immune cells and cutaneous structures.

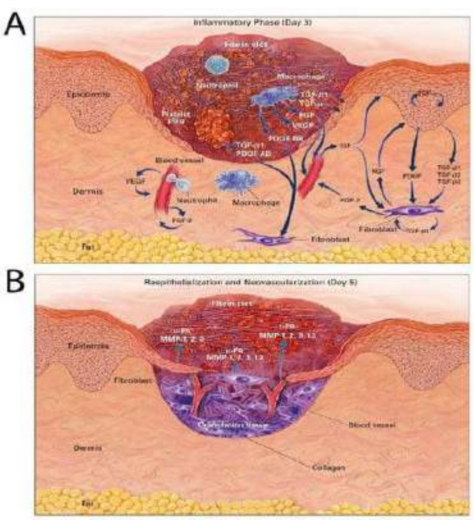
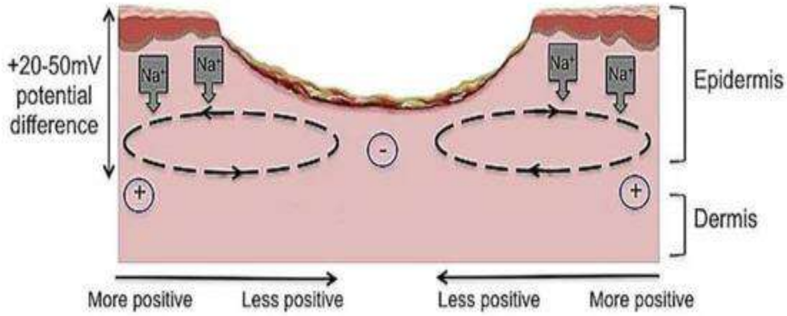


Figure 1. (**A**) A cutaneous wound 3 days after injury. Growth factors thought to be necessary for cell movement into the wound are shown. TGF-β1, TGF-β2, and TGF-β3 denote transforming growth factor β1, β2, and β3, respectively; TGF-α transforming growth factor α; FGF fibroblast growth factor; VEGF vascular endothelial growth factor; PDGF, PDGF AB, and PDGF BB platelet-derived growth factor, platelet-derived growth factor AB, and platelet-derived growth factor BB, respectively; IGF insulin-like growth factor; and KGF keratinocyte growth factor. (**B**) A cutaneous wound 5 days after injury. Blood vessels are seen sprouting into the fibrin clot as epidermal cells resurface the wound. Proteinases thought to be necessary for cell movement are shown. The abbreviation u-PA denotes urokinase-type plasminogen activator; MMP-1, 2, 3, and 13 matrix metalloproteinases 1, 2, 3, and 13 (collagenase 1, gelatinase A, stromelysin 1, and collagenase 3, respectively); and t-PA tissue plasminogen activator. Images reproduced with permission from [**4**]. The wound healing process is also influenced by our skin's endogenous electric potential [**5**], also dubbed the endogenous "skin battery" [**6**]. In undamaged skin, a natural electrical potential of 10–60 mV between the epidermal and sub-epidermal layer exists [**6**]. This is largely attributed to the transport of ions through ion channels and the frequent depolarization and repolarization of cells [**7**]. This trans-epithelial voltage (TEP) largely increases around a wound. The disruption to the epithelium by an injury creates a short-circuit to the TEP, driving positive electrical flow towards the wound, as depicted in **Figure 2**. [**8**,**9**].



Injuries produce an electric current [**10**], and clinical studies have shown that the voltage difference between the wound site and the undamaged skin ranges between 100 and 150 mV/mm [**7**,**8**,**9**].

Figure 2. The current of injury is thought to be significant in initiating repair. Undamaged human skin has an endogenous electrical potential and a transcutaneous current potential of 20–50 mV. This is generated by the movement of sodium ions through Na+/K+ ATPase pumps in the epidermis. The current of injury is generated through epithelial disruption. Following an injury to the skin, a flow of current through the wound pathway generates a lateral electrical field and this is termed the "current of injury" or "skin battery" effect. Image reproduced with permission from [**11**]. These endogenous electric fields play a critical role in wound healing [**7**,**8**], with resulting endogenous currents acting as a cue for cellular migration which concomitantly help heal wounds [**8**]. In addition, it is noteworthy that without this current, it is estimated that the average healing rate decreases by 25% [**12**]. This phenomenon motivates the exploration of the use of electrical stimulation (ES) to accelerate wound healing for various applications [**13**]. Most cutaneous lesions take a week or two to heal. However, this is prolonged in chronic wounds, which do not progress systematically through the healing stages [**14**]. This can be due to factors that hamper the wound healing process such as age, obesity, smoking, nutritional deficiencies or underlying diseases that predispose patients to develop chronic wounds (e.g., diabetes mellitus and/or peripheral venous disease) [**14**]. In conditions such as diabetes, wounds remain in a chronic inflammatory phase due to impaired cellular migration, growth factor release, and poor microcirculation [**15**]. In addition to this, chronic wounds host various microbes that colonize and multiply within the unhealed tissue, further contributing to impaired healing [**15**]. Chronic wounds broadly include diabetic ulcers, pressure sores, and ulcers caused by arterial and venous insufficiency (vascular ulcers) [**16**]. **Figure 3** depicts these chronic wounds and their pathophysiology is briefly described in **Table 1** [**17**]. The staging of pressure and diabetic foot ulcers is outlined in **Appendix A**. Some researchers postulate that the endogenous current observed upon injury is markedly reduced in chronic wounds, contributing to its impaired healing [**18**]. Although these wounds have different etiologies, they possess common characteristics including: Excessive inflammation, tendency to get recurrent infection, improper vascularization, and slower migration of epithelial cells to mediate repair [**17**,**18**,**19**].

Figure 3. Types of chronic wounds (from left to right): Venous leg ulcer, arterial leg ulcer, neuropathic diabetic foot ulcer, and pressure ulcer. Image adapted with permission from [**20**].

Types of electrical stimulation

Electrical stimulation is used for a variety of clinical applications, such as fracture repair, pain management, and wound healing. Several different applications of electricity have been described, including direct current (DC), alternating current (AC), high-voltage pulsed current (HVPC), and low-intensity direct current (LIDC). Physicians are probably most familiar with pulsed electromagnetic field (PEMF) for repair of fracture non-unions and transcutaneous electrical nerve stimulation (TENS) for pain control (29, 30). Frequency rhythmic electrical modulation systems (FREMS) is a form of transcutaneous electrotherapy using electrical stimulation that automatically varies in terms of pulse, frequency, duration, and voltage (31). Even through the electrical stimulation and wound healing literature uses several different types of electrical stimulation, they all seem to have positive results.

Treatment Side Effects

the concept of using electricity on the body may sound painful and horrifying, it is not so in actuality. Many people find the sensation relaxing. People experience a tingling, vibrating, or buzzing sensation which is not unpleasant. It includes a range of treatments using electricity to **reduce pain, improve circulation, repair tissues, strengthen muscles and promote bone growth**. However, the most common side effect with electrotherapy is skin irritation, which is even caused by the overuse of adhesives electrodes or the tape holding the electrodes in place.

Although **there are no side effects of Electrotherapy devices,** there are few recommendation applies to the following people: Pregnant women. People with epilepsy. People with heart problems or any type of electrical or metal implant. Not to be applied over organs or infectious area of the skin.

Study limitations

There are several limitations to this review. There were several different applications of electrical stimulation (PEMF, TENS, high voltage galvanic stimulation), different doses, and durations of therapy that were studied and reported. In addition, many of the studies were small and may have been underpowered. And unlike industry-sponsored phase-three clinical trials, many studies looked at percent change in wound area at 4–6 weeks as the primary outcome rather than complete wound healing at 12 or 20 weeks. Despite variations in the type of current, duration, and dosing of electrical stimulation, the majority of trials showed a significant improvement in wound area reduction or wound healing compared to the standard of care or sham therapy (Table 2) as well as improved local perfusion (Table 1). In fact, these factors were different in all 16 RCTs

Author	Pathology of interest	Duration of treatment	Treatment specification: voltage, current, phase duration, frequency	Population	Outcome
Gilcreast (Citation45)	Perfusion in DFU and high-risk population using HPVC	Once Span: 1 day	100 V, 100 pps, 0.07 pulse duration	Treatment *n* =132	TcpO2 significant improvement in 27% of subjects (p<0.05). No change in 73% of study subjects. Laser Doppler flow NS. Capillary density NS.
Clover (Citation35)	Perfusion in stable claudication using TENS	1 hour, TID, for 6 weeks Span: 6 weeks	1.0 V, 10 mA, 8 Hz	Treatment *n* =24, Control: *n*= 12	Capillary density increased treatment 25% vs. control 0% p<0.005 TcpO2 was greater in treatment group vs. control, p>0.05, raw value NS. Laser Doppler flow NS.
Cramp (Citation36)	Perfusion in health humans using TENS	Once, 15 min Span: 1 day	High frequency = 110 Hz, 200 µs Low frequency 4 Hz, 200 µs	High frequency *n* =10 Low frequency *n* =10 Sham** *n*= 10	TcpO2 NS. Laser Doppler blood flow was greater in the low-frequency group compared vs. other groups p=0.01. Capillary density NS.
Forst (Citation46)	Perfusion in neuropathic patients using TENS	Once, 3 min Span: 1 day	0.2 ms at 4 cycles/s 70 mA or painless muscle contraction	NP-/RP− *n*=14, NP +/RP− *n*=14, NP −/RP+ *n*=8, NP + /RP + *n*=21, Non-diabetic *n*=21	TcpO2 NS. Laser Doppler blood flow increased with ES in all groups at the dorsum of the foot p>0.05. Capillary density NS.
Peters (Citation44)	Perfusion in diabetics using DC	60 min, QID, for 1 day Span: 2 days	50 V, 100 twin-peak monophasic pps	Diabetics with PAD *n*= 11 and without PAD *n*=8	TcpO2 significant improvement in patients with PAD 27% (p<0.05) No change in patients without PAD. Laser Doppler blood flow no difference (p=0.27) Capillary density NS.
Griffin (Citation41)	Venous flow with TENS	Twelve increments in stimuli per minute (spm)	0–5 V, 50 ms, 2–120 spm	Healthy volunteers *n* = 24	Peak systolic velocity in popliteal artery was 10 times higher at 2–8 spm than baseline Ejection volume was 19 times higher at spm than 120 spm.

*Single-blind RCT; **double-blind RCT; NS, not stated; pps: pulse per second.

Author	Pathology of interest	Duration of treatment	Treatment specification; voltage, current, phase duration, frequency	Population	Outcome
Peters (Citation54)	DFU using DC	8 hours, nightly, for 12 weeks Span: 12 weeks	50 V, 80 twin-peak monophasic pps for 10 min, 8 pps for 10 min, then 40 min standby cycles	Treatment *n*=20. Sham** *n*=20	Wound healing ES 65% vs. sham 35% *p*=0.058. Wound area reduction ES 86% vs. sham 71% *p*>0.05. Adverse Event: 10% ES and 15% sham infection.
Adunsky (Citation56)	Pressure ulcers using DC	20 min, TID, 7 day a week, for 2 weeks. Then BID for 6 weeks Span: 8 weeks	NS	Treatment *n*=19 Sham** *n*=19	Wound healing ES 26% vs. sham 16% *p*=0.39. Wound area reduction ES 31% vs. sham 4% *p*=0.9. Adverse events: 14% ES and 18% sham medical reasons. 31% ES and 14% sham had clinical deterioration, consent withdrawal or technical difficulties.
Griffin (Citation57)	Pressure ulcers ion males using HVPC	60 min, daily, for 20 consecutive days Span: 20 days	200 V, total current 500 µA, 100 pps	Treatment *n*=8 Sham* *n*=9	Wound healing ES 38% s 22% *p*>0.05. Wound area reduction was greater in ES group vs. sham *p*=.05, raw value NS. Adverse events: NS.
Houghton (Citation58)	Pressure ulcers using HVPC	60 min, TID, for 3 months. Span: 3 months	50–150 V. 50 µs pulses. 20-min intervals at 100 Hz, 10 Hz, then off cycle Polarity was alternated weekly	Treatment *n*=16, Sham* *n*=18	Wound healing ES 38% vs. control 28% *p*>0.05. Wound area reduction ES 70% vs. control 36% *p*=.048. Adverse events: NS.
Salzberg (Citation59)	Pressure ulcers in males using PEMF	30 min, BID, 7 days a week, for 12 weeks Span: 12 weeks	Radio frequency of 27.12 MHz, 80–600 pps, a duty cycle between 0.5–3.9% and 293–975 W	Treatment *n*=9. Sham** *n*=10	Wound healing ES 100%, average 14 days vs. sham 100%, average 35 days *p*=0.007. Wound area reduction NS. Adverse events: 10% ES patients were missing data.
Wood (Citation60)	Pressure ulcer using DC.	Three time a week, for 8 weeks. Span: 8 weeks	600 µA, 0.8 Hz.	Treatment *n*=41 Shams** *n*=30	Wound healing ES 58% vs. sham 3% *p*<0.0001. Wound area reduction NS. Adverse events: NS.
Ieran (Citation61)	Venous ulcers using PEMF	3–4 hours, daily, 7 days a week, for 90 days.	2.8 mT, 75 Hz, 1.3-ms pulse width	Treatment *n* = 18 Sham** *n* = 19	Wound healing ES 67% vs. sham 32% *p*<0.02. Wound area reduction ES 47% vs. sham 30%, *p*>0.05. Adverse event: 9% ES and

		Span: 90 days			14% sham non-compliance, 5% ES allergic reaction, and 5% ES was diagnosed with rheumatoid arthritis.
Lundeberg (Citation62)	Venous ulcers using AC	20 min, BID, for 12 weeks. Span: 12 weeks	80 Hz, 1-ms pulse width. Polarity was reversed after each treatment	Treatment *n*=24 Sham* *n*=27	Wound healing ES 41% vs. sham 15% *p*<0.05. Wound area reduction ES 59% vs. sham 39% *p*<0.05. Adverse event: 6% ES and 3% sham had allergy, 9% ES and 6% sham had pain, 9% ES and 6% sham non-compliant.
Stiller (Citation20)	Venous ulcers using PEMF	3 hours, daily, 7 days a week, for 8 weeks. Span: 8 weeks	0.06 mV/cm. The signal is 3-part pulse (+, −, +) of 3.5-ms width	Treatment *n*=18, Sham** *n*=13	Wound healing NS. Wound area reduction ES 48% vs. control 42% increase *p*<0.0002. Adverse event: No events.
Santamato (Citation9)	Venous leg ulcer healing using FREMS	25 min, 5 days a week, 3 weeks Span: 3 weeks	Maximum impulse amplitude preset to the value according to patient's sensitivity threshold	Treatment *n*=10 Control *n*=10	Wound healing NS. Wound area reduction ES (58%) vs. control (25%) (*p*<0.005). Adverse events: none.
Carley (Citation8)	Mixed ulcers using DC	2 hours, BID, 5 days a week, for 5 weeks. Span: 5 weeks	300–500 µA for normally innervated and 500–700 µA for denervated skin 30–110 µA/cm^2	Treatment *n*=15, Control *n*=15.	Wound healing NS. Wound area reduction ES 89% vs. control 37% *p*<0.01. Adverse event: NS.
Feedar (Citation53)	Mixed ulcer using pulsed DC	30 min, BID, 7 days a week, for 4 weeks. Span: 4 weeks	29.2 V, maximum 29.2 µA, 128 pps. Polarity reversed every 3 days until stage II was reached, then daily reversal with 64 pps	Treatment *n*=26 Sham** *n*=24	Wound healing ES 0% vs. sham 4%, *p*>0.05. Wound area reduction ES 66% vs. shams 33% *p*<0.02. Adverse event: NS.
Houghton (Citation63)	Mixed ulcers using HVPC	45 min, 3 times a week, for 4 weeks. Span: 4 weeks	150 V, 100 µs, 100 Hz	Treatment *n*=14 Sham** *n*=13	Wound healing NS. Wound area reduction ES 44% vs. sham 16% *p*<0.05. Adverse event: NS.
Jankovic (Citation64)	Mixed ulcers using FREMS	40 min, daily, 5 days a week, for 3 weeks	300 V, 1,000 Hz, 10–40 µs, 100–170µA.	Treatment *n*=20 Control *n*=15	Wound healing NS. Wound area reduction ES 82% vs. control 46% *p*<0.001. Adverse event: NS.

		Span: 3 weeks			
Lawson (Citation65)	Mixed wounds using DC	30 min, three times a week, for 4 weeks. Span: 4 weeks	5 V, 30 Hz, pulse width 200 µs. Current of 20 mA	DM I or II: *n*=8 Without DM: *n*=9	Wound healing NS. Wound area reduction diabetics 70% non-diabetics 38% *p*<0.01. Adverse event: 20% of diabetic group was hospitalized. Ten percent of non-diabetic dropped out secondary to vertigo.
Sarma (Citation55)	Leprosy ulcers using PEMF	30 min, daily, 5 days a week, for 35 days. Span: 35 days	Sinusoidal form 0.95–1.05 Hz; amplitude ± 2,400 nT	Treatment *n*=18 Sham** *n*=15	Wound healing ES 6% vs. sham 0%, *p*>0.05. Wound volume reduction ES 86% vs. sham 48% *p*=0.04. Adverse event: 10% ES and 10% sham removed for irregularity in attendance and 15% sham removed for suspicion of malignancy.

*Single-blind RCT; **double-blind RCT; NS, not stated; pps, pulse per second; NP, neuropathy; RP, retinopathy.

CONCLUSION

The future of electricity in healing wounds holds incredible potential for transforming the landscape of healthcare. Electrical healing techniques, with research ongoing, thoughtful implementation, and a commitment to safety, can transform the face of wound care and present new hope for patients around the globe. As we continue to study this innovative field, the possibilities are so electrifying. Thus, as we conclude, the remarkable possibility of electricity bringing healing to wounds at an accelerated rate opens up a future where innovative medical technologies could revolutionize healthcare practices. Further research and careful implementation promise to enhance patient outcomes, thereby advancing the field of wound care. We stand at the cusp of a new era, where healing is not just faster but more effective and transformative by embracing this electrifying frontier in medicine.

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