THE TREATMENT OF PRESSURE SORES USING PULSED ELECTROMAGNETIC ENERGY (PEME)

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This study investigated the efficacy of pulsed electromagnetic energy (pulsed shortwave diathermy) in the treatment of pressure sores. Four different treatment protocols were compared. These comprised the electrostatic (E) field at pulse train frequencies of 20 and 110 pulses per second (pps) and the electromagnetic (H) field also at 20 and 110 pps. Twenty non-ambulatory male subjects (mean age 79.5 ± 9.8 years) all with pressure sores in the trochanteric or sacral region were referred by their physicians for participation in the study. Subjects were randomly divided into 4 groups of five subjects, each group receiving one of the four treatment protocols. Group 1; H field at 20 pps, Group 2; E field at 20 pps, Group 3; H field at 110 pps and Group 4; E field at 110 pps. The same therapist was responsable for all treatment applications. An ABAB repeated measures experimental design was used, each phase lasting one calendar week. Multifactorial analysis of variance showed highly significant reductions in sore surface areas within each treatment group at the 4th ($p<.007$) and 5th ($p<.001$) evaluation (E_4 and E_5) compared with the initial evaluation (E_1). No significant between group difference was observed. These results suggest that pulsed electromagnetic energy can be an effective means of treating pressure sores. Further testing with larger groups might isolate the most effective treatment protocol.

KEY WORDS: Pressure sores, pulsed electromagnetic energy, pulsed shortwave diathermy, geriatric patients

L'objet de la présente étude était d'examiner l'efficacité de l'énergie électromagnétique pulsée (diathermie à ondes courtes pulsées) dans le traitement des plaies de décubitus. On a comparé quatre protocoles de traitement différents, y compris le réglage du champ électrostatique (E) à des fréquences du train d'impulsions de 20 et 110 impulsions par seconde (i/s) et le champ électromagnétique (H), également à 20 et 110 i/s. Vingt sujets non ambulatoires de sexe masculin (âge moyen 79,5 ± 9,8 ans), ayant tous des plaies de décubitus dans la région du trochanter ou du sacrum, ont été envoyés par leurs médecins pour participer à l'étude. Les sujets ont été répartis au hasard en 4 groupes de cinq sujets, chaque groupe recevant un des quatre protocoles de traitement. Groupe 1, champ H à 20 p/s; groupe 2, champ E à 20 p/s; groupe 3, champ H à 110 p/s; et groupe 4, champ E à 110 p/s. Le même thérapeute avait pour responsabilité d'appliquer tous les traitements. On a utilisé une conception expérimentale ABAB à mesures répétées, chaque phase étant d'une durée d'une semaine. L'analyse multifactorielle de variance indiquait des réductions très significatives de la superficie des plaies au sein de chaque groupe de traitement lors de la 4e (p,007) et de la 5e évaluation (p,001) (E_4 et E_5), en comparaison de l'évaluation initiale (E_1). On n'a observé aucune différence significative d'un groupe à l'autre. Ces résultats semblent indiquer que l'énergie électromagnétique pulsée peut être un moyen efficace pour traiter les plaies de décubitus. De nouveaux essais avec des groupes plus nombreux permettraient peut-être d'isoler le protocole de traitement le plus efficace.

Of all the perils inherent in prolonged bedrest the pressure sore remains among the most persistent. Measures aimed at its prevention have only partially succeeded in reducing its prévalence and its treatment presents a continuing challenge to therapists.

A pressure sore is a localised region of necrotic tissue resulting from ischemia of the skin and subcutaneous tissue[1]. The primary cause is pressure[2] but numerous other factors can contribute to the formation of pressure sores, among which are; decreased sensation, poor arterial circulation, muscle atrophy, malnutrition and humidity stemming from perspiration or incontinence[3]. The incidence of pressure sores varies considerably in hospital populations but is particularly high among spinal cord injured patients (60%) and elderly immobilised patients (66%)[4] with the incidence increasing with length of stay. A pressure sore can rapidly become infected, a factor which greatly increases the difficulty of its control and treatment [5].

Over the years physiotherapists have employed numerous physical measures to treat pressure sores, among which can be listed; hydrotherapy [6] infrared radiations[7] ultraviolet radiations [8,9], electrical stimulation [7,10-13] ultrasound [1,9] shortwave diathermy[14] and, most recently, laser therapy[9].

Shortwave diathermy (SWD) in the continous mode, usually at a frequency of 27.12MHz, is used primarily for its heating effect[15]. Heat is produced in the tissus by the induction of the electromagnetic energy using either the electrostatic or the electromagnetic field. When electromagnetic energy is interrupted at regular intervals (pulsed SWD) temperature changes can occur, dependent on the total peak power delivered[15]. However, at very low pulse frequencies and moderate peak power, thermal changes appear to be minimal, often with no physical evidence such as erythema, skin temperature increases or any subjective sensation of heating being evident[16,17].

Pulsed electromagnetic energy (PEME) has been used with varying rates of success in the treatment of post-surgical skin grafts[17], nerve regeneration[18] ankle sprains[19] and hand injuries[20]. Therapeutic effects reported include; reduced pain and swelling and a more rapid return of function[19,20]. The wide range of pulse frequencies, power settings and dosages used however, are confusing and the negative results reported by some workers for PEME [21,22] may reflect this confusion. A comprehensive list of clinical trials, compiled from the literature by Kitchen and Partridge[15] illustrates this point.

The mechanism by which PEME is thought to produce its positive effects remains speculative[15]. A general improvement in the local circulation and the resultant increases in oxygenation and phagocytosis are advanced as hypotheses[18]. It may be that these improvement serve mainly to reduce the obstacles to normal healing. Evans[23] has remarked that the healing process cannot be accelerated, but poor or delayed healing can be prevented. If this is the case PEME might be especially beneficial in older patients, where recuperative processes are already slowed [23].

A search of the literature could find no mention of the use of PEME for the treatment

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Table 1: Pressure sore classification

Stage	Tissue involvement	Appearance
A	Redness of skin	Skin blanching on light finger pressure
B	Dusky, cyanotic skin	Non-blanching erythema
C	Epidermal and dermal layers	Superficial break extending to and including the dermis
D	Epidermis and adipose tissue	Shallow ulceration
E	Epidermis to muscle tissue	Deep decubitus ulceration
F	Epidermis to bone and joint	Very deep ulceration involving bone frequently with septic dislocation and/or osteomyelitis

Adapted from: Judd CO[24]. The prevention and treatment of pressure sores. 1981; Can Pharm J , 114: 88-93.

of pressure sores, although the effects claimed for this modality ie: the reduction of inflammation and oedema, increases in fibroblasts and white blood cell counts and a more rapid laying down of collagen in experimental animals[18] might presuppose its successful use.

The aim of this study was to evaluate the effectiveness of PEME in the treatment of pressure sores, using four different treatment protocols. In the absence of any documented guidelines regarding the treatment of pressure sores with PEME, we were obliged to formulate our own. In this respect we were guided primarily by the current practice of the therapists of the hospital in which the study was conducted. The recommendations of the manufacturer of the machine used in the study, were also taken into consideration. These protocols were designed to test the effects of PEME using both E and H fields at energy levels shown to be insufficient to create noticeable heating of the skin. This decision was made for two reasons: Firstly, many of our patients could not be relied upon to accurately respond to skin tests for thermal sensation. Secondly, energy levels necessary to produce effects in human tissues, apart from heating, are believed to be very low [16].

METHOD

Subjects

Twenty non-ambulatory male hospital inpatients aged between 60 and 101 years (mean79.5 $\pm$ 9.8) were referred as participants in the study by their physicians. All had pressure sores in the area of the trochanteric or the sacral region. The mean duration of the pressure sores was 13.5 weeks (max 52, min 0.5 weeks). The sores treated during this study were classified by the referring physician, according to tissue involvement, in six stages (Table 1).

Based on this classification, the sores treated ranged from stage C (penetration to epidermis and dermis) to stage F (bony involvement). The majority however (14 sores) were stage D and E (adipose and muscle tissu involvement). All 20 sores were laboratory tested and swab cultures proved negative for pathogenic organisms and anaerobic bacteria. The only exclusion criterion from participation in this study was that none of the patients should have a cardiac pacemaker (Table 2).

The patients were randomly allocated one of four treatment protocols. A series of 20 unmarked envelopes were prepared, each containing one of the four protocols, five envelopes for each treatment protocol. These envelopes were arranged in random order and remained in the possession of the treating therapist. On referral by his physician, each patient was allocated an envelope and the protocol contained was applied for the twenty treatments. The same therapist performed all the treatments for the twenty patients participating in the study.

This method of randomisation resulted in groupings less equal than we would have hoped regarding age, sore duration and particularly for sore surface area. Stratified randomisation by age, sore surface area and duration, might have given more balance between the four groups. However stratification is cumbersome to administer and execute and with the small number comprising each group in this study, these wide variations could still occur[25]. The skewed distribution resulting from a few high values, was partially compensated for by using the logarithm of the sore area in the statistical analyses. Routine nursing procedures were maintained throughout the course of the study, these included dry dressings for the sores and a turning regime wherein each patient was turned, or assisted to turn, every four hours throughout the day and night. No additional medication which could affect wound healing was administered and no concurrent physiotherapeutic

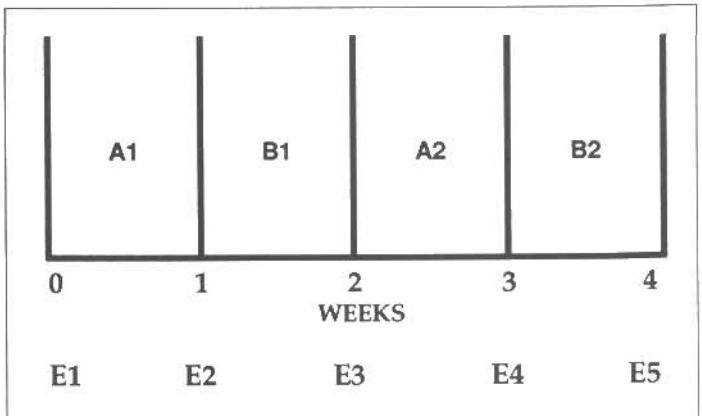

Figure 1. ABAB repeated measures experimental design.

Table 2: Descriptive statistics of subjects, pressure sore surface areas and duration ($\bar{x} \pm SD$) for each treatmentprotocol.

Protocol	Age (years)	Sore duration (weeks)	Sore surface area at E.1 (mm2)
	93	01	15
	83	06	667
1	75	41	91
	90	34	109
	91	03	45
$\bar{x}$	86.4	17.0	185.4
SD	7.4	18.96	271.77
	101	01	33
	94	08	18
2	75	52	474
	84	26	64
		04	728
$\bar{x}$	83.8	18.2	263.4
SD		21.24	321.43
	68	33	43
	69	02	2481
3	74	03	174
	78	06	50
	92	00.5	69
$\bar{x}$	76.2	8.9	563.4
SD	9.7	13.62	1073.27
	71	*	301
	78	20	91
4	79	*	952
	60	01	21
	70	02	115
$\bar{x}$	71.6	7.66	296.0
SD	7.63	10.69	381.04
Total sample			
$\bar{x}$	79.5	13.52	327.05
SD	11.14	16.44	511.91

* Missing data

interventions were initiated during the study.

In this study an ABAB repeated measures design was used. This design has been shown to be an effective means of evaluating treatment interventions in individuals and small groups, it permits controlled testing without a separate control group, each patient serving as his own control. This design is considered a strong source of support with regard to causal interventions and is capable of supplying information about functional relationships between variables[26]. In the ABAB design (Figure 1) the baseline is established during the first phase A, between the first and second evaluations (E_1 & E_2) and the treatment is applied during the first phase B (2nd week). The treatment is discontinued in the second phase A (3rd week) then reinstated in the second phase B (4th week).

It must be stressed that PEME is not an experimental treatment. It was routinely used for the treatment of pressure sores at the hospital in which this study was undertaken. The dosages applied and the method used (condensor field or magnetic field) were however completely arbitrary and empiric, depending on the individual therapists experience and 'feeling'. The purpose of this study was to try to bring order out of chaos, to attempt to identify optimal parameters for the most effective use of this modality in the treatment of pressure sores. Consequently, each patient, when prescribed PEME by his physician was treated, using one of the predetermined protocols, with no additional informed consent being required.

Method and materials

The treatment device for all treatment applications was a Curapuls 419* high frequency (HF) generator which transmits pulsed and continuous electro-magnetic waves at a frequency of 27.12 MHz. The Curapuls 419 has a maximum HF pulse power of 1000 watts (W). Pulse duration is fixed at 400 microseconds (us) and pulse train frequency is variable from 15 to 200pps in ten separate settings. A peak pulse power of 700W was used throughout the study for all treatments. At 700W the pulse train frequencies of 20 and 110 pps give a mean pulse power of 5.6W and 30.8W respectively (Table 3). The effects on the tissues of these low mean power outputs is such that no measurable increases in temperature occur[16,17]. Under these conditions it has been postulated that the so called non-thermal 'biological' effects ie: changes in cell sensitivity, permeability and membrane potential, may predominate over the thermal effects[27].

Procedure

The treatment for each patient consisted of two 20 minute applications of PEME daily (Monday through Friday) one each in the morning and the afternoon, during the second and forth week of the study, for a total of 20 treatments. The frequency of two daily applications and duration of two weeks was aimed at maximizing the number of applications over a short time period. This was thought necessary as the transport of the patient (often in his bed) to the physiotherapy department, was

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frequently a time consuming and complicated procedure.

The method of application and dosage for each of the four treatment protocols varied, with two of the groups receiving PEME at 110 pulses per second (pps) using either the electromagnetic (H) field or the electrostatic (E) field, the other two groups receiving PEME at 20 pps again using the H or E field. The E field was applied using two rigid condensor electrodes (13cm diameter) in a coplanar technique, an electrode being positioned on each side of the sore, parallel with and at a distance of 2 cm from the skin. The H field was applied using a 'circuplode' (Figure 2). The 'circuplode' is a drum type induction electrode, 14 cm in diameter, which contains a coil and a condensor. In addition, the 'circuplode' incorporates a screen, which is designed to block the transmission of the electrostatic field allowing only the passage of the electromagnetic field. The 'circuplode' was positioned directly over the sore at a distance of 1 cm.

Due to the advanced age of our patients, many of whom were also confused and unaware of the proceedings, no skin tests were performed. However, as the dosages used in all four protocols were shown to be imperceptible, any danger of excessive thermal reactions was considered unlikely. In all cases the patient remained under close observation throughout the treatment.

Table 3: Treatment parameters for the 4 treatment protocols

Protocol	Field	Frequency (pps)	Electrode surface area (cm^2)	Power Peak (W)	Average	Power density (W/cm^2)
1	H	20	155	700	5.6	0.036
2	E	20	133	700	5.6	0.042
3	H	110	155	700	30.8	0.199
4	E	110	133	700	30.8	0.230

H = magnetic field
E = electric field

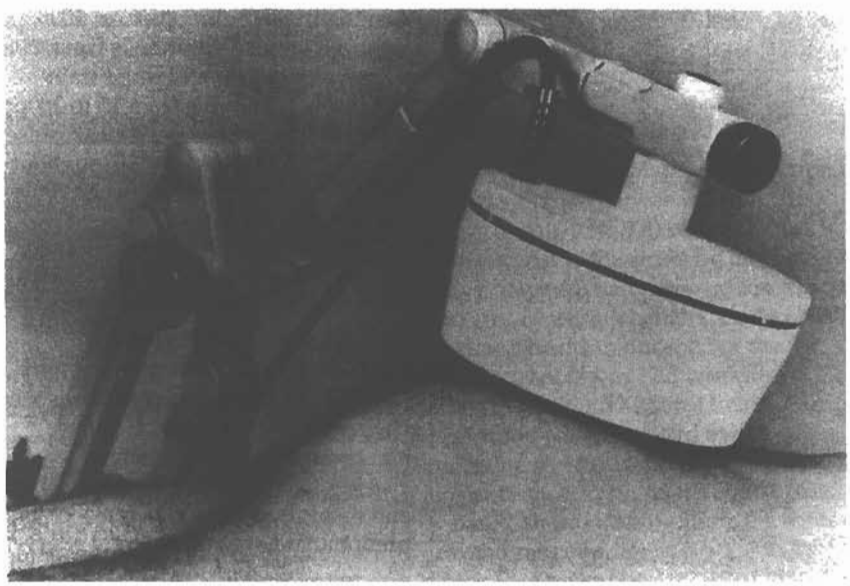

Figure 2. The circuplode inductive electrode. (Permission Enraf Nonius Delft).

Measurement of the sore

The surface area of each sore was recorded a total of five times by an independent evaluator, who remained blind to the protocol used and to her previous ratings. Sore surface area was recorded by tracing the outline on sterile transparent wrap. This outline was subsequently transposed by one of the investigators onto graph paper and its surface area calculated, by counting the number of millimetric squares. Sore measurements were taken at the beginning of each week of the study and immediately following the final treatment (E1- E5) for a total of five individual measurements. The reliability of the tracing procedure has been reported by Griffin and coworkers[28] who compared photographic and transparency based methods of measuring ulcers. They reported a significant correlation between the two methods (r=.996 -.999, p .001).

Data analysis

The initial measurement (E_1) of each sore area was recorded as 100%. At E_2 through E_5 the mean sore surface area for each group was calculated as a percentage of the group mean at E_1. These group mean percentages were subjected to multifactorial analysis of variance to determine between and within group differences over time, for frequency, field and frequency times field. To correct for skewness and non-normality, the logarithm of the sore surface area was used in the analysis of variance. All results were based on the variation between consecutive measures.

RESULTS

The mean sore surface area (±SEM) for all groups at each evaluation is shown in Figure 3. Figure 3 illustrates the evolution over time, for each treatment group, as a percentage of the group mean sore area at E_1. Differences over time for the four groups were statistically significant only at the 4th (p.007) and 5th (p.001) evaluation

Table 4: Within subject comparisons for 'Time' effect, for all subjects (n = 20)

Comparisons	t-value	p
E_1 v E_2	0.52	.640
E_1 v E_3	1.11	271
E_1 v E_4	2.79	. .007
E_1 v E_5	3.34	001

To correct for normality, the logarithm of the sore area was used in the analysis of variance.

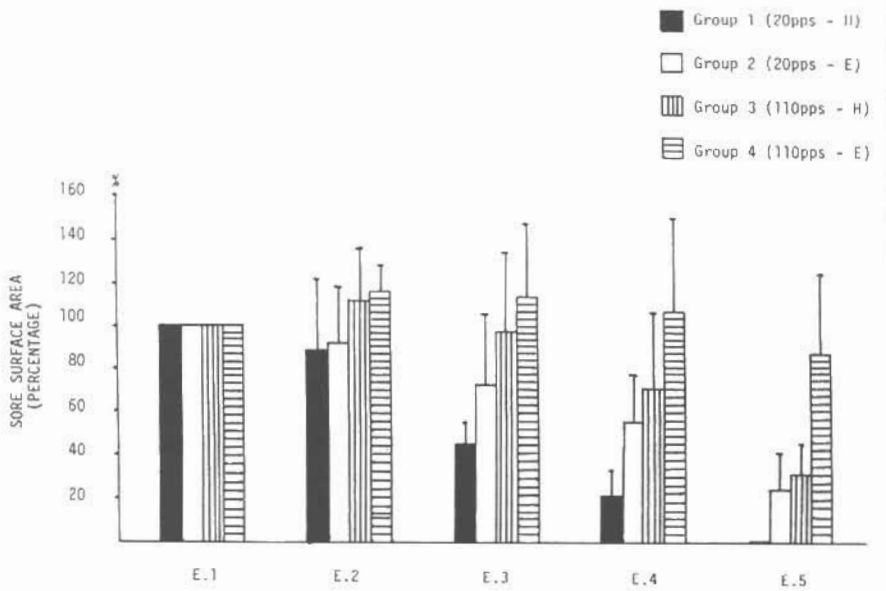

Figure 3. Percentage mean surface area ($\pm$ SEM) at E1— E5 (E1 = 100%) for the four treatment groups

(Table 4). No significant differences were determined between groups for either frequency or field (Table 5).

DISCUSSION

Significant reductions in the mean sore surface area at E_4 and E_5 compared to E_1 were observed for all four protocols during this study. In attempting to explain these reductions four factors can be cited as possible causative mechanisms. These are;

1. *Time;* the four weeks elapsing between E_1 and E_5 could have played a role in the healing process.
2. *Nursing procedures;* these procedures could have been influenced ie: intensified, by the nursing staff, cognisant of the fact that the patient was participating in the study and would therefore be under closer than normal scrutiny.
3. *The placebo effect;* the knowledge that he/she is receiving 'special' treatment has been shown to enhance results in experimental trials. This placebo or 'Westinghouse' effect has been estimated variously at between 32% and 50% [29].
4. *The effects of PEME.*

Although we cannot entirely eliminate any of these factors as having influenced the observed changes, it is possible to put them in perspective in regard to the results obtained, and to attribute a relative degree of importance to each.

With respect to 'Time', the mean sore duration at E_1 was 13.5 weeks (Table 1). Between E_1 and E_2 ie: before the first treatment was implemented, the mean sore surface area increased by an average of 20% (Table 6). From E_3 to E_5 there is a gradual decrease, so that by E_5 the mean sore area is 47% less than at E_2. This decrease in mean sore surface area of over 47% in a period of just three weeks, we feel, excludes the passage of time as being a major factor in producing these results.

One of the main weaknesses of this study was the lack of control of nursing procedures. The hospital in which the treatments were performed has over 950 beds and the participating patients were correspondingly widely dispersed, making any form of close observation, outside of the physiotherapy department, impracticable. However, by the same token, we feel it to be highly improbable that a conscientious nursing staff could have conspired to give our subjects special treatment throughout the period of the study. Nevertheless, any future studies of this nature should attempt some form of nursing control, to reduce to a minimum any possibility of bias.

As regards the possibility of any placebo effects influencing the results of this study, we feel it to be unlikely. It should be remembered that the average age of our subjects was 79.5 years, the majority were long-term residents of the hospital, many were confused, some habitually slept throughout the entire treatment session and, in fact, were unaware that they had been treated. Under these circulstances, although it cannot be totally discounted, any 'Westinghouse' effect would be minimal.

If these three alternatives are excluded, the 20 PEME treatments remain the only mechanism likely to have brought about these changes. As was stated earlier, the reduction in sore surface area began with the commencement of the treatments, no concurrent therapeutic interventions were initiated during the period of the study. The reductions in sore surface area were statistically significant only at the 4th and 5th evaluation (E_4 & E_5), possibly indicating a gradual healing process concommitant with successive treatment applications.

Although no significant differences were observed between the four treatment protocols, this does not necessarily indicate that none exist. Visual comparisons (Fig 3) suggest

Table 5: Analysis of variance of sore surface area between and within subjects over time for Frequency, Field and Frequency x Field (n = 20)

Source of variation	DF	MS	F	p
A. Between subjects:				
Frequency	1	22.72	1.36	.261
Field	1	4.45	0.27	.613
Frequency x Field	1	9.37	0.56	.465
Error (1)	16	16.71		
B. Within subjects:				
Time	4	18.03	20.82	.000
Time x Frequency	4	0.97	1.12	.353
Time x Field	4	0.53	0.62	.653
Time x Frequency x Field	4	1.43	1.65	.172
Error (2)	64	0.87		

* To correct for normality, the logarithm of the sore area was used in the analysis of variance.

Table 6: Descriptive statistics of sore surface area (mm^2) for all subjects for E_1 through E_5 (n = 20)

Time	Mean	SD	Min	-	Max
E_1	327.1	575.9	15	-	2481
E_2	397.1	861.6	10	-	3749
E_3	369.5	969.6	5	-	4268
E_4	310.2	904.3	0	-	4030
E_5	209.6	639.0	0	-	2834

trends, but these are not supported by the statistical analyses. Our groups were small, another weakness in this study, but it is possible that further studies of this nature using larger experimental groups, might clarify this point.

With five subjects per group and with a significance level of .05 (two-way tests), only standardized effect sizes (expected effect sizes divided by standard deviations) of 2.25 can be detected with a power of 0.80. Sample sizes of at least 16 subjects per group would be necessary to detect standardized effect sizes of 1.00 with a significance level of .05 and a power of 0.80.

Although the results of this study suggest that PEME may have a salutary effect on the evolution of pressure sores, the manner by which it produces the effects remains a contentious issue. On the one hand it is argued that, if HF waves in any form are induced into human tissues, even for minute periods, heating will be produced[30] and any biological or therapeutic changes will be the result of this induced thermal effect. The alternative mechanism, the so called athermal effect, suggests that the E and H fields induced by PEME at low power densities produce physiological effects apart from heating, which in some manner alters the membrane potential, resulting in improved cellular metabolism and an enhanced healing effect[31,32,33].

It is beyond the scope of this report to arbitrate between these two opinions, whether or not there is a separate physiological mechanism cannot be demonstrated by this study. However, if the effects produced are those resulting from the heating of the tissues, then the degree of temperature change has to be of critical importance. Goldin[17] treating donor sites in plastic surgery, obtained similar results at low power densities ($0.143 W/cm^2$). Wagstaff[34] in the treatment of back pain also noted the more effective use of PEME at very low power densities ($0.150 W/cm^2$).

In this study the mean power intensities delivered to the tissues with both the E and H fields ranged from a low of 5.6 W at 20 pps (power density 0.036 to $0.042 W/cm^2$) to a high of 30.8 W at 110 pps (power density of 0.199 to$0.230 W/cm^2$). This conforms to the Arndt-Schultz law and supports the contention of Presman[35] that maximum effects of HF radiations occur with relatively weak power densities. We feel that this study confirms that PEME at low power densities can be an effective treatment modality for pressure sores.

CONCLUSION

The findings of this study suggest that PEME, using either the E or H fields, at low power densities, can be an effective treatment for pressure sores. No differential effects between the four treatment protocols were demonstrated by these results. Future studies using larger experimental groups might determine the relative effectiveness of the two fields and different pulse frequencies.

Acknowledgements

The authors gratefully acknowledge the invaluable assistance of Dr Jacques Caillé and the clinical staff of the physiotherapy department of l'Hôpital Ste-Anne-de-Bellevue, whose enthusiastic and unflagging support over the period of one year, made this work possible.

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