

Transcutaneous oxygen tension in subjects with paraplegia with and without pressure ulcers: A preliminary report

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Abstract--This study compared transcutaneous oxygen tension ($TcpO_2$) in subjects with paraplegia and pressure ulcers (PU), those with paraplegia and no pressure ulcer (NPU), and ambulatory controls. $TcpO_2$ was measured using a surface-electrode monitoring system, recorded at 1-min intervals for 5 min and averaged. Mean $TcpO_2$ was significantly lower in the PU than the NPU and control groups (23.53 ± 1.83 vs. 58.93 ± 2.53 and 79.70 ± 6.77 mmHg, respectively, $p < 0.05$). In a PU subgroup ($n=4$) mean $TcpO_2$ of the pressure ulcer and nonpressure ulcer sides (trochanter or ischium) were significantly different (21.05 ± 2.98 vs. 67.65 ± 2.11 mmHg, respectively, $p < 0.001$). Additionally, the NPU group demonstrated significantly lower $TcpO_2$ than the controls. PUs had a greater reduction in $TcpO_2$ levels relative to controls than NPUs. No association was found between $TcpO_2$ and duration of injury, completeness of lesion, or smoking history. Thus, $TcpO_2$ may be an effective method to identify individuals who are susceptible to pressure ulcers. The further attenuation of $TcpO_2$ observed in the PU group may be useful to help predict whether ulcers will heal with local care or will require additional treatment.

Key words: *paraplegia, pressure ulcer, transcutaneous oxygen tension ($TcpO_2$).*

INTRODUCTION

A pressure ulcer is defined as any lesion caused by unrelieved pressure resulting in damage of underlying tissue (1), usually occurring over bony prominences receiving maximum pressure. The ischium (24 percent), sacrum (23 percent), trochanter (15 percent), heel (8 percent), and scapula (5 percent) are the most common sites for these ulcers (2). Individuals with spinal cord injury (SCI) are more susceptible to pressure and shear forces involved in the formation of ulcers due to a variety of factors including immobility, muscle atrophy, and sympathetic nervous disruption, all of which may contribute to poor circulation in the lower limbs.

The development of ulcers is more prevalent in the elderly, in individuals confined to bedrest, and especially in persons with SCI. In 1994, the incidence of pressure ulcers in subjects with acute and chronic SCI reported over an 18-mo period ranged from 8 to 34 percent (3,4). Over a 5-year period, pressure ulcer occurrence in groups with acute SCI reported by Richardson et al. (5) was 60 percent in those with complete tetraplegia, 42 percent in incomplete tetraplegia, 52 percent in complete paraplegia, and 29 percent in incomplete paraplegia. Furthermore, individuals with a history of such ulcers demonstrated an increased risk of developing a recurring pressure ulcer (4).

Transcutaneous oxygen tension (T_{cpO_2}) is a reliable, noninvasive measure of local oxygen supply to the tissue (6,7), and has been used as a predictor of success after amputation and in wound healing (8,9). Recently, Mawson et al. (10) demonstrated differences in T_{cpO_2} levels at the sacral region in persons with SCI compared with ambulatory controls, but they did not differentiate between SCI subjects with and without pressure ulcers. Therefore, it is unclear whether SCI subjects without such ulcers exhibit reduced oxygen supply to the tissue. The purpose of this study was to determine whether SCI outpatients without pressure ulcers have different T_{cpO_2} levels than ambulatory controls, as well as to further evaluate the T_{cpO_2} of subjects with paraplegia who have pressure ulcers and are hospitalized for treatment.

METHODS

The study was conducted at the Spinal Cord Damage Research Center located at the VA Medical Center, Bronx, NY, from September 1996 to October 1997, and was approved by the Institutional Review Board for human studies. After obtaining informed consent, the investigators collected anthropometric data and a brief medical history from 21 male subjects between the ages of 22 and 73 years, who were classified into 1 of 3 groups: 7 with paraplegia and pressure ulcers (PU), 7 with paraplegia and no pressure ulcers (NPU), and 7 ambulatory controls. Subjects were matched for height and weight. All subjects with SCI had durations of injury greater than 1 year and were screened for cardiovascular diseases and diabetes mellitus. Based upon neurologic evaluations documented in the medical records, these subjects were categorized as having

complete injuries (complete motor and sensory loss) or incomplete injuries (incomplete motor and/or sensory loss). With the exception of the presence of pressure ulcers, subjects with SCI were otherwise medically stable and were not prescribed medications known to interfere with circulation and/or thermoregulation.

TcpO₂ was measured using a surface electrode monitoring system (Radiometer, Copenhagen, Denmark). A fixation ring, consisting of a top section containing the mount for the membrane electrode and bottom comprised of an adhesive disk, was placed on the skin. Externally applied heat (44 °C) emanating from the electrode was used in transcutaneous monitoring to produce an increase in cutaneous blood flow. Thus, TcpO₂ measurements reflect the level of tissue perfusion. A liquid gel was applied to the sensor membrane to facilitate proper contact. Measurements of TcpO₂ were performed with the subjects resting in the supine position. In the NPU and control groups, the membrane electrode was placed for 20 min each at the right and left trochanter. The membrane electrode was calibrated at each site for 15 min, after which TcpO₂ levels were recorded at 1-min intervals for 5 min and averaged. Measurement of TcpO₂ for the PU group was performed by placing the membrane electrode 1 cm from the pressure ulcer located on either the trochanter, ischium, or sacrum. TcpO₂ was also measured on the opposite, intact side of those subjects with either trochanter or ischial pressure ulcers. Pressure ulcer assessments were performed by a registered nurse on the day of TcpO₂ measurement, and were documented as either stage III or IV. The nurse recorded the number of ulcers and staged each ulcer, using the system suggested in Guidelines for Pressure Ulcer Prevention (11). Data are reported as mean plus or minus standard error of the mean (SEM). A two-way ANOVA with post-hoc analysis was applied using Scheffe's pairwise comparisons for the three groups; the level of significance was set at p<0.05. A paired Student's t-test was conducted to compare differences between TcpO₂ levels on the ulcerated and unulcerated sides in a subgroup of PU (n=4). A second paired t-test was used to compare differences between the right and left trochanter in the NPU and control subgroups (n=8). Regression analysis was performed to determine whether a significant association exists between mean TcpO₂ levels and duration of injury, completeness of lesion, or smoking history.

RESULTS

No significant differences were found among the three groups for height, weight, body surface area (BSA), or supine systolic and diastolic blood pressure (**Table 1**). Duration of injury was not significantly different between PU and NPU groups (**Table 1**). The control group was significantly younger than both the PU and NPU groups (31±3.20 vs. 56±6.00 and 52±5.78 yrs, respectively, p<0.05; **Table 1**). The PU group included four active smokers and three who never smoked; the NPU group consisted of two active smokers, three former smokers, and three who never smoked; and the control group contained one active smoker and six who never smoked. Both SCI groups included subjects with lesions between T4 and L2.

Table 1.

Characteristics of subjects.

Parameter	Group PU	NPU	Controls
Age (years)	56±6.00	52±5.78	31±3.20*
Height (m)	1.80±0.02	1.81±0.03	1.82±0.03
Weight (kg)	72.2±5.42	83.2±6.69	82.7±14.41
BSA (m ²)	1.79±0.06	2.03±0.08	2.03±0.08
DOI (years)	18±6.84	17±6.27	N/A
Diastolic (mmHg)	66±2.76	77±4.89	67±2.18
Systolic (mmHg)	111±4.04	121±7.30	110±2.41

Data presented as means±SE; PU=pressure ulcer group, NPU=non-pressure ulcer group; BSA=body surface area; DOI=duration of injury; *= $p<0.05$ for control versus PU and NPU.

The mean TcpO₂ was significantly lower (58.93±2.53 vs. 79.70±6.77 mmHg, respectively, $p<0.05$) in the NPU group than in the control group (**Figure 1**).

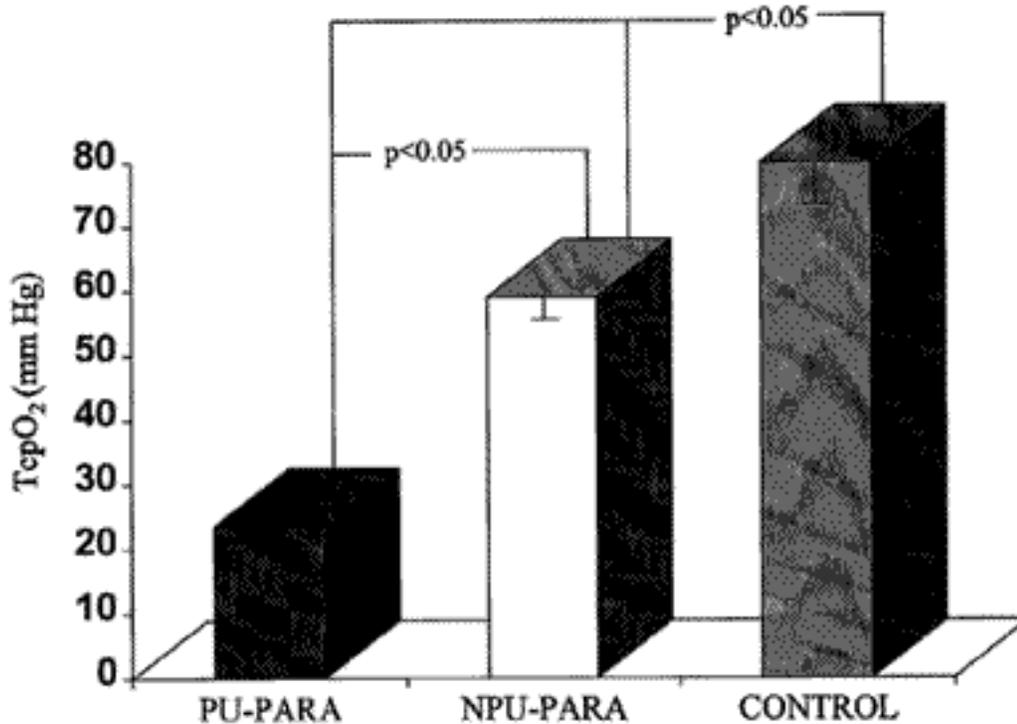


Figure 1.

Data represent means ±SE for transcutaneous oxygen tension (TcpO₂ mmHg). PU=group with paraplegia and pressure ulcer, NPU=group with paraplegia and no pressure ulcer, CONTROL=ambulatory group.

There were no significant differences between mean TcpO₂ levels obtained at the right and left trochanter in either the NPU (n=4) or control (n=4) subgroups (**Table 2**).

Table 2.

TcpO₂ levels at pressure ulcer and contralateral sides at trochanter or ischium, compared with those at right and left trochanters of persons without pressure ulcers.

Group	Ulcer Side or Right Trochanter	Contralateral Side or Left Trochanter
PU	21.05±2.5*	67.65±2.11
NPU	55.25±0.63	55.13±0.94
Controls	70.50±6.56	66.75±7.45

Data presented as means±SE; units=mmHg; PU=pressure ulcer group, NPU=non-pressure ulcer group; number of subjects in each group=4; *= $p < 0.05$ for pressure ulcer versus non-pressure ulcer side.

The PU group demonstrated a statistically reduced mean TcpO₂ level (23.53±1.83 vs. 58.93±2.53 and 79.70±6.77 mmHg, respectively, $p < 0.05$) compared with the NPU and control groups (**Figure 1**). The mean TcpO₂ levels in the PU (n=4) subgroup proximate to the pressure ulcer (trochanter or ischium) and no pressure ulcer sites (trochanter or ischium) were significantly different (21.05±2.98 vs. 67.65±2.11 mmHg, respectively, $p < 0.001$; **Table 2**). Regression analysis revealed no association between mean TcpO₂ levels and duration of injury, completeness of lesion, or smoking history.

DISCUSSION

In our study, we observed significantly lower levels of TcpO₂ at the trochanter region in NPU subjects with paraplegia compared with controls. In addition, we have demonstrated that PU subjects have further reduced levels of TcpO₂ at the ulcer site, relative to an appropriate intact skin site or to the other groups. Our results are similar to a study by Mawson et al. (10) who demonstrated sacral TcpO₂ in the prone and supine positions to be significantly lower in subjects with SCI (including ulcerated and nonulcerated regions) than in controls. Our findings strongly suggest that persons with SCI having no evidence of pressure ulcers exhibit reduced oxygenation of the tissue of the lower limbs.

Sympathetic nerves innervating blood vessels originate from the spinal cord between T1 and L2 (12). Partial to complete denervation of the sympathetic chain may result in unopposed parasympathetic activity and decreased systemic arterial pressure, with the level and completeness of transection of the spinal cord determining the extent of sympathetic disruption. Abnormal regulation of peripheral blood flow in otherwise healthy subjects with SCI may be attributed, in part, to the disruption of the sympathetic nervous system; however, the absence of an association between completeness of lesion and $TcpO_2$ suggests that other factors contribute to the development of pressure ulcers. These factors may include prolonged immobilization, muscle atrophy, the absence of muscle spasticity, and certain psycho-social conditions (13). Further support of this finding comes from Mawson et al. (10,14).

Assessing peripheral blood flow using $TcpO_2$ has become increasingly useful in individuals with limb ischemia due to vascular diseases, such as diabetes or pressure ulcers. Lalka et al. (15) used pre- and postoperative transcutaneous tension as a predictor of surgical outcome of revascularization in persons with peripheral vascular disease. $TcpO_2$ of 20 mmHg or less indicated severe limb ischemia requiring revascularization, and postoperative $TcpO_2$ of 22 mmHg or less indicated that the revascularization was likely to fail. These findings strengthen the supposition that subjects with SCI and pressure ulcers having $TcpO_2$ levels of 20 mmHg or lower are less likely to heal without advanced treatment and may be candidates for surgery. In economic terms, the costs of pressure ulcer treatment can vary greatly; however, it has been estimated that the total national cost of treatment exceeds \$1.33 billion (1). The regular use of $TcpO_2$ monitoring to assess pressure ulcers may help reduce these costs by indicating timely surgery and, thus, more effective treatment.

Several recent studies (16,17) by our group have shown an increased energy expenditure in subjects with SCI and pressure ulcers. Post hoc analyses were used to identify the effect of pressure ulcers on the percent of predicted resting metabolic rate (RMR) and RMR/kg body weight: these measures were significantly higher in PU than in both the NPU and controls (17). The RMR/m² body surface area was significantly higher in PUs than in both NPUs and controls. Similar findings have been seen in persons with tetraplegia (16), where measured resting energy expenditure and percent predicted RMR were both significantly higher in ulcerated subjects than in those without pressure ulcers. Both studies demonstrated a significant increase in energy expenditure in subjects with SCI and pressure ulcers, suggesting that a higher metabolic demand is required for the healing of the ulcers. Our current findings support the previous work, since the quantity of oxygen available for diffusion to the skin depends on the difference between the quantity delivered by the influx of blood and that extracted by the tissue to meet local metabolic demands. $TcpO_2$ and energy expenditure measurements, therefore, should be useful in assessing care for individuals with pressure ulcers.

In a small study of three ambulatory male controls, Newson et al. (18) reported similar relationships between $TcpO_2$ and applied average pressure for the sacrum, trochanter, and lateral aspect of the thigh. In our study, electrode placement at the sacral, trochanter, or ischial regions resulted in similar significantly lower mean $TcpO_2$ levels in PU subjects. Furthermore, despite the location of the ulcer (ischial or trochanter) in a subgroup of four such subjects, we found a

significant difference in oxygen tension levels at the ulcer and non-ulcer sites. In contrast, those in the NPU and control groups demonstrated no difference in TcpO₂ levels between sides.

Although no association was found for TcpO₂ and smoking status, it is well accepted that cigarette smoking may impede the healing of a pressure ulcer due to vasoconstriction and reduced blood flow to the skin, resulting in tissue ischemia (19). Rodriguez and Garber reported a 39 percent increased incidence of pressure ulcers in current smokers with SCI, relative to nonsmokers (4). Moreover, smokers in general may have a decreased awareness or concern for health practices that would help to prevent or heal pressure ulcers. Increasing age is also associated with decreased peripheral circulation and possibly reduced blood flow to the tissue, which may play a role in TcpO₂ levels. In fact, Gothgen and Jacobsen (20) found a tendency for TcpO₂ levels to diminish with increasing age. In our study, the control group was significantly younger than both the PU and NPU groups; however, when statistically controlling for age through multiple regression analysis, the significant differences for TcpO₂ among the SCI and ambulatory subjects were maintained (F=38.51, p<0.001). Therefore, it is unlikely that the age differences among the groups were responsible for the differences in oxygen tension levels.

In summary, we have shown that subjects with paraplegia and without pressure ulcers demonstrated reduced tissue oxygenation below the spinal cord lesion compared with ambulatory controls. As a result of the decreased blood flow, individuals with paraplegia may be more susceptible to develop pressure ulcers. In subjects with existing pressure ulcers, routine measurement of TcpO₂ may provide clinically useful information with regard to the potential of the lesion to heal and as a means to evaluate the need for more aggressive therapy.

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