บทความปริทัศน์

Impaired skin microcirculation in hospitalized elderly and in patients with spinal cord injuries: a pressure ulcer risk

วิภา แซ่เซี้ย

Impaired skin microcirculation in hospitalized elderly and in patients with spinal cord injuries: a pressure ulcer risk

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Songkla Med J 2009;27(2):179-193

Abstract:

Pressure ulcers are complications among patient with chronic illness in most settings, especially among the hospitalized elderly and patients with spinal cord injuries. Impaired skin microcirculation combined with loading pressure, friction and shear force are crucial risk factors for development of pressure ulcers. This article aims to a review available research that has been conducted on skin microcirculation,

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as defined by skin blood flow and skin temperature, in hospitalized elderly and patients with spinal cord injuries. A review of 18 studies revealed that, healthy or ill elderly, as well as patients with spinal cord injuries, were all experience impaired skin microcirculation. A combination of loading pressure and impaired skin microcirculation were found to be crucial pressure ulcer risk factors for the hospitalized elderly and patients with spinal cord injuries. Implication of effective strategies to promote skin blood flow and decrease skin temperature for nursing practice and future research are suggested.

Key words: *elderly, pressure ulcers, skin microcirculation, spinal cord injury*

บทคัดย่อ:

แผลกดทับยังคงเป็นปัญหาที่เกิดขึ้นกับผู้ป่วยที่รับไว้ในโรงพยาบาลโดยเฉพาะผู้ป่วยสูงอายุและผู้ป่วยบาดเจ็บไขสันหลัง การไหลเวียนโลหิตบริเวณผิวหนังที่บกพร่องร่วมกับแรงกดทับ แรงเสียดทานและแรงไถลเป็นปัจจัยเสี่ยงที่สำคัญที่ทำให้เกิดแผลกดทับ รายงานฉบับนี้มีวัตถุประสงค์เพื่อทบทวนงานวิจัยที่ศึกษาเกี่ยวกับการทำงานของระบบไหลเวียนโลหิตบริเวณผิวหนัง ซึ่งประเมินโดยการไหลเวียนโลหิตบริเวณผิวหนังและอุณหภูมิผิวหนังในผู้สูงอายุและผู้ป่วยบาดเจ็บไขสันหลัง ผลการทบทวนงานวิจัยจำนวน 18 เรื่อง พบว่าไม่ว่าจะเป็นผู้สูงอายุที่สุขภาพแข็งแรงและเจ็บป่วยหรือผู้ป่วยบาดเจ็บไขสันหลังก็มีการทำงานของระบบการไหลเวียนโลหิตบริเวณผิวหนังบกพร่องทั้งสิ้น นอกจากนี้ยังพบว่าผิวหนังที่ถูกกดทับร่วมกับการทำงานของระบบการไหลเวียนโลหิตบริเวณผิวหนังที่บกพร่องเป็นปัจจัยเสี่ยงที่สำคัญของการเกิดแผลกดทับในผู้ป่วยกลุ่มนี้ ดังนั้นการหาวิธีการที่มีประสิทธิภาพในการส่งเสริมการไหลเวียนเลือดโลหิตและลดอุณหภูมิผิวหนังจึงมีความสำคัญต่อการปฏิบัติและการวิจัยทางการพยาบาลต่อไป

คำสำคัญ: *การไหลเวียนโลหิตบริเวณผิวหนัง, บาดเจ็บไขสันหลัง, ผู้สูงอายุ, แผลกดทับ*

Introduction

Many clinical guidelines, best practice guidelines, risk assessment tools and treatment modalities for care of pressure ulcers have been developed based on findings from research.[1-4] However, hospitalized patients, especially the elderly and those with a spinal cord injury, continue to develop pressure ulcers. In Thailand, the incidence of pressure ulcer development among hospitalized Thais ranges from 11.2% to 47%.[5-7] A pressure ulcer is defined as any skin lesion caused by constant pressure to the skin and muscle resulting in damage to underlying tissue.[8]

Pressure ulcers prove to be costly for patients, their families and health care facilities,[9-10] and have been shown to affect the person with a pressure ulcer in terms of pain, infection, altered body image and decreased quality of life.[11-12] The etiology of pressure ulcer development is multifactorial. Pressure, shearing force and friction, in combination with impaired tissue tolerance, are factors that alone and in combination lead to development of pressure ulcers.[13-14] The development of a pressure ulcer is also effected by skin microcirculation. Although impaired skin microcirculation and pressure ulcer development has been discussed in the literature, the information has not been presented in an organized manner. Thus, the purpose of this paper is to present, in an organized manner, an overview of available published research regarding skin microcirculation and what is known about the function of skin microcirculation in hospitalized elderly and patients with a spinal cord injury. It is hoped that this review will be useful both for practice and further research.

Skin microcirculation is the regulation of blood flow to the smallest level of vessels, including the arterioles, capillaries and venules. It is regulated by the nervous system, as well as in the skin tissue via maintenance of blood flow in the face of pressure changes in the vessels. Skin microcirculation is also an indicator of the skin tissue's tolerance for pressure and oxygenation.[14] Assessment of an individual's skin microcirculation can be accomplished through measurement of: skin blood flow (SBF); skin temperature (Ts); transcutaneous oxygen (tcO_2) and transcutaneous carbon dioxide ($tcCO_2$) level; and the reactive hyperemia (RH) response to pressure loading.[15-16]

The main functions of skin microcirculation are to support the: 1) flow of nutrients to the skin; and, 2) regulation of body temperature.[15] Typically, blood flows through one's capillaries, facilitating the exchange of gases and nutrients between one's blood and tissue, in a process known as nutritional flow.[15] The amount of nutritional flow can be determined through measurement of the skin's transcutaneous oxygen and carbon dioxide levels.[17] The body's internal temperature is controlled by the flow of blood through vessels in the subcutaneous tissues, i.e. arterioles, so as to transmit internal body heat to the skin via conduction, convection, and evaporation.[15] This blood flow is considered to be non-nutritional and often is referred to as shunt flow.[15] Non-nutritional blood also is called thermoregulatory flow, since it is instrumental in controlling body temperature. Thermoregulatory flow can be determined by measurement of skin temperature, skin blood flow and reactive hyperemia response.[15]

Skin microcirculation is both centrally and locally controlled.[16] The vasomotor center, located at the hypothalamus, centrally controls blood flow via neurotransmission of signals to the sympathetic nervous system at the dermis, thereby controlling vasomotion, the constriction and dilatation, of the skin blood flow.[15-16] In contrast, local control of blood flow is auto-regulated by the process of maintaining pressure and skin blood flow at the local area.[16] Autoregulation includes the phenomena of (active) hyperemia (increased blood flow in response to increased tissue metabolite) and reactive hyperemia (increased blood flow after release of arterial occlusion).[15-16] Reactive hyperemia is considered to be a preventive physiological response, after the release of pressure loading, to prevent tissue damage and restore the skin blood flow to a normal level.[15] A decrease in skin blood flow, in response to prolonged pressure loading, also may cause increased tissue hypoxia and ischemia.[18]

It is assumed that prolonged increase of one's skin temperature may lead to decreased tissue tolerance. Generally, each increase of 1 °C in body temperature results in approximately a 10% increase in tissue metabolism.[19] The consequences of increased tissue metabolism include: increased oxygen consumption, increased tissue carbon dioxide production and increased tissue waste productions. All of these, ultimately, lead to tissue ischemia and pressure ulcer development.[20-21] Changes in skin temperature, in response to loading pressure, are considered to be indicators of skin blood flow.[15] Increased skin temperature can be an indication of tissue inflammation in response to a developing stage I pressure ulcer.[22] Thus, changes in one's skin blood flow and skin temperature may be useful in predicting pressure ulcer development. In particular, hospitalized individuals, with impaired skin microcirculation, seem to be at high risk for pressure ulcer development when exposed to prolonged pressure loading in combination with shear and friction force.

The development of pressure ulcers is believed to be multifactorial. Extrinsic factors interacted with intrinsic factors are known to be crucial risk factors for pressure ulcer development. Extrinsic factors include pressure loading, shear and/or friction force. Elderly patients and patients with spinal cord injuries are exposed to loading pressure by lying on the same position, such as supine position or lateral position, for a prolonged time. Shearing force occurs when a patient slides down in the bed when a head of bed is elevated higher than 30 degree. Friction can cause skin damage when a patient is dragged over the surface of bed linens.[1,13] These three forces when interact with intrinsic factors can alter microvascular function of these groups of population.

The intrinsic factors include aging and pathophysiology of spinal cord injury. The aging process often leads to impaired vasomotion skin blood flow due to decreased elasticity of the vessels, even in healthy elderly individuals.[23] The elderly, in particular, are prone to impaired vasomotion, due to the aging process and their underlying pathology, i.e. stroke or diabetes.[24-25] In addition, patients with a spinal cord injury usually have impaired skin microcirculation function, due to disruption of their sympathetic nervous pathways, which, in turn, leads to impaired vasomotion of their capillaries and thermoregulation.[20] Thus, both hospitalized elderly and patients with spinal cord injuries are considered at high risk for pressure ulcer development.[23]

Search engines used to collect literature for review for this paper included Medline, CINAHL, and Pub-Med. Keywords used to locate published research on skin microcirculation included "skin temperature", "skin blood flow", and "skin microcirculation". Keywords used to locate published research on development of pressure ulcers included "pressure ulcer", "pressure sore", and "bed sore". In addition, the keywords "spinal cord injury" and "elderly" were used to seek published studies on patients with a spinal cord injury and hospitalized elderly, which also might contain findings regarding skin microcirculation and pressure ulcer development among both populations. The abstracts of all attainable research using the aforementioned keywords which had been published between 1990 and June 2008 were reviewed. Only those studies that addressed the selected keywords in regards to human subjects were chosen for review, yielding a total of twenty-four studies. Six of the 24 studies were quickly excluded from review, as an initial reading determined them to be irrelevant to the study. Thus, 18 studies were reviewed for this paper; their findings are presented in Table 1 and Figure 1.

Table 1 Skin microcirculation studies in elderly and patients with spinal cord injuries (SCI)

Authors	Sample	Main outcomes	Methods	Pressure duration (minutes)	Findings
Sae-Sia, et al., 2007[18]	Acute SCI (n=20), orthopedic trauma (n=35), healthy adults (n=47)	- Ts - SBF - RH response	- Sacral SBF and Ts measured simultaneously in the following order: lateral, supine, and lateral position	120	- Decreased SBF in acute SCI group on supine position compared to acute orthopedic and healthy subjects ($P<0.01$)
Thorfinn, et al., 2006[35]	SCI (n=8) and healthy subjects (n=8)	- SBF - RH response	- Left tuberosity SBF and RH response measured before, during, and after short and long period of loading pressure	3 & 15	- Increase in preload SBF and RH response in SCI subjects greater than in healthy subjects
Sae-Sia, et al., 2005[7]	CVA (n=11), SCI (n=6)	- Ts	- Sacral Ts measured during and after loading pressure	Did not identify	- Ts during and after loading pressure in subjects who developed a PrU was higher ($P<0.01$) than in those who did not develop a PrU
Baldwin, 2001[30]	38 ICU patients: 71% with neurological problems	- TcO_2 / $TcCO_2$ - Ts	- TcO_2 / $TcCO_2$ and Ts measured before and after loading pressure - Subject observed for gross (impaired movement) or subtle (intact movement) movement	120	- Pre-pressure Ts was 0.8 °F lower than that during loading pressure - Subjects with impaired movement had relatively flat TcO_2 / $TcCO_2$ compared to intact movement subjects
Springle, et al., 2001[37]	Disability patients (n=65) with pressure induced erythema at risk area for PrU	- Ts	- Ts at risk area compared with intact area	Not applicable	- 63% of subjects had increased Ts at the risk area compared to intact area

Table 1 (Continued)

Authors	Sample	Main outcomes	Methods	Pressure duration (minutes)	Findings
Schubert, 2000[22]	Elderly patients (n=28) with PrU stage 2 and 3	- SBF - TcO_2	- SBF and $TcPO_2$ measured at the ulcer area and in a reference area - Decreased TcO_2 at the ulcer area	Not applicable	- Increased RH response at the ulcer area
Yamasaki, et al., 2000[36]	- Adult Paraplegics (n=7) - Able-bodied subjects (n=7)	- SBF - Ts - Body temperature	- Subject rested at ambient temperature (25 °C, 50% humidity) and SBF, Ts at thigh, and body temperature were measured - Then, subject sat in a chamber at 33 °C and 50-55% humidity	60	- Subjects with high lesion of SCI (T6-T10) showed no increased SBF, whereas the lower lesion of SCI subjects (T11-T12) showed increased SBF in the hot environment
Knox, 1999[27]	Elderly residents (n=26)	- Body temperature - Ts	- Ts measured at sacrum, right and left trochanters at 1, 1.5 and 2 hr turning intervals	60, 90, 120	- Negative relationship b/w body temperature and Ts - Increased duration of pressure related to increased Ts ($P > 0.05$)
Bogie, et al., 1995[32]	SCI injured with lesion below (n=19) or above T6 (n=23)	- TcO_2 / $TcCO_2$	- TcO_2 & $TcCO_2$ measured at ischial tuberosity while sat on a cushion	30	- SCI subjects with lesions below T6 showed decreased SBF than those with lesions higher than T6
Knox, et al., 1994[26]	Healthy elderly (n=16)	- Ts at 1, 1.5, and 2 hr loading interval	- Threee groups of subjects based on turning position: right trochanter, left trochanter, and supine - Subjects lay on each loading interval	60, 90, 120	- Ts at the 2 hr turning interval was the greatest Ts and significantly different from the other two-time intervals

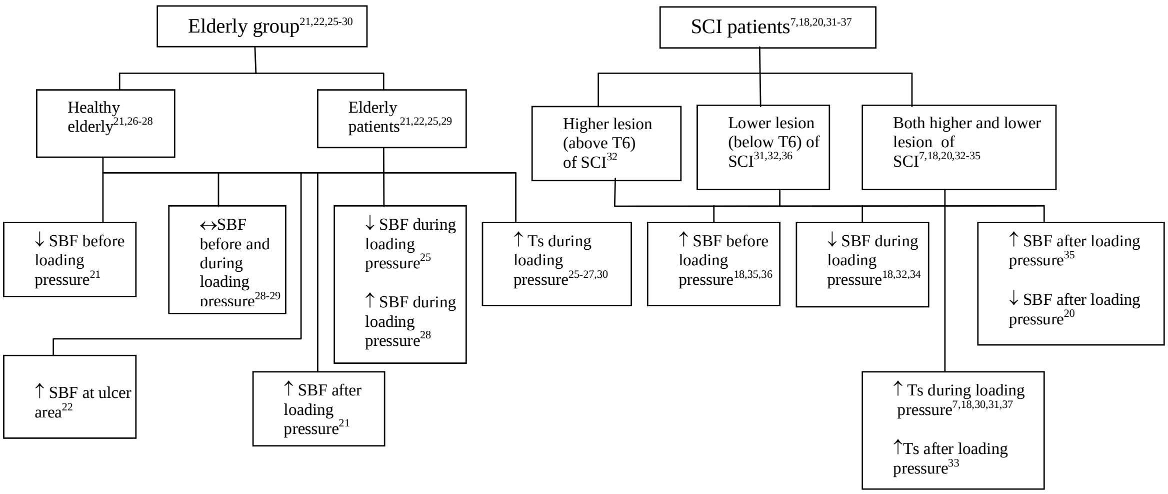
Table 1 (Continued)

Authors	Sample	Main outcomes	Methods	Pressure duration (minutes)	Findings
Schubert, et al., 1994[25]	- Group1 (G1) (n=20) "not at risk" elderly patients - Group 2 (G2) (n=10) "high risk" elderly patients with CVA	- SBF - Ts - RH response	- Sacral SBF & Ts measured in supine (0^{o}) and recumbent (45^{o}) positions - SBF and Ts at lateral position (baseline) measured for 3 min, then at either supine (0^{o}) or recumbent 45 position for 30 min, and then lateral position again	30	- Increased Ts in G1 was greater than in G2 during loading pressure - Increased Ts positively correlated with peak RH at supine position in both groups, but not in 45^{o} position
Barbenel, et al., 1993[31]	- Young paraplegics (n=6) - Young healthy adults (n=10)	- SBF - Ts	- SBF and Ts measured at thigh with no plastic surface insulation and then measured again with plastic surface insulation	2-3	- Healthy subjects had higher Increased SBF &Ts when applying plastic surface insulation compared to paraplegia - No change in SBF in paraplegics while increased Ts during applying plastic surface insulation
Frantz, et al., 1993[29]	Elderly > 60 years old (n=16) who high risk for PrU	- SBF	- Subjects lying on a low air loss mattress - SBF measured at greater trochanter before and during loading pressure	60	- SBF before and during pressure loading was not significant difference
Xakellis, et al., 1993[28]	Younger (n=19) and older adults (n=22)	- SBF	- SBF measured on a low air loss mattress	60	- Both groups showed increased SBF during pressure loading but differences not significant

Table 1 (Continued)

Authors	Sample	Main outcomes	Methods	Pressure Duration (minutes)	Findings
Mawson, et al., 1993[34]	SCI subjects (n=21) - Able-bodied controls (n=11)	- TcO_2	- Sacral TcO_2 measured before and during pressure loading	30	- TcO_2 before and during loading pressure in SCI subjects was significantly lower than in controls
Schubert, et al., 1991a[20]	- Group 1 (G1) =10 tetraplegia - Group 2 (G2) =10 paraplegia - Group 3 (G3) =10 healthy adults	- Ts - SBF - RH response	- Sacral Ts and SBF was measured before, during, and after loading pressure	3	- Ts during loading pressure in G2 was significantly lower than in G1 and G3 - Increased RH in SCI group was less than in healthy adults
Schubert, et al., 1991b[21]	- Group 1 (G1) = 10 healthy young adults - Group 2 (G2) = 10 healthy elderly subjects - Group 3 (G3) = 10 elderly patients	- Resting SBF (rSBF) - RH response	- SBF measured before, during,and after external pressure loading device	3	- No significant difference in rSBF b/w groups - the rSBF in G3 significantly lower than G2 , but no significant difference b/w G3 and G1 - Peak RH response was significantly lower in G3 than in G1
Finestone, et al., 1991[33]	SCI young patients (n=6)	- Ts	- Sacral Ts measured on each loading and unloading pressure period	60-240	- Increased Ts was found after release of loading pressure

Notes: Ts = skin temperature, SBF = Skin blood flow, RH = reactive hyperemia response, LDF = laser Doppler flowmeter, TcO_2 = transcutaneous oxygenation, $TcCO_2$ = transcutaneous carbon dioxide, b/w = between, CVA = cerebrovascular accident, PrU = pressure ulcer



Note: SCI = spinal cord injury, T6 = spine injury at thoracic level 6, Ts = skin temperature, SBF = Skin blood flow, ↑ = increase, ↓ = decrease, ↔ = no change

Figure 1 Skin blood flow and skin temperature in elderly group and patients with spinal cord injuries

Skin microcirculation in hospitalized elderly patients and patients with spinal cord injuries

All 18 reviewed studies employed an experimental design. The first finding that the review of the studies revealed was that skin microcirculation, skin temperature, skin blood flow, transcutaneous oxygenation and carbon dioxide, and reactive hyperemic response were different among hospitalized elderly patients as well as among patients with spinal cord injuries when compared with healthy adults. All but one of the studies found that a relationship existed between skin temperature, skin blood flow and loading pressure in: healthy elderly[21,26-28], ill elderly[22,25,29], and patients with spinal cord injuries.[7,18,20,36]

Although half of the studies (9 of 18 studies) measured only one of the parameters (SBF, Ts, or TcO_2/$TcCO_2$),[7,26-29,32-34,37] the others combined two (SBF and Ts, SBF and RH, or SBF and TcO_2/$TcCO_2$)[21,22,31,35-36] or three parameters (SBF,Ts and RH; Ts, Ts and TcO_2/$TcCO_2$; or, SBF, Ts and RH)[18,20,25,30] to assess the functioning of skin microcirculation. Pressure loading across all studies varied from 3 minutes to 2 hours.[7,9-14,17-37] Only one study measured, among patients with spinal cord injuries, the effect of the position of sitting on skin temperature over a one to four hour period.[33] However, two of the studies did not identify the length of time of pressure loading during their investigation.[7,37]

Various types of equipment were used to measure skin blood flow, reactive hyperemia response, skin temperature and transcutaneous oxygen and carbon dioxide levels. Two types of equipment were used to measure the subjects' skin blood flow or reactive hyperemia response. Although nine studies [6,18,20-21,25,28-29,31,36] employed a laser Doppler flow meter (LDF) sensor/probe to measure skin blood flow or reactive hyperemia response, one study[35] used a laser Doppler perfusion imager to measure skin blood flow.

Half of the studies used a thermocouple skin temperature sensor to measure skin temperature. The other studies used either a disposable Thermistor temperature probe[26-27] or a tympanic thermometer[30] to measure skin temperature. All of the studies used transcutaneous oxygenation and carbon dioxide content sensors to measure the transcutaneous oxygen and carbon dioxide levels.[22,30,32,34]

Skin microcirculation in the elderly group

Five studies recruited healthy elderly as a comparison group with healthy adults or hospitalized elderly.[21-22,28-30] Skin blood flow in the healthy elderly, both during pressure loading and unloading, was not found to be significantly different, compared with healthy adults.[21,28] However, the hospitalized elderly in one study were found to have impaired skin blood flow when the area examined was not under pressure, as well as during pressure loading and after release of pressure loading.[21]

Schubert and Fagrell[21] found the baseline skin blood flow, during the absence of pressure loading over the sacrum, of hospitalized elderly (mean age = 85 years) to be significantly lower than that in healthy elderly and healthy adults. They also found significantly less external pressure was required to stop skin blood flow, when loading was applied to the sacrum of the hospitalized elderly, compared to healthy elderly and healthy adults. The peak skin blood flow, after release of pressure loading, also known as reactive hyperemia response, of the hospitalized elderly was found to be significantly lower than that of the healthy elderly and healthy adults. However, one study found no significant difference in skin blood flow during loading and unloading pressure in hospitalized elderly patients lying on a low air loss mattress, a group at high risk for pressure ulcer development.[29]

It also was found that the skin blood flow in the sacral area of hospitalized elderly patients following a cerebrovascular accident decreased 28% when supine for 30 minutes and 14% when in a semi-Fowler position. On the other hand, the skin blood flow in the sacral area of healthy elderly, when supine, increased 35% and 13% when in a semi-Fowler position.[25]

Two studies measured skin temperature of healthy elderly.[26-27] However, only one study measured skin temperature of hospitalized elderly patients.[25] Within these three studies, one[25] measured skin temperature in relation to skin blood flow, whereas the other two[26-27] measured only skin temperature. Skin temperature, among the healthy elderly, during loading pressure, was higher than that of the hospitalized elderly patients.[25] This finding suggests there was more skin blood flow to the occluded area among the healthy elderly than there was among the hospitalized elderly patients. No significant relationships were found in the hospitalized elderly patients between increased duration of loading pressure and increase in skin temperature, or between body temperature and skin temperature.[26-27] These insignificant findings may have been due to the heterogeneity of the subjects, the small sample size, or methodological flaws.

Skin microcirculation in individuals with spinal cord injuries

All studies found that those with spinal cord injuries had impaired skin microcirculation, compared to healthy adults. For example, patients with an acute spinal cord injury, within 24-96 hours after incurring the injury, showed decreased sacral skin blood flow, over 2 hours of loading pressure, in a supine position, compared to patients with acute orthopedic trauma and healthy subjects.[18,35] Thorfinn, et al.[35] found that patients who had experienced a spinal cord injury 3 months to 3 years prior to being studied, were noted to have an increased skin blood flow during unloading of pressure and an increased reactive hyperemia response after release of the pressure, compared to healthy persons. However, Schubert and Fagrell[20] found that patients with spinal cord injuries usually had less of an increased reactive hyperemic response in their sacral area, compared to healthy adults

The level and extent of the spinal cord injury also was found to affect the skin microcirculation function.[18] However, the results appear to be inconclusive. Patients with a spinal cord lesion below T6, had a progressive decrease in ability to maintain ischial tuberosity skin blood flow, determined by transcutaneous oxygen level while sitting on a support cushion in normal ambient temperature, compared to patients with a spinal cord lesion higher than T6.[32] On the other hand, Yamasaki, et al.[36] found that patients with spinal cord lesion of T11-T12 had an increase in thigh skin blood flow in a hot environment (33 °C), compared to those with a spinal cord lesion of T6-T10. One needs to note that it is difficult to compare these findings due to their use of different measurement sites, sample size and level of lesion.

All reviewed studies revealed that the patients with spinal cord injuries had impaired thermoregulation functioning. Those with an acute spinal cord injury had less of an increase in skin temperature, when under pressure loading for 2 hours, compared to healthy subjects and patients with acute orthopedic problems.[18] The same group of patients with spinal cord injuries also had decreased skin blood flow during exposure to 2 hours of pressure loading. Other studies reported finding that patients with spinal cord injuries had less of an increase in skin temperature with prolonged exposure to a plastic surface compared to healthy adults.[31] These findings suggest that less of an increase in skin temperature when exposure to a plastic surface indicates decreased skin blood flow to the occluded area, which may lead to increased tissue waste production, tissue necrosis and pressure ulcer development.

Skin microcirculation in hospitalized elderly and patients with spinal cord injuries who developed a pressure ulcer

Patients in the reviewed studies who had a pressure ulcer also had impaired skin microcirculation function.[22,34] Patients with spinal cord injuries, upon measure of their transcutaneous oxygenation, were assessed as having diminished nutritional blood flow. Half of the subjects, who had transcutaneous oxygenation below the median transcutaneous oxygen level, developed a pressure ulcer, compared to only 9% of patients with spinal cord injuries who had transcutaneous oxygenation above the median transcutaneous oxygen level.[35] Similarly, among hospitalized elderly, the area surrounding the pressure ulcer was found to have decreased transcutaneous oxygenation, compared to nearby, intact, non-pressure ulcerated skin.[22] These findings suggest that both patients with spinal cord injuries and hospitalized elderly with a pressure ulcer had diminished tissue oxygenation which may have lead to tissue ischemia and/or impaired wound healing.

Five studies[18,20,31,33,36] revealed that there was an alteration in the skin temperature of patients with spinal cord injuries who developed a pressure ulcer. Sae-Sia, et al.[18] found that patients with spinal cord injuries who developed a pressure ulcer were more likely to have increased skin temperature during pressure unloading, compared to those who did not develop a pressure ulcer. Although no significant difference was found between the two groups, perhaps due to the small sample size, the same trend, compared to a prior study[37] was found. The researchers[7] found that patients with a stroke, or with a spinal cord injury, who developed a stage I pressure ulcer had a mean sacral skin temperature 24-96 hours before development of the pressure ulcer of 1.2 ^{o}C. This sacral skin temperature was significantly higher than the sacral skin temperature of those who did not develop a pressure ulcer. In addition, two of the studies found that patients with a stage I pressure ulcer had increased skin temperature at their erythematic site, either during loading or unloading pressure, compared to the control site.[33,37] These findings suggest that increased skin temperature at a Stage I pressure ulcer may be due to tissue inflammation.

Syntheses critique of studies and direction for practice and future research

The findings of these studies suggest that healthy elderly, hospitalized elderly and patients with spinal cord injuries are at high risk for development of a pressure ulcer due to impaired skin microcirculation. The strength of the majority of the studies was the use of a good experimental design, using two group[25,28-32,34-36] or three-group comparisons.[18,20-21] Five of the studies did use a single group design,[22,26-27,33,37] which may have resulted in less accuracy of their findings.

A limitation of most of the studies was the use of a small sample size.[18,20-21,25-26,29,31,35-36] The heterogeneous characteristics of the sample of some of the studies,[13,27,30] as well as their use of an unequal sample size of the groups studied in some with more than one group,[12,30] may also have lead to insignificant differences between the groups and to insignificant findings. Due to the use of different pressure loading durations, as well as the use of different equipment to measure skin temperature, comparison across the studies was difficult. Therefore, future studies need to use an increased sample size and assure homogeneity of the sample studied; i.e. hospitalized elderly with the same diagnosis, patients with a spinal cord lesion at the same level, or comparable groups of quadriplegics or paraplegics, which would make the findings more valid and generalizable. In addition, future research should seek strategies that increase skin blood flow and decrease skin temperature during exposure to loading pressure.

The implications for practice of these reviewed studies are directly related to the fact that hospitalized elderly and patients with spinal cord injuries are at high risk for pressure ulcer development. Thus, healthcare providers, when providing care to these groups of patients, need to be aware of preventing pressure ulcers. Pressure ulcer prevention guidelines need to be developed and implemented for individuals at high risk of pressure ulces beginning with their first day of hospitalization. Such guidelines should include: 1) a turning interval no greater than every 2 hours, to reduce arterial occlusion and release heat accumulation between the skin and support surface, and 2) gradual changing of the position of the patient with a spinal cord injury to decrease his/her reactive hyperemic response and reduce abrupt changes of skin blood flow after the release of loading pressure, which would reduce the shearing stress at the capillaries and minimize injury to the tissue, thereby decreasing the potential for pressure ulcer development. However, future studies are needed to monitor the incidence of pressure ulcer development when implementing these preventive nursing care techniques.

Furthermore, review of the studies suggests that hospitalized elderly, as well as patients with spinal cord injuries, who develop a pressure ulcer, have higher skin temperature than do those who do not develop a pressure ulcer. Therefore, skin temperature appears potentially to be a useful objective indicator, in addition to other paper-pencil pressure ulcer screening tools (e.g. the Braden Scale),[18] to assess the risk of pressure ulcer development, especially among hospitalized elderly and patients with spinal cord injuries. In addition, skin temperature might be a useful indicator in monitoring the effectiveness of various strategies, i.e. hospital mattresses or wheelchair cushions, designed to prevent the development of pressure ulcers.

Conclusions

The main risk factors regarding pressure ulcer development in elderly group and patients with spinal cord injuries involve a combination of loading pressure, shear stress, friction, aging process, and impaired skin microcirculation indicated by decreased skin blood flow and increased skin temperature. The information gleaned from this review of 18 related published studies suggests that healthcare providers need to be aware of the potential for pressure ulcer development in hospitalized elderly and in patients with a spinal cord injury, since both have impaired skin microcirculation. Healthcare providers also need to implement effective strategies to promote skin blood flow and decrease skin temperature, in order to reduce the incidence of pressure ulcer development in the hospitalized elderly, as well as in patients with spinal cord injuries. Further research in relation to skin microcirculation in both these populations is needed.

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