Utilisation of skin blood flow as a precursor for pressure injury development in persons with acute spinal cord injury: A proof of concept

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Abstract
People with spinal cord injury (SCI) are at high risk of developing a pressure injury. It is unclear why some people with SCI develop pressure injury while others with similar predisposing risk factors do not during acute hospitalisation. This may hinder healthcare utilisation to prevent pressure injuries. The purpose of the study was to examine the proof-of-concept objective bedside skin blood flow measurements before a pressure injury develops in spinal cord injured patients during acute hospitalisation. This was an observational study. All participants had acute traumatic SCI and were pressure injury-free upon enrollment. Skin blood flow patterns were collected at both heels under two circumstances: localised pressure for reactive hyperemia, and localised heating for heat hyperemia. Our results showed that reactive and heat hyperemia were successfully induced in all eleven participants. Two participants developed pressure injury and nine did not have pressure injury at discharge. Heat hyperemia was smaller in participants with pressure injury. No difference was observed in reactive hyperemia between the groups. In conclusion, skin blood flow measurements could be obtained at bedside during acute hospitalisation of SCI for the purpose of research. Further examination of a larger group is warranted to determine clinical use of heat hyperemia pattern as predictor for pressure injury development.

Key Messages
• Objective and non-invasive skin blood flow measurements could be obtained at the bedside during acute hospitalisation of spinal cord injury (SCI) for research purpose, and without interfering with standard medical care.
Reactive and heat hyperemic skin blood flow responses of eleven adults during acute hospitalisation of SCI were examined with laser Doppler flowmetry system before a pressure injury develops or discharge from hospital.

Reactive and heat hyperemic responses could be induced successfully at the bedside in adults with acute SCI. Further investigation of reduced heat hyperemic response is warranted as a predictor of pressure injury development in the spinal cord injured population.

1 | INTRODUCTION

A pressure injury is a prevalent secondary complication in people with spinal cord injury (SCI). Approximately 20% to 30% of newly injured patients will develop a pressure injury within the first year of having SCI. Pressure injury accounts for a considerable amount of repeated healthcare utilisation of inpatient visits in this population, with chronic ulcer of the skin being the most frequent diagnosis codes (~50% of hospitalisation). Clinical manifestations of SCI, such as limited mobility and lack of sensation are well-acknowledged risk factors of pressure injury. However, it is unclear why some people with SCI develop pressure injury while others with similar predisposing risk factors do not. Given that prolonged tissue ischemia and subsequent tissue damage is a main etiological pathway of pressure injury formation, skin blood flow measurements were selected for exploration in this pilot study.

Non-invasive skin blood flow measurement is objective and blood flow is regulated locally and systemically by various mechanisms. Previous studies demonstrated that analysing skin blood flow patterns after the application of standardised stimulus can detect the changes in microvascular function that occurs among patient groups. The two most examined localised stimuli are ischemia and heating. Ischemia induced hyperemia, also known as reactive hyperemia, is increased skin blood flow after temporary obstruction. It is mainly mediated by local mechanisms, including sensory nerve and endothelium. Heat hyperemia is an increase in skin blood flow induced by localised heating between 41°C to 44°C. The initial part of this response is mediated by both local and systemic mechanisms, including axon reflex and sympathetic nervous system respectively. Endothelial regulation occurs later when heating persists. Previous studies in chronic SCI population showed that reactive hyperemia and heat hyperemia were diminished in this population as compared to those without any neurological deficits. This suggested an altered microvascular function, regulated both locally and systemically, during the chronic stage of SCI. To understand the linkage between altered microvascular function and pressure injury development in this population, we believed the next logical step was to develop a proof-of-concept study in a hospital setting where participants were all newly injured, bed or wheelchair bound, and were treated by the same clinician team to eliminate confounding factors. The hospital setting would also allow direct and prospective observation of pressure injury formation during the participants hospital stay. Examining the skin blood flow patterns before a pressure injury develops may hold high clinical relevance.

The primary purpose of this pilot observational study was to examine the proof-of-concept objective skin blood flow measurement as a bedside approach to quantify microvascular function during acute hospitalisation of traumatic SCI. The secondary and exploratory goal was to compare differences in reactive hyperemia and heat hyperemia in participants who developed a pressure injury vs those that did not prior to discharge from hospital. Based on the findings from previous studies on people with chronic SCI, we hypothesized that participants who developed a pressure injury prior to discharge would have lower reactive hyperemia and heat hyperemia.

2 | MATERIALS AND METHODS

2.1 | Study design

This was a proof-of-concept observational study on patients during acute hospitalisation of SCI. Acute stage is defined as within 60 days of SCI in this study. Participants were recruited from two hospitals in an urban area of Midwestern United States: a level 1 urban trauma unit, and an inpatient rehabilitation unit. Participants' length of stay is approximately 1 to 2 weeks in this trauma unit and 3 to 5 weeks in this inpatient rehabilitation unit. Since patients only stay at the trauma unit for a short period of time, recruitment from two settings allow us to capture pressure injury development at any time during the first 60 days of SCI. This approach also allowed us to recruit participants that were treated at other trauma units not accessible to...
our research team. Potential participants from the trauma unit were screened for eligibility once their vital signs were stable upon admission after acquiring SCI. Potential participants from the inpatient rehabilitation unit were screened for eligibility once they were transferred from a trauma unit. People who developed one or more pressure injury prior to admission to the inpatient rehabilitation unit were not included. All study protocols were approved by the committee on research ethics at the institutions in which the research was conducted in accordance with the Declaration of the World Medical Association, including the institution review board offices of the leading university and the two hospitals where the project took place. Written informed consent was obtained from all participants before any procedure was performed. All participants underwent the skin blood flow measurements upon enrollment, and the research team followed up with unit nurses to document whether the participant developed a pressure injury prior to discharge from the hospital. The period of recruitment is from the day of consent and skin blood flow measurements until a pressure injury develops or discharge from the hospital. The period of recruitment was expected to vary from within a day to up to 5 weeks.

2.2 | Participants

Recruited participants were between 18 to 64 years old and had been diagnosed with traumatic SCI within 60 days of recruitment. Participants had injury level at L3 and above, with American Spinal Injury Association Impairment Scale A (no sensory or motor function preserved in the sacral segments) or B (sensory but not motor function preserved below neurological level), and were bed or wheelchair bound throughout their hospital stay. Electronic medical record review was performed to exclude participants with diabetes, unstable blood pressure at rest, ongoing infection, and other cardiac disease diagnoses. Only participants who did not currently have any pressure injury were enrolled in this study. Since the objective of the study was to examine the proof-of-concept of skin blood flow measurements at the bedside to generate pilot data, the sample size was not determined prior to performing the study.

2.3 | Procedures

Upon enrollment, skin blood flow responses toward localised pressure and heating were measured in all participants. Reactive hyperemia and heat hyperemia were collected at two skin sites that are prone to pressure injury development: left and right heels. The order of our data collection on right or left heel was based on randomization. For each side, we measured the reactive hyperemia first followed by the heat hyperemia, and a 10-minute washout period was provided in between reactive hyperemia and heat hyperemia at the same site to minimise any cross-over effect. All data were collected in a side-lying position. The heels were selected as test sites for the following reasons: heels are prone to developing pressure injuries during prolonged bedrest; heels reflect the skin microcirculatory function below the level of injury; and heels are more accessible for the research team as compared to other high-risk sites such as sacral and buttoc areas.

2.4 | Equipment

The skin blood flow was collected with the laser Doppler flowmetry system (Moor Instruments, Wilmington, Delaware) at 20 Hz, and the PowerLab 8/35 with LabChart V8 (AD Instruments, Colorado Springs, Colorado) was used for data acquisition. Reactive hyperemia was induced with a customised hand-held indenter at 8 kPa (60 mm Hg) for 3 minutes with the CP2T-1000 laser Doppler flowmetry skin probe going through the centre for continuous skin blood flow measurement. The heat hyperemia was induced with a heater probe (VHP2) and a laser Doppler flowmetry needle probe (CP3-500) going through the center for skin blood flow collection before, during and after localised heating at 41°C for 5 minutes. Figure 1 demonstrates the indenter during light touch and 60 mm Hg of pressure on the skin, and the heating and laser Doppler flowmetry needle probes. To ensure easy incorporation of our skin blood flow measurement protocols at the bedside, the study team carried a small cart (45 cm × 65 cm, height 81 cm) equipped with the laser Doppler flowmetry system, data acquisition system and a laptop. The nurses at both hospital units inspected the skin on a daily basis until discharge. Once a pressure injury was formed and identified anywhere on the participant’s body, the nurses examined the stage, depth, and size of the wound. According to the National Pressure Injury Advisory Panel Clinical Practice Guideline, pressure injury is ‘localized damage to the skin and/or underlying tissue, as a result of pressure or pressure in combination with shear’. Based on current literature, aetiology of pressure injury includes soft tissue deformation and tissue ischemia. The clinicians at both hospital units provided the diagnosis of pressure injury and its stage to the research team.

2.5 | Outcome measures

Demographic data (age, gender, ethnicity, and body mass index) and SCI data (level and American Spinal Injury
Association Impairment Scale) were retrieved. The use of pressure redistribution devices during hospitalisation was also collected. To obtain the reactive hyperemia and heat hyperemia variables for statistical analyses, all skin blood flow signals were processed using the MatLab program (The MathWorks, Natick, Massachusetts). Skin blood flow signals were first down-sampled to 0.5 Hz using the function ‘decimate’. The down-sampled skin blood flow signals were then processed with curve-fitting technique using the ‘curvefit’ toolbox. Based on the shape of the skin blood flow signal, we used the second order exponential curve-fitting and first order gaussian curve-fitting methods for the reactive hyperemia and heat hyperemia signals respectively. A least squares model fit was applied. Reactive hyperemia variables were calculated as followed. Baseline skin blood flow is the skin blood flow averaged over 1-minute period right before localised pressure was applied. The peak skin blood flow is the maximum value of the fit curve of reactive hyperemia. Normalised peak skin blood flow is calculated using the formula \([(\text{peak skin blood flow} - \text{baseline skin blood flow})/\text{baseline skin blood flow}] \times 100\%\). Perfusion area is the area under the reactive hyperemia curve subtracted by the baseline skin blood flow. Heat hyperemia variables were calculated as followed. Baseline skin blood flow is the skin blood flow averaged over the 1.5-minute period right before localised heating was applied. The peak heat hyperemia is the maximum value of the fit curve of heat hyperemia. The variable area under curve is the integrated area under the fit curve of heat hyperemic response.

2.6 | Statistics

Since this is a proof-of-concept study with small sample size, Fisher’s exact tests were used to compare the categorical variables. Based on previous literatures showing that complete injury, higher level of injury, and lack of pressure redistributing devices contribute to increased risk of pressure injury development, one-tailed results of the Fisher’s exact tests were reported. Continuous variables, such as reactive hyperemia and heat hyperemia were summarised using mean, SD, median and quartiles. To compare reactive hyperemia and heat hyperemia variables between the two groups, each variable was averaged from the data collected from the left and right heels within each participant. Since this is a proof-of-concept study with small sample size, Mann–Whitney U tests were employed to compare the reactive hyperemia and heat hyperemia variables between groups. Based on prior studies showing that reactive hyperemia and heat hyperemia are reduced in people with SCI as compared to healthy controls, one-tailed results of the Mann–Whitney U tests were reported. The significant level was set at 0.05. Due to the small sample size of this study, it is important for us to report the effect size (r) for Mann–Whitney U tests to examine the magnitude of difference in skin blood flow patterns between the groups. By obtaining the effect size from this pilot study, we could calculate the sample size needed for a full-scale prospective cohort study. SPSS ver24 (IBM Corp., Armonk, New York) was used for all statistical analyses.

3 | RESULTS

Thirteen adults were screened for eligibility; two of them were not interested in participating in the study, and eleven of them were recruited, completed, and analysed for this study. Table 1 includes the demographic, SCI, and pressure injury information of all participants. Three people were recruited from the trauma unit, and eight people were recruited from the inpatient rehabilitation unit. Since the trauma unit and the inpatient rehabilitation unit are located at an urban area of Midwestern
United States, all participants were African American male. Two out of eleven participants developed a pressure injury before discharge from the hospital. Participant A2 had a stage 2 pressure injury at the occiput (1 cm × 1 cm), and participant R3 had a deep tissue injury at the sole of left foot (2 cm × 1.5 cm) prior to discharge. All participants were provided with heel off-loading devices throughout hospitalisation, and participants A2 and A4 were provided low air loss mattresses throughout hospitalisation. Fisher’s exact tests showed that there was no significant difference in American Spinal Injury Association Impairment scale (\( P = .382 \)), level of injury (\( P = .655 \)), and the use of pressure redistribution devices (\( P = .345 \)) between participants with pressure injury vs participants without pressure injury at discharge. Period of recruitment (follow-up time) for all participants ranged from 7 to 21 days with an average of 12 days.

Reactive hyperemia and heat hyperemia were only collected once on each participant’s left and right heel, and the development of pressure injury was documented only once when it was first noticed by the unit nurse or on the day of discharge if no pressure injury developed throughout hospitalisation. Figure 2 shows typical reactive hyperemia and heat hyperemia responses collected from one participant. For a visual representation of the skin blood flow pattern between participants with and without pressure injury at discharge, Figure 3A demonstrates boxplots of reactive hyperemia with skin blood flow averaged every 5 seconds after localised pressure removal, and Figure 3B demonstrates boxplots of heat hyperemia with skin blood flow averaged every 30 seconds. Table 2 shows the mean ± 1 SD, median, and quartiles of reactive hyperemia variables from participants with and without pressure injury at discharge. There was no significant difference between the

| ID | Clinical setting | Age (y) | BMI (kg/m²) | Level of injury | AIS | Pressure injury developed prior to discharge | Pressure injury location and stage |
|----|-----------------|--------|-------------|----------------|-----|------------------------------------------|----------------------------------|
| A2 | Trauma          | 20     | 19.4        | T3             | A   | Yes                                      | Occiput, stage 2                  |
| A4 | Trauma          | 25     | 24.2        | T4             | A   | No                                       |                                  |
| A5 | Trauma          | 36     | 14.8        | T3             | A   | No                                       |                                  |
| R1 | Rehabilitation  | 29     | 22.1        | T12            | A   | No                                       |                                  |
| R2 | Rehabilitation  | 19     | 23.7        | T4             | A   | No                                       |                                  |
| R3 | Rehabilitation  | 36     | 27.2        | T2             | A   | Yes                                      | Sole of left foot, deep tissue Injury |
| R4 | Rehabilitation  | 20     | 19.1        | L1             | B   | No                                       |                                  |
| R5 | Rehabilitation  | 27     | 25.1        | T3             | A   | No                                       |                                  |
| R6 | Rehabilitation  | 57     | 21.8        | C5             | B   | No                                       |                                  |
| R7 | Rehabilitation  | 28     | 28.1        | T2             | B   | No                                       |                                  |
| R8 | Rehabilitation  | 31     | 25.1        | C4             | B   | No                                       |                                  |

| Table 1 | Demographic and spinal cord injury information of all participants |

Abbreviations: AIS, American Spinal Injury Association impairment scale; BMI, body mass index.
participants with pressure injury vs those without pressure injury at discharge with regard to reactive hyperemia, including peak skin blood flow (80.92 ± 63.87 vs 161.50 ± 100.20 perfusion unit), normalised peak skin blood flow (300.22 ± 292.84% vs 326.39 ± 166.35%), and perfusion area (2674.37 ± 2478.33 vs 5449.77 ± 4740.03 perfusion unit). The effect size of the difference in reactive hyperemia is considered small (r < 0.3) to moderate (0.3 ≤ r < 0.5). Table 3 shows the mean ± 1 SD, median, and quartiles of reactive hyperemia variables for participants with and without pressure injury at discharge. The reactive hyperemia variables were significantly lower in patients who developed a pressure injury vs those without a wound at discharge: baseline skin blood flow (6.50 ± 4.82 vs 31.62 ± 17.01 perfusion unit), peak heat hyperemia (15.74 ± 9.78 vs 109.26 ± 112.93 perfusion unit), and area under curve (1921.71 ± 851.80 vs 13 814.20 ± 22 419.41 perfusion unit). The effect size of the difference in heat hyperemia is considered large (r ≥ 0.5).

### DISCUSSION

There are two main findings from this pilot study. First, the objective and non-invasive skin blood flow measurement protocols could be conducted successfully at the bedside for research purposes without interfering with medical care. Second, heat hyperemia and reactive hyperemia are objective skin blood flow measurements that could be obtained successfully in patients during acute hospitalisation of traumatic SCI.

This proof-of-concept study was the first one to incorporate non-invasive blood flow measurement protocols at the bedside. The equipment and measurement protocols were demonstrated to the clinicians in both units prior to data collection, including the small space needed for the research team at the bedside, the semi-portable feature of the measurement protocols, and the setup for each measurement at the bedside. The research team coordinated with the units to conduct measurements after the patients were positioned to side-lying by the nursing staff. The trained research team members were able to set up

![Figure 3](image)

**FIGURE 3** (A) Boxplots of skin blood flow averaged every 5 seconds during reactive hyperemia at both heels. The * represents outliers from participant R8. (B) Boxplots of skin blood flow averaged every 30 seconds during heat hyperemia at both heels. The * represents outliers from participant A4 at 0.5 minutes, and R5 and R6 at 2 minutes. The * represents outliers from participant R6 at 5.5 and 7 minutes

### TABLE 2

| Reactive hyperemia variables (unit) | Pressure injury developed prior to discharge |
|-----------------------------------|---------------------------------------------|
|                                   | Yes (n = 2)                                  | No (n = 9)                                |
|                                   | Mean ± 1 SD, Median (1st-3rd quartile)       | Mean ± 1 SD, Median (1st-3rd quartile)    | P  | r  |
| Baseline skin blood flow (perfusion unit) | 27.40 ± 12.30 (27.40 (23.05–31.75)          | 44.36 ± 32.05 (35.75 (21.00–62.16)       | .267 | .25  |
| Peak skin blood flow (perfusion unit)  | 80.93 ± 63.87 (80.93 (58.34–103.51)         | 161.50 ± 100.20 (116.97 (85.41–226.53)   | .200 | .33  |
| Normalised peak skin blood flow (%)   | 300.00 ± 292.74 (300.00 (196.50–403.50)     | 326.39 ± 166.35 (339.50 (230.25–395.00)  | .556 | 0    |
| Perfusion area (perfusion unit)       | 2674.37 ± 2478.33 (2674.37 (1789.15–3550.59)| 5449.77 ± 4740.03 (3313.66 (2191.09–9175.17) | .356 | .17  |
Both studies demonstrated that reactive hyperemia and heat hyperemia were preserved below the level of lesion in participants with chronic SCI. Our findings are consistent with data collected in people with chronic SCI in the laboratory settings. This suggests that objective skin blood flow measurements could be implemented as a research approach to quantify microvascular function objectively during acute hospitalisation of SCI. Further investigation of clinical application of this approach at the bedside is warranted. For the secondary and exploratory goal of the present study, we observed a relatively compromised heat hyperemia in the two participants who developed a pressure injury. Previous studies showed that the first 5 minutes of the heat hyperemia is regulated by both local (axon reflex, sensory nerves) and systemic (sympathetic nerves) mechanisms. The two participants may have some degree of alterations in local or systemic mechanisms that regulate the skin blood flow, and further examination with a larger sample size is needed to determine whether adaptations to these mechanisms contribute to pressure injury formation during acute hospitalisation of SCI. We did not observe any changes in the reactive hyperemic response in the two participants that developed a pressure injury as compared to other participants. The reactive hyperemia is mainly mediated by local mechanisms, such as endothelium-derived hyperpolarizing factor pathway and intact sensory nerves. A previous study examined the reactive hyperemia at the sacrum and the gluteus maximus muscle areas in people with chronic SCI. They compared the reactive hyperemia between two sub-groups of SCI: individuals with and without sensation over the sacrum; and the results varied between the data collected at the sacrum and that at the gluteus maximus muscle. They reported that the only differences in reactive hyperemia between individuals with (n = 8) and without (n = 12) sensation over the sacrum were: higher peak of reactive hyperemia at the gluteus maximus muscle in participants without sensation, and longer time to peak reactive hyperemia at the sacrum in participants without sensation. Although the two participants that developed a

| TABLE 3 | Mean ± 1 SD, median, and quartiles of heat hyperemia variables and Mann–Whitney U test results between groups |
|---------|----------------------------------------------------------------------------------------------------------------------------------|
| Heat hyperemia variables (unit) | Pressure injury developed prior to discharge |  |
| | Yes (n = 2) | No (n = 9) | P | r |
| | Mean ± 1 SD | Median (1st-3rd quartile) | Mean ± 1 SD | Median (1st-3rd quartile) |  |  |
| Baseline skin flow (perfusion unit) | 6.50 ± 4.82 | 6.50 (4.79-8.20) | 31.62 ± 17.01 | 28.85 (18.81-42.19) | .022 | 0.66 |
| Peak heat hyperemia (perfusion unit) | 15.74 ± 9.78 | 15.74 (12.28-19.19) | 109.26 ± 112.93 | 60.21 (49.09-125.50) | .044 | 0.58 |
| Area under curve (perfusion unit) | 1921.71 ± 851.80 | 1921.71 (1620.55-2222.86) | 13 814.20 ± 22 419.41 | 4264.54 (3114.79-9284.22) | .044 | 0.58 |

the devices for measurement in ~5 minutes, finishing collecting each reactive hyperemia for ~5 minutes, and each heat hyperemia for ~10 minutes. If needed, medical care could be implemented between two measurement protocols. With sufficient training, adequate practices in an empty ward, and thorough communication with the clinicians at the units, we demonstrated that incorporating the blood flow measurement protocols to bedside for the purpose of research is feasible and did not interfere with medical care.

To the best of our knowledge, skin microvascular function during the acute stage of SCI in the hospital setting has not been studied. Our pilot study was the first one that examined skin blood flow patterns, that is, reactive hyperemia and heat hyperemia, at the bedside and on people during the acute stage of traumatic SCI. Previous studies that examined reactive and heat hyperemia were performed on participants with chronic SCI in laboratory settings. For the reactive hyperemic response, two studies examined this response in participants with SCI using customised computer-controlled indenters to induce 8 kPa of pressure on the sacrum for 20 minutes. Both studies demonstrated that reactive hyperemia could be successfully induced in adults with SCI at the sacral skin, which is below the level of injury and a location that is at risk of developing pressure injury. For the heat hyperemic response, one study examined the heat hyperemia in participants with SCI by applying 44°C on the foot for 15 minutes, and another study examined the heat hyperemia by applying 41°C for 30 minutes on the lower trunk representing the dermatoome below the level of injury. Both studies showed that the heat hyperemia was preserved below the level of lesion in participants with chronic SCI. In our present study, we demonstrated that both reactive hyperemia and heat hyperemia were preserved at the heels (located below the level of injury) and could be induced and collected successfully at the bedside in the hospital setting.
pressure injury in our study both had complete SCI, we did not observe any trends in altered reactive hyperemia as described in this previous study. Several factors may explain why our preliminary findings differed from this previous study. First, we applied the same amount of localized pressure (8 kPa) for all participants to induce reactive hyperemia, whereas relatively higher amount of pressure (varies among participants between 15-35 kPa) was applied to completely occlude the blood flow and obtain reactive hyperemia in this previous study. They also showed that the amount of pressure required to completely occlude skin blood flow at the sacrum is higher for individuals without sensation. By completely occluding skin blood flow at the bedside during acute hospitalisation of SCI, we may be able to observe a difference between groups; however this approach may not be clinically appropriate. Second, our participants that did not develop a pressure injury are a mixture of individuals with complete (n = 4) and incomplete SCI (n = 4). The difference in reactive hyperemia between individuals with and without preserved sensation noted in previous study may be hindered in this mixture of individuals with complete and incomplete SCI. Last, our participants were recruited within 60 days of traumatic SCI, whereas all participants had SCI for more than 1 year in the previous study. The structural or functional adaptations in peripheral vasculatures that deteriorate from the onset of SCI may not be reflected in our measurement during acute hospitalisation of SCI. Findings from our secondary and exploratory goal need to be interpreted with caution given the pilot nature of this study and should be considered preliminary data. Further studies on whether skin blood flow control mechanisms contribute to pressure injury development is warranted with a larger sample size, and a prospective cohort study design.

Our study has limitations. Due to the exploratory nature of the study, the sample size was small. All participants are African American male due to the geographical location of the hospitals. Participants were only followed until discharge, and the study team did not have access to data of participants who developed a pressure injury after discharge. Due to time restraints in trauma intensive care unit and inpatient rehabilitation hospital, skin blood flow data collection was only performed at the heels and may not be generalised to other areas that are below the level of injury. We understand that length of stay correlates with pressure injury occurrence in the literature. However, since the end point for our recruitment is development of a pressure injury or discharge from the unit, we were only able to collect and report the period of recruitment (period of follow-up) but not the length of stay for each participant. Therefore, the effect of length of stay on our results could not be excluded.

5 | CONCLUSION

This study demonstrated that objective and non-invasive skin blood flow measurements could be obtained at the bedside on people during acute hospitalisation of SCI for the purpose of research, without interfering with medical care. Our preliminary data showed that difference in microvascular function could be detected between participants who developed a pressure injury vs those who did not prior to discharge. Further investigation of microvascular function adaptation and its underlying mechanisms are warranted in understanding their contribution to pressure injury development in the SCI population.

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CONFLICT OF INTEREST

One author of this manuscript (Wukich) has the following conflict of interest that does not involve this manuscript: serves as a consultant with Orthofix Medical Inc., Stryker and Wright Medical, and receives royalties from Arthrex Inc.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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