Use of transcutaneous pressure of oxygen in the evaluation of edematous wounds

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Dooley J, Schirmer J, Slade B, Folden B. Use of transcutaneous pressure of oxygen in the evaluation of edematous wounds. Undersea Hyperbaric Med. 23(3):167–174.—Transcutaneous pressure of oxygen (PtcO₂) was measured in edematous wounds before and after a regimen of hyperbaric oxygen (HBO₂) therapy, in patients breathing normobaric air (AIR), 100% normobaric oxygen (O₂), and 100% O₂ at 239 kPa (2.36 atm abs; HBO). Wounds also were scored for severity, including three ratings for periwound edema. Only during AIR was pre PtcO₂ of markedly edematous wounds significantly lower than that of moderately edematous and non-edematous wounds (P < 0.001). After HBO₂ therapy, wound severity score and periwound edema rating decreased significantly (P < 0.001), and periwound edema ratings could no longer be distinguished by PtcO₂. Although pre periwound PtcO₂ measured during both O₂ and HBO evaluations was significantly greater than that measured during AIR (P < 0.0001) and was positively correlated with subsequent change in wound severity (P < 0.05), regression analyses failed to yield a significant prediction equation. The authors conclude: a) dramatically marked increases in PtcO₂ of normally hypoxic (<30 Torr O₂) edematous wounds during O₂ and HBO challenges demonstrate that periwound edema is an O₂ diffusion barrier during normal conditions; b) HBO₂ therapy significantly reduces periwound edema in markedly edematous wounds; c) despite significant correlations between pre-therapy periwound PtcO₂ measured during O₂ and HBO challenges and changes in wound severity, single PtcO₂ measurements are not predictive of changes in periwound edema or overall wound severity.

wound edema; hyperbaric oxygen therapy

Since the validation of the transcutaneous pressure of oxygen (PtcO₂) use in adults (1), its usefulness in evaluation of nonhealing wounds and hypoperfused tissue has been extensively studied. PtcO₂ evaluations have been used successfully to assess the presence or severity of peripheral vascular disease (2,3), predict surgical outcome after revascularization (4), detect failing tissue transfers (5,6), and determine the appropriate level of indicated amputations (7–10).

The possible utility of PtcO₂ to predict healing outcome of problem wounds also is of significant medical interest, but findings in this area remain equivocal. Particularly perplexing are findings in both conventional (7–10) and hyperbaric oxygen (HBO₂) therapies (11–15), in which many wounds with remarkably low pre-therapy periwound PtcO₂ values have eventually healed. However, PtcO₂ of problem wounds evaluated under HBO₂ conditions (253 kPa) before hyperbaric therapy has been demonstrated to be predictive of healing (11,13–15). Conversely, pre-therapy PtcO₂ values of these same problem wounds measured during air-breathing (11,13,14) and normobaric 100% O₂ breathing (13–15) were not predictive of healing.

The failure of periwound PtcO₂ obtained during normobaric air- and O₂-breathing evaluations to predict healing outcome has not been adequately addressed and warrants further study. Several wound or host conditions seem to have substantial impact on wound oxygenation and may be responsible for the inability of PtcO₂ obtained under normobaric conditions to predict wound healing outcome. Of significant interest to us is the effect of periwound edema on both wound oxygenation and the attainment of useful PtcO₂ information during normobaric evaluations.

Periwound edema, characterized by the presence of excessive extracellular fluid in periwound regions, is principally associated with venous disease (16–19) and inflammatory reactions (20,21) and has been reported to form or influence the formation of an oxygen diffusion barrier around the wound. In addition to its contribution to a non-healing wound outcome, periwound edema’s effect as an oxygen diffusion barrier also potentially limits the clinical usefulness of periwound PtcO₂ monitoring under normobaric, air-breathing conditions (22).

The purpose of the present study was to evaluate the utility of PtcO₂ measurements in determining oxygenation status of edematous, nonhealing extremity wounds and in predicting their healing following HBO₂ therapy. More specifically, we attempted to determine: a) how periwound edema affects pre- and post-therapy periwound PtcO₂ measurements of patients breathing air and 100% O₂ under normobaric conditions and 100% O₂ under hyperbaric conditions; b) whether periwound
edema ratings and severity scores of edematous wounds are significantly altered by a regimen of HBO$_2$ therapy; and c) whether PtcO$_2$ obtained prior to hyperbaric oxygen therapy can be used to predict the healing of edematous, lower extremity problem (non-healing) wounds in patients subsequently completing a regimen of HBO$_2$ therapy.

METHODS

Subjects: Sixty informed adult patients (39 males; 21 females) with non-plantar, nonhealing wounds of the lower extremities consented to PtcO$_2$ monitoring before and after a regimen of HBO$_2$ therapy. The average age of the subjects was 61 yr (ranging from 26 to 82 yr). All subjects were military medical beneficiaries or Veterans Administration patients.

Instrumentation and equipment: Two of three interconnecting multiplace chambers in the David Grant USAF Medical Center hyperbaric facility were fitted with PtcO$_2$ monitoring systems (model TCM2; Radiometer) with associated E5242-0 oxygen electrodes, modified for and used with 20-ft cables to allow in-chamber monitoring. Patients sequentially breathed air and 100% O$_2$ from clear vinyl hoods (Sealong) supplied through standard U.S. Air Force A-14 breathing gas regulators. Regulators were mounted inside patient protocol boxes designed to allow attendantsto select air, O$_2$, or custom gas mixtures as sources of patient breathing gas. The hoods were exhausted to outside air by differential pressure, assisted at low chamber pressures and during sea level monitoring by boost pumps, to prevent O$_2$ and CO$_2$ accumulation in the chambers and the chamber facility. Temperature and relative humidity inside the chambers were maintained at 21°–23°C and approximately 50–55%, respectively, by an automated atmospheric conditioning system. In addition to the maintenance of a comfortable environmental temperature, each patient’s monitored limb was kept covered with a sheet or light blanket to avoid extremity heat loss sufficient to elicit shunting of the cutaneous circulation in the area being monitored.

Experimental procedures: Before PtcO$_2$ monitoring and initiation of HBO$_2$ therapy, patient wounds were evaluated and given a wound score according to all but several components of the Knighton wound severity score method (23). Ranges of values for each variable of the Knighton severity score criteria employed in our study are presented in Table 1. Because extremity arterial pulse data are often inaccurate in diabetic patients (representing 32% of the patients studied), the dorsalis pedis pulse (7 points maximum) and posterior tibial pulse (7 points maximum) were not assessed. Further, in assessment of patient history, because a substantial number of the patients studied were unable to accurately identify the duration of their wound (10 points maximum), the variable also was not assessed. A maximal potential wound severity score for patients in our study was 73 points vs. the 97 possible points if all of Knighton’s variables (23) had been included in the assessment.

In addition to the total severity score for wounds, we specifically studied periwound edema (PWE). Wounds with little or no discernible edema were given a score of 0 (PWE0); moderately edematous wounds were given a score of 2 (PWE2); and markedly edematous wounds were given a score of 4 (PWE4).

After further medical evaluation and chamber therapy orientation, patients were seated in chairs or wheelchairs with the monitored extremity supported on a cushioned, low stool (semi-dependent) or were assisted to a nearly supine (slight torso and head elevation) position on a medical gurney, according to wound status and/or the patient’s general health. However, extremities were never elevated. All PtcO$_2$ assessments were performed inside either of two research-configured multiplace hyperbaric chambers.

The periwound area to be monitored was shaved, if necessary, repeatedly stripped with tape, wiped clean with alcohol, and rubbed dry with gauze in preparation for placement of the PtcO$_2$ electrode fixation ring. An anterior chest site, 5 cm below the midclavicle, was identically prepared for the placement of a reference electrode. The electrodes were warmed to 44°C and calibrated to read 0.5 times actual values. That is, the partial pressure of O$_2$ at sea level atmospheric conditions (~160 Torr) was halved (80 Torr O$_2$) for calibration purposes, allowing measurement of PtcO$_2$ which at full value could potentially exceed the three-digit upper range limit of the monitoring system encountered during HBO monitoring conditions. Calibration values also were appropriately adjusted, as needed, to accommodate occasional substantial variations in atmospheric pressure. Each electrode then was secured to a fixation ring filled with a standard contact solution.

These experimental procedures were repeated on the patient’s last treatment to determine post PtcO$_2$ wound score, and PWE values, enabling statistical testing for pre-to-post mean score changes and for correlations between percent change in wound score and pre PtcO$_2$. The regimen of HBO$_2$ therapy continued until the wound healed, when successive weekly evaluations failed to show improvement, or the patient’s overall medical condition warranted cessation of therapy.

After being fitted with a neck ring and clear vinyl hood assembly, the patients breathed from a compressed air source (21 kPa O$_2$

AIR) until a stable, peak PtcO$_2$ was obtained. Stability of PtcO$_2$ was achieved within the 25-min monitoring sessions. After peak air-breathing PtcO$_2$ values were obtained, the patient’s hood was removed, the breathing gas
Table 1: Values for Selected Variables Wound Severity Score *

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*Adaptation of the Knighton Wound Severity Score (23).*

was switched to 100% \( \text{O}_2 \) (101 kPa \( \text{O}_2; \text{O}_2 \)), and the hood was ventilated with \( \text{O}_2 \). The hood was then replaced, initiating a second 25-min \( \text{PtcO}_2 \) monitoring period. Upon completion of the normobaric \( \text{O}_2 \) breathing period, the patient's hood again was removed, and the chamber was compressed before initiating a final 25-min \( \text{O}_2 \)-breathing period at 2.36 atm abs (239 kPa \( \text{O}_2; \text{HBO} \)).

Statistical analysis: Pre- and post-therapy scores for \( \text{PtcO}_2 \), wound severity, and PWE were recorded and analyzed to test for statistically significant changes in and relationships between those variables. Analysis of variance (ANOVA) with Newman-Keuls tests of pairwise comparisons were used to test for significant mean \( \text{PtcO}_2 \) (Torr) and wound severity score (units) differences between PWE categories (PWE0, PWE2, PWE4) before and after HBO<sub>2</sub> therapy. ANOVA with tests for simple main effects and Student’s \( t \) tests were used to test for significant pre- to post-therapy \( \text{PtcO}_2 \) and wound severity score differences within PWE categories.

The Wilcoxon Sign-Rank test was used to test for significant pre- to post-therapy differences in the non-parametric ratings (0, 2, 4) for PWE. Additionally, statistical tests of correlation (Pearson) were used to test for significant correlations of pre-therapy \( \text{PtcO}_2 \) values to wound severity score and the percent change in wound severity score within PWE categories across \( \text{AIR}, \text{O}_2 \), and HBO conditions. Finally, regression analyses were accomplished using percent change in wound severity score and \( \text{PtcO}_2 \) variable means to determine if a statistically significant prediction equation could be generated. An alpha of 0.05 was selected as significant for all statistical tests.

RESULTS

Fifty-three of the 60 patients studied actually completed therapy. Of these 53 patients, 33 had complete pre- and post-therapy \( \text{PtcO}_2 \) data; 46 had complete pre- and post-therapy wound severity score data. Results of data analyses are as follows.

\( \text{PtcO}_2 \) according to PWE: Peak pre- and post-therapy periwound \( \text{PtcO}_2 \) of monitored wounds progressively and
markedly increased within all PWE categories when the patients’ breathing gas was sequentially changed from AIR to O2 to HBO ($P < 0.0001$). Mean values for pre- and post-therapy periwound $P_{\text{CO}_2}$, according to PWE group and breathing gas condition, are illustrated in Fig. 1. During AIR (Fig. 1 top), pre-therapy $P_{\text{CO}_2}$ for markedly edematous wounds (23 Torr) was significantly less than that for non-edematous (53 Torr) and moderately edematous wounds (32 Torr) ($P < 0.01$ and $P < 0.05$, respectively). Post-therapy $P_{\text{CO}_2}$ for moderately edematous wounds (47 Torr) was significantly less than that for non-edematous wounds (63 Torr) ($P < 0.05$). Additionally, post-therapy $P_{\text{CO}_2}$ was significantly greater than pre-therapy $P_{\text{CO}_2}$ for PWE2 wounds ($P < 0.02$) and PWE4 wounds ($P < 0.001$).

Changes in wound severity score according to PWE category: Analysis of pre- and post-therapy wound severity score data revealed pre-therapy wound severity scores for PWE4 were significantly greater than those for PWE2 and PWE0 ($P < 0.01$ and $P < 0.002$, respectively). However, the difference in post-therapy wound severity scores between the original PWE groups was not statistically significant (Table 2). Pre- to post-therapy wound severity score reductions for PWE0, PWE2, and PWE4 were statistically significant ($P < 0.002$, $P < 0.001$, and $P < 0.0001$, respectively). Comparisons of wound severity scores across all levels of PWE also revealed that the pre- to post-therapy percent change in wound severity score for PWE4 was significantly greater than that for PWE0 ($P < 0.01$) and PWE2 ($P < 0.01$).

Changes in non-parametric PWE ratings: PWE rating did not increase for any wound. Periwound edema was significantly reduced in 23 of the initially 33 edematous wounds. Six of twelve originally markedly edematous (PWE4) wounds improved category rating to PWE2 ($P < 0.001$); the remaining six originally PWE4 wounds showed no evidence of edema (PWE0; $P < 0.001$) by the end of therapy.

**Table 2: Mean Values ($\pm$sd) for Pre- and Post-Therapy Wound Score and Pre- and Post-Therapy Percent Change According to PWE Category**

<table>
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<th>Category</th>
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<th>Percent Wound Score Change</th>
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<td>PWE0, $n = 11$</td>
<td>pre-therapy: 15.6 $\pm$ 7.6</td>
<td>post-therapy: 9.2 $\pm$ 4.0</td>
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<tr>
<td>PWE2, $n = 22$</td>
<td>pre-therapy: 22.8 $\pm$ 10.4</td>
<td>post-therapy: 4.3 $\pm$ 9.5</td>
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<tr>
<td>PWE4, $n = 13$</td>
<td>pre-therapy: 30.7 $\pm$ 10.2</td>
<td>post-therapy: 10.8 $\pm$ 5.2</td>
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*Means with the same subscript differ ($P < 0.01$ to $P < 0.001$.)

**Fig. 1. Top**, mean $\pm$ sd for pre-therapy and post-therapy $P_{\text{CO}_2}$ (Torr) during AIR according to PWE category; **center**, mean $\pm$ sd for pre-therapy and post-therapy (Torr) during O2 according to PWE category; **bottom**, mean $\pm$ sd for pre-therapy and post-therapy $P_{\text{CO}_2}$ (Torr) during HBO according to PWE category.
PtcO\textsubscript{2} EVALUATION OF EDEMATOUS WOUNDS

Eleven of twenty-one PWE2 wounds changed to PWE0 (\( P < 0.001 \)); conversely, 10 PWE2 wounds did not change to a lower PWE rating. The overall change for all edematous wounds (PWE2 and PWE4) from a mean pre-PWE score of 2.73 to a mean post-PWE score of 0.97 after HBO\textsubscript{2} therapy was highly significant (\( P < 0.0001 \)).

Relationship between PtcO\textsubscript{2} and percent change in wound severity score: Tests of statistical correlation failed to show significant correlations between absolute changes in wound severity score and pre-therapy PtcO\textsubscript{2} values for all wounds or for wounds categorized according to PWE category. However, when tests of correlation were run between relative changes in wound severity score (% change from pre- to post-therapy) and pre-therapy PtcO\textsubscript{2} according to PWE category, two significant correlations were revealed. Percent change in wound severity score correlated significantly with pre-therapy PtcO\textsubscript{2} values obtained in PWE4 wounds during O\textsubscript{2} (\( R = 0.62; \ P < 0.03 \)) and during HBO\textsubscript{2} (\( R = 0.71; \ P < 0.01 \)). However, despite these significant correlations, step-wise regression analyses of these variables yielded nonsignificant regression equations for prediction of percent wound severity score change from pre-therapy PtcO\textsubscript{2}.

DISCUSSION

PtcO\textsubscript{2} and PWE: The results of our study confirm the reports of others that periwound PtcO\textsubscript{2} obtained from patients with problem wounds breathing 100\% O\textsubscript{2} under hyperbaric conditions is markedly increased (11-15, 24). However, the magnitude of the PtcO\textsubscript{2} increase above baseline (AIR) we observed during HBO\textsubscript{2} (Fig. 1) was substantially greater than that previously reported of apparently similar patient populations treated under similar multiple chamber treatment pressures (11,13-15). The overall mean pre-therapy PtcO\textsubscript{2} of 1,005 Torr during HBO\textsubscript{2} (2.36 atm abs) in our study was 28\% greater than the 786 Torr reported by Wattel et al. for lower extremity lesions in diabetic patients breathing 100\% O\textsubscript{2} at 2 atm abs (14,15). Further, the 256 Torr pre-therapy PtcO\textsubscript{2} of our patients during O\textsubscript{2} was 251\% greater than their reported 102 Torr PtcO\textsubscript{2} of diabetic lesions in patients during normobaric O\textsubscript{2}-breathing conditions. Because diabetic lesions also represented a substantial portion (32\%) of our study population and did not differ significantly in periwound PtcO\textsubscript{2} from other wound types we studied, we have attempted to explain the large PtcO\textsubscript{2} differences observed between our findings and those of Wattel et al. (13-15). First, electrode temperature was maintained at 44\°C for our PtcO\textsubscript{2} recordings. Mathieu et al. (11) and Wattel et al. (13-15) did not report electrode temperature for specific studies, but in one report stated "...electrode elements maintain a (preset) temperature between 42 and 44\°C under constant thermistor monitoring." (14). At electrode temperatures 1\°-2\°C less than 44\°C, substantially lower PtcO\textsubscript{2} would be expected (25). However, electrode temperature alone would not be expected to account for the magnitude of observed mean periwound PtcO\textsubscript{2} differences.

Another factor which may possibly have contributed to the difference in PtcO\textsubscript{2} is the method of O\textsubscript{2} delivery to patients. Inboard leaks of air into improperly fitted O\textsubscript{2} delivery masks have been demonstrated to significantly diminish partial pressures of inspired O\textsubscript{2} (PtcO\textsubscript{2}) potentials (26). Mask leaks may have been responsible for diminished O\textsubscript{2} delivery to the patients of Mathieu et al. (11) and Wattel et al. (13-15). Patients in the present study used O\textsubscript{2} delivery hoods.

Yet another possible factor may be the increased PtcO\textsubscript{2} from increases in hydrostatic pressure (27) secondary to positioning the monitored leg on a low stool, as was the common practice with our patients. Even though PtcO\textsubscript{2} in heated skin may not be truly representative of tissue oxygenation in unheated tissues, semi-dependent positioning of the monitored leg may have a substantial gravitational influence on PtcO\textsubscript{2} measured in the lower extremities (1,25,28).

Although the conditions previously described may have contributed to the discrepancies noted in PtcO\textsubscript{2} values, we believe the most relevant factor explaining these discrepancies is the difference in anatomic site of the wounds being monitored. In particular, Wattel et al. (14) reported that the lower extremity diabetic lesions being monitored in their clinic included plantar-perforating lesions. It has been our clinical experience that plantar lesions exhibit much higher PtcO\textsubscript{2} values during AIR and much lower PtcO\textsubscript{2} values during O\textsubscript{2} and HBO than do dorsal lesions of similar etiology and severity. The extremity lesions investigated in the present study were all lower leg or dorsal foot lesions. Therefore, the inclusion of PtcO\textsubscript{2} data for plantar lesions by others (11,13-15) may be the single greatest contributing factor explaining the relatively lower PtcO\textsubscript{2} values for O\textsubscript{2} and HBO they have reported. Considering the aforementioned factors, we believe that our periwound PtcO\textsubscript{2} data are representative of non-plantar wound oxygenation status in nonhealing extremity wounds of patients being monitored with transcutaneous electrodes maintained at 44\°C.

During O\textsubscript{2} and HBO\textsubscript{2}, pre-therapy PtcO\textsubscript{2} of markedly edematous wounds increased to 12 and 49 times AIR PtcO\textsubscript{2} values, respectively. The hypoxic pre-therapy PtcO\textsubscript{2} status of PWE4 wounds during AIR was not suggestive of the tremendous PtcO\textsubscript{2} increases demonstrated during O\textsubscript{2} and HBO (Table 1). Further, post-therapy PtcO\textsubscript{2} of markedly edematous wounds was not substantially different from that of PWE0 and PWE2 wounds. This relative "normalization" of the initially hypoxic markedly edematous wounds during hyperoxigenation supports the hypothesized existence of O\textsubscript{2}-
diffusive barriers surrounding edematous wounds during normal (AIR) conditions (16–19).

Several vascular studies have suggested that pericapillary fibrin cuffs, generally associated with venous disease ulceration, allow blood flow while acting as a barrier to \( O_2 \) diffusion through the capillaries to the skin (16,17,19). This hypothesis is supported by a study in which \( \text{P} \text{Tco}_2 \) of patient hypoxic venous ulcers increased to or surpassed normoxic levels during short-term supplemental \( O_2 \) challenges (18). Similarly, \( \text{P} \text{Tco}_2 \) of skin disturbed by elicitation of the tuberculin reaction was significantly lower than that of undisturbed skin (20). Although others have disclaimed generalized limb edema as a barrier to oxygenation of chronic venous ulcers (29), they did not report the specific presence or absence of periwound edema which, from our \( \text{P} \text{Tco}_2 \) data, does appear to establish \( O_2 \) diffusive barriers around, if not within, edematous wounds.

Although \( \text{P} \text{Tco}_2 \) during AIR increased from pre- to posttherapy for both moderately edematous \( (P < 0.02) \) and markedly edematous wounds \( (P < 0.001) \), the moderately edematous wound \( \text{Tco}_2 \) increase of 47% was only one-third that of the 139% increase for markedly edematous wounds (Fig. 1 top). After \( \text{HBO}_2 \) therapy, \( \text{P} \text{Tco}_2 \) of markedly edematous wounds during AIR (Fig. 1 top) was not significantly different from that of non-edematous wounds. Conversely, \( \text{P} \text{Tco}_2 \) of moderately edematous wounds remained significantly lower than that for non-edematous wounds \( (P < 0.05) \). These findings suggest a relatively recalcitrant nature of moderately edematous wounds. Additionally, during \( \text{O}_2 \), the significant increase in \( \text{P} \text{Tco}_2 \) for markedly edematous (PWE4) wounds from pre- to post-therapy represented the only statistically significant pre- to post-therapy change in \( \text{P} \text{Tco}_2 \) \( (P < 0.05) \) among PWE groupings. This suggests that marked edema may be more characteristic of an acute wound condition, whereas moderate edema may be more characteristic of a chronic edematous wound condition.

Wound severity scores and PWE: Relative change in wound severity scores (pre-therapy minus post-therapy score divided by pre-therapy score) was evaluated according to PWE category. Like others, we observed a significant correlation of \( \text{P} \text{Tco}_2 \) to percent change in wound severity scores during \( \text{HBO} \) test conditions (11,13–15), although this correlation occurred to a significant degree only in PWE4 wounds \( (R = 0.71; P < 0.01) \). However, in contrast to the reports of these previous studies (11,13–15), we observed a significant correlation between \( \text{P} \text{Tco}_2 \) and percent change in wound severity scores in markedly edematous wounds during \( \text{O}_2 \) \( (R = 0.62; P < 0.03) \). Further, during AIR, we found no correlation between percent change in wound severity scores and \( \text{P} \text{Tco}_2 \) for non-edematous wounds \( (R = -0.07) \) or moderately edematous wounds \( (R = -0.01) \). The significant correlations of \( \text{P} \text{Tco}_2 \) with percent change in wound severity scores in markedly edematous wounds during \( \text{O}_2 \) and \( \text{HBO} \) but not during AIR, plus our findings of hyperoxygenation during \( \text{O}_2 \) and \( \text{HBO} \) of initially hypoxic PWE4 wounds, are strongly suggestive of selective \( O_2 \) diffusive barriers in edematous wounds during normal (AIR) conditions.

Prediction of healing: Although we found a statistically significant relationship between percent change in wound severity scores and pre-therapy \( \text{P} \text{Tco}_2 \) during both \( \text{O}_2 \) and \( \text{HBO} \) for markedly edematous wounds, we also must emphasize that pre-therapy \( \text{P} \text{Tco}_2 \) failed to accurately predict percent change in wound severity scores for any PWE group under any breathing gas condition. This finding does not support reports of other investigators that \( \text{P} \text{Tco}_2 \) measured under \( \text{HBO} \) conditions is predictive of wound healing (11,13–15). However, there are two major differences between the methods of the present study and those employed by others (11,13–15).

First, we did not score wounds as healed or non-healed, but rather as percent change in wound severity score. Although not specifically studied, some of our patients with small wound score changes eventually healed. Second, the predictions of others (11,13–15) were based, not on true predictive equations, but on a "threshold" \( \text{P} \text{Tco}_2 \) measured during \( \text{HBO} \) conditions, above which wounds healed. However, we were unable to identify such a threshold \( \text{P} \text{Tco}_2 \) in our study for assurance of any percent change in wound severity scores. In fact, several wounds with pre-Pt less than 400 Torr during \( \text{HBO} \) conditions had very significant reductions in wound severity, and some healed completely by the end of \( \text{HBO} \) therapy. Conversely, other wounds with pre-therapy \( \text{P} \text{Tco}_2 \) greater than 1,000 Torr during \( \text{HBO} \) conditions failed to show any severity reduction throughout a full regimen of \( \text{HBO} \) therapy.

In a further attempt to find a possible threshold \( \text{P} \text{Tco}_2 \), we analyzed wounds according to arbitrary wound healing response standards, i.e., wounds with a percent change in wound severity score greater than 65% were classified as "responders," and those of less than 35% were "non-responders." Mean \( \text{P} \text{Tco}_2 \) of the responders \( (n = 11) \) during \( \text{HBO} \) conditions was 1,035 Torr; non-responder mean \( \text{P} \text{Tco}_2 \) during \( \text{HBO} \) conditions \( (n = 16) \) was 951 Torr. This relatively slight difference certainly does not discriminate between those wounds which will and those which will not respond to \( \text{HBO} \) therapy. Consequently, we believe it is more prudent for providers of \( \text{HBO} \) therapy to use short-term (e.g., 10–14 days) changes in \( \text{P} \text{Tco}_2 \) and objective wound status to determine whether to continue therapy, rather than attempting to predict outcome or deny therapy.
on the basis of a single pre-therapy \( \text{Pco}_2 \) threshold criterion.

In conclusion, the \( \text{Pco}_2 \) and percent change in wound severity score findings of our study of edematous wounds lead us to conclude the following: a) periwound \( \text{Pco}_2 \) measured during air-breathing is inversely related to the severity classification of PWE; b) marked PWE constitutes an \( \text{O}_2 \)-diffusive barrier during normobaric, normoxic breathing conditions (AIR); c) \( \text{O}_2 \) delivery to edematous wounds is markedly increased during sessions in which patients breathe 100% \( \text{O}_2 \) during normobaric and hyperbaric conditions (O2 and HBO); d) percent change in wound severity score following a regimen of HBO2 therapy is more favorable for markedly edematous (PWE4) than for moderately (PWE2) or non-edematous (PWE2) wounds; e) relative changes in wound severity scores of edematous wounds are significantly related to pre-therapy \( \text{Pco}_2 \) measured during both O2 and HBO conditions—however, these changes cannot be accurately predicted from pre-therapy \( \text{Pco}_2 \) obtained during any breathing gas condition; and f) results of this study indicate that it may be more prudent for providers of HBO2 therapy to use short-term (e.g., 10–14 days) changes in \( \text{Pco}_2 \) and objective wound status to determine whether to continue HBO2 therapy, rather than attempting to predict outcome or deny HBO2 therapy on the basis of a single pre-therapy \( \text{Pco}_2 \) threshold criterion.

The views expressed in this material are those of the authors, and do not reflect the official policy or position of the U.S. government, the Department of Defense, or the Department of the Air Force.

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