Effect of Alternating Pressure Overlay on Skin Blood Flow and Interface Pressure in Spinal Cord Injury

BY

RUDRI MILIND PUROHIT

B.S., Sancheti College of Physiotherapy, 2015

THESIS

Submitted as partial fulfillment of the requirements for the degree of Master of Science in Rehabilitation Sciences in the Graduate College of University of Illinois at Chicago, 2019
Chicago, Illinois

Defense Committee:

Alexandar.S. Aruin, Professor, Physical Therapy, Chair and Director of Graduate studies.

Yi-Ting Tzen, Assistant Professor, Physical Therapy, Advisor

Aileen Eviota, Clinical Assistant Professor, Physical Therapy.

This thesis is dedicated to my father, Milind Purohit, my mother, Harsha Purohit, and my sister Aastha Joshi, without whom it would have never been possible

ACKNOWLEGEMENTS

I appreciate the support and guidance of many people who have helped me through this research project from the bottom of my heart. I would like to thank: Dr. Yi-Ting Tzen for providing me the best guidance for the development of the project and helping me through each and every part of the research study. Dr.Alex Aruin for providing me positive guidance and being a strong support throughout this research project. My beloved parents Milind Purohit and Harsha Purohit for continuous support both day and night, remotely from India. My beloved sister, Aastha Purohit Joshi, for being my constant strength and inspiration throughout this master's journey.

My best friends Aboli Ajgaonkar, Kinjal Sangani, Shraddhali Sawant, Saurabh Bandhavkar, and Siddhika Kore for constantly supporting me and motivating me throughout this journey.

TABLE OF CONTENTS

List of Tables

List of Figures

SUMMARY

Pressure injuries are serious complications that are difficult and expensive to treat. Preventative strategies, such as alternating pressure mattress has been prescribed to patients with pressure injury in the hospital setting to prevent recurrence. However, the underlying mechanism of alternating pressure (AP) on tissue tolerance to ischemia is not explored to a full extent. The main purpose of this study was to investigate the protective mechanism of alternating pressure overlay on load-bearing tissue like sacrum in people with spinal cord injury (SCI). We hypothesized that the low-profile AP overlay would redistribute the interface pressure and subsequently increase the skin blood flow (SBF) to the sacral area when lying on top of an operating room (OR) pad. A secondary aim was to investigate if AP overlay could be used as a preconditioning strategy for weight-bearing skin at the sacrum. We hypothesize that after preconditioning with AP overlay, the interface pressure would remain unchanged during supine lying on OR, but SBF would increase. A repeated measures design was utilized on 15 adults with SCI. All the individuals underwent three protocols: supine lying on (1) OR pad only for 40 minutes (control), (2) OR pad with AP overlay for 40 minutes, and (3) OR pad only (AP-OR) for 40 minutes after AP (protocol 2). Outcome measures included interface pressure and SBF at sacrum. For the primary aim, we found that the peak interface pressure during APdeflation (51.47±30.18 AU) was significantly lower than that during AP-inflation (89.27±53.92 AU) and OR (control protocol) (114.13±60.97 AU). The averaged SBF during AP-deflation

(15.54±15.33 AU) was significantly higher than that during inflation (12.65±12.45 AU) and OR

(control) (11.96±10.26 AU). For secondary aim, we found that peak interface pressure during

AP-OR (104.62±58.17 AU) was significantly lower than that during OR (control) protocol (114.13±60.97 AU), and SBF during AP-OR (15.78±15.82 AU) was significantly higher than that during OR (control) (11.96±10.26 AU). Findings of this study suggested that alternating pressure overlay is an effective strategy to redistribute interface pressure and subsequently improves tissue perfusion at the weight-bearing area in people prone to pressure injury.

Further study is warranted in other population at risk of pressure injuries

1. INTRODUCTION

A. Background

People with SCI are at risk of developing a pressure injury due to several risk factors such as sensory deficit, limited mobility and incontinence (Groah et al. 2015). It is one of the most common (41%) secondary complications occurring in SCI population next to urinary tract infection (62%), and autonomic dysreflexia (43%) during the first year of injury (McInnes et al. 2012). It was reported that 17.7% of individuals with SCI have been re- hospitalized due to occurrence of pressure injury during the first year following injury and the incidence of re hospitalization has increase to 34.4% by the 20th year post spinal cord injury (Groah et al. 2015). The most common sites for developing a pressure injury in patients with SCI is the sacrum (43%), and the second most common site is the heels (19%) followed by ischial tuberosities (15%) (Kruger et al. 2013). The treatment expenditure for a single full-thickness pressure injury ranges from $20,000-$70,000 and the annual cost for treating pressure injury goes up to approximately 11 million dollars (Redelings, Lee, and Sorvillo 2005). The average monthly cost of treating one pressure injury in community dwelling individuals with SCI is around $4,765 and among that the maximum percentage of cost was attributed to the admission to hospital (Chan et al. 2013). Pressure injuries, once developed, are very difficult to treat and the cost of management of a pressure injury is expensive. Therefore, preventive strategies are crucial for high-risk population.

General recommendations for preventing a pressure injury include a thorough and frequent risk assessment, repositioning of patient, assessment of skin especially on bony prominences every, and avoidance of prolonged pressure exposure (Groah et al. 2015). Despite preventive options like repositioning the body every 2 hours to relieve the pressure, routine skin care, assisted active and passive movement of the limbs below the level of lesion, pressure injury remains a challenging issue for the healthcare industry (Arias et al. 2015). Using suitable support surfaces is recommended to help prevent pressure injury (Groah et al. 2015; Jan et al. 2011), and its effectiveness is researched and utilized in healthcare settings as a measure of redistributing prolonged pressure over bony prominences (Masterson and Younger 2014a).

Support surfaces are "specialized devices for pressure redistribution designed for management of tissue loads, microclimate, and/or other therapeutic functions (i.e., any mattress, integrated bed system, mattress replacement, overlay, or seat cushion, or seat cushion overlay" (NPUAP, Support surfaces standard initiative, 2017). Alternating pressure is one of the active characteristics of support surface often prescribed for patients with existing pressure injuries. Previous studies showed that patients who used alternating pressure support surfaces had lower incidence of pressure injury (McInnes et al. 2012). However, the underlying mechanism of such effect is not fully explored, especially in the high risk population, such as SCI.

B. Objective

The purpose of our study is to investigate the efficacy of low-profile AP overlay on interface pressure and skin blood flow in weight-bearing tissues (sacral area) in individuals with chronic SCI. The **primary aim** of this study is to investigate the protective mechanisms of alternating pressure overlay in comparison to the regular operating room (OR) pad in redistributing the interface pressure and subsequently increasing the skin blood flow to avoid tissue ischemia when the areas are loaded. The **secondary aim** of our study is to investigate if AP could be used as a preventive strategy to precondition the tissue before it is exposed to long term weight-bearing condition.

C. Hypothesis

For **primary aim: AP effect on interface pressure and SBF,** six hypotheses were tested.

1) Participants will demonstrate **lower interface pressure** at sacrum in supine lying during deflation cycle as compared to inflation cycle of AP overlay protocol.

2) Participants will demonstrate **higher skin blood flow** at sacrum in supine lying during deflation cycle as compared to inflation cycle of AP overlay protocol.

3) Participants will demonstrate **lower interface pressure** at sacrum in supine lying during inflation cycle of AP overlay as compared to OR pad only (control).

4) Participants will demonstrate **higher skin blood flow** at sacrum during supine lying during inflation cycle of AP overlay as compared to OR pad only (control).

5) Participants will demonstrate **lower interface pressure** at sacrum in supine lying during deflation cycle of AP overlay as compared to OR pad only (control).

6) Participants will demonstrate **higher skin blood flow** at sacrum during supine lying during deflation cycle of AP overlay as compared to OR pad only (control).

For **secondary aim: lasting effect of AP (preconditioning) on interface pressure and SBF,** two hypotheses were tested.

7) Participants will NOT demonstrate **difference in interface pressure** between supine lying on OR after AP (AP-OR) as compared to lying on OR pad only (control).

8) Participants will demonstrate **higher skin blood flow** during supine lying on OR after AP (AP-OR) as compared to lying on OR pad only (control).

D. <u>Significance</u>

Although previous studies have tested the effect of alternating pressure surfaces as wheelchair cushions in seating position to unload the ischial tuberosities or in lying position to unload heel in the intensive care unit (ICU), they have not been widely tested in load-bearing conditions in individuals with SCI in a laboratory setting. The potential effect of AP overlays on pressure redistribution and tissue perfusion remains unclear (Jan et al. 2011; Rithalia 2004). Findings from this study helped us understand the potential mechanisms of AP strategy in preventing the occurrence of pressure injuries in individuals with chronic SCIs.

2. REVIEW OF LITERATURE

A. Pressure Injury

Pressure injuries previously known as pressure ulcers, pressure sores, bed sores (The National Pressure Ulcer Advisory Panel, 1989), decubitus ulcers (Kosiak 1961), have been recently defined by The National Pressure Ulcer Advisory Panel in 2016 as "A pressure injury is localized damage to the skin and/or underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear" (Edsberg et al. 2016b). The most common sites of developing a pressure injury are bony prominences like the sacrum, the heels, and ischial tuberosities among others (Bansal et al. 2005).

The incidence of pressure injury is high in populations that are immobilized or bedridden for a long period of time, such as patients in ICU, mechanically-ventilate patients, post-surgery long-term immobilized individuals, individuals with SCI and elderly individuals that are bedridden for long period of time. The incidence of pressure injury in acute care patients ranges between 0.4-38% , 3.3-39.3% in critically-ill patients admitted to ICU (González-Méndez et al. 2018), 3-33.9% in skilled nursing facilities homes (Tran et al. 2016) up to 17% in home care settings (Manzano, Colmenero, Pérez-Pérez, Roldán, Jiménez-Quintana, et al. 2014), and up to 29% in individuals that are mechanically-ventilated. The incidence of pressure injuries in surgical care units varies between 5.5- 66% (Defloor 2000; Defloor and De Schuijmer 2000). For high-risk populations such as people with SCI, the prevalence of pressure injury in acute rehabilitation care is about 49% and about 30 % in chronic stage (Sunn 2014). It is also recognized as one of the most prevalent secondary complication of SCI (National Spinal Cord Injury Statistical Center,

2006).

A.1. Alternating pressure mattress

AP mattress used in the prevention of pressure injuries has a long history. A prospective trial that tested the efficacy of AP mattress and water mattress showed that similar number of patients developed pressure injuries in both groups; however, the number was significantly lower than the control group subjected to standard mattress (Andersen et al. 1983). The potential benefit of AP mattress was demonstrated in one study that AP mattress system significantly reduced interface pressure at the trochanters in healthy subjects (mean deflation pressure: 30, mean inflation pressure: 67, mean hospital mattress pressure: 95 mmHg) (Vanderwee, Grypdonck, and Defloor 2008). The other study evaluating four types of AP mattresses in the lab setting also supported that the AP mattresses were capable of momentarily reducing interface pressure and that contact pressures were significantly reduced in individuals where the inflation pressure was adjusted according to the body mass of the individual (Rithalia and Gonsalkorale 1998). To date, there was no clear evidence of any specific alternating pressure mattress outperform the others on the market (Rithalia and Gonsalkorale 2000), however a review study revealed that large-celled AP mattress was more effective that small-celled mattress in preventing pressure injuries (Bliss and Thomas 1993).

A.2. Alternating pressure overlay

Alternating pressure overlay has mechanism of alternating and redistributing pressure similar to that of alternating pressure mattress (Bethell 1994). One study showed that there was no significant difference between the AP and silicone overlays in individuals with chronic neurological disease in case of healing time, incidence and duration of pressure injuries, however both overlays were more effective than a foam overlay (Conine, Daechsel, and Lau 1990). One

study found that, as compared to other cost-effective overlays on the market, AP overlays demonstrated effectiveness in reducing the rates of occurrence of pressure injuries and also healed the existing pressure injuries in individuals that failed to regularly be positioned (Bliss 1995). Most recently, a low-profile AP overlay was tested to investigate its effect on hospital-acquired pressure injuries during surgeries, and the results showed that AP was 100% successful in preventing hospital-acquired pressure injuries during operations (Joseph et al. 2019). Despite the positive features of AP overlay, there is no evidence thus far showing that AP overlays demonstrate the same effectiveness in preventing pressure injuries as compared to AP mattresses. There was only one study which compared pressure incidence on mechanical ventilated patients in ICU. They found that alternating pressure mattress had lower pressure injury incidence as compared to AP overlay (Manzano, Colmenero, Pérez-Pérez, Roldán, Jiménez-Quintana Mdel, et al. 2014), this suggested that AP mattress is more effective in preventing pressure injuries than AP overlay.

B. Pressure injury etiology

Previous studies suggested that pressure injuries are a multi-factorial medical condition (Nixon, Cranny, and Bond 2005). The most critical factors leading to the development of a pressure injury are prolonged or sustained pressure leading to tissue ischemia, tissue reperfusion injury and cell deformation (Herrman et al. 1999b; Peirce, Skalak, and Rodeheaver 2000; Yarkony 1994).

Prolonged pressure is common in bed-ridden, immobilized and individuals undergoing long surgeries (Kosiak 1961). Based on the inverse relationship between amount and duration of pressure exposure (Reswick and Rogers 1976), the chances of tissue breakdown increase with exposure to

prolonged or sustained pressure (Nixon, Cranny, and Bond 2005). Prolonged exposure to pressure leads to occlusion of blood (ischemia); this results in accumulation of waste products and lack of oxygen supply, and subsequently tissue breakdown (Bader et al. 2005).

In addition to prolonged pressure, shear force is another factor contributing to tissue ischemia. Shear force is "the force per unit area exerted parallel to the plane of interest"(Black et al. 2007). A previous study showed that in the presence of shear force, magnitude of pressure required to cause lack of blood flow to the tissue is significantly less compared to the magnitude of pressure alone (Goossens et al. 1994).

Reperfusion injury occurs when prolonged tissue ischemia is removed and leads to liberation of free radicals. This results in inflammatory reactions, cellular infiltration, advance cell damage and cell death (Nixon, Cranny, and Bond 2005). Pressure injury could also result from mechanical deformation of cells. The pressure induced compression leads to changes in the cell volume, cell diameter and structure (Bouten et al. 2001; Jan and Brienza 2006). These changes trigger a cascade of tissue breakdown leading to tissue necrosis and cell death (Breuls et al. 2003). These researches have also suggested that pressure injury formation could occur directly by cell deformation (Breuls et al. 2003).

C. Pressure injury risk factors

There are multiple risk factors that lead to pressure injury. Immobility is the primary risk factor of pressure injury. This is supported by the fact that high-risk population of pressure injury are mostly immobile, for example people with SCI (Byrne and Salzberg 1996; Chen, DeVivo, and

Jackson 2005), critically-ill patients in an ICU (Masterson and Younger 2014b), intraoperative units and surgical units (Xiong et al. 2019; Walton-Geer 2009) elderly people (Allman 1989; Amlung, Miller, and Bosley 2001; Horn et al. 2004), and in individuals suffering from other critical diseases (Bergstrom et al. 2005).

Other risk factors include aging, increased duration of mechanical ventilation, diabetes, vasopressor administration, hypotension and presence of cardiovascular disease in ICU patients (Li et al. 2016; Cox 2017). In the elderly population, other risk factors include underlying pathology (cardiovascular, peripheral vascular, lung disease), primary illness (infection or hip fractures), functional status of performing activities, poor nutrition or swallowing difficulties and co-morbidities like diabetes or dementia (Jaul 2010).

For people with SCI, despite motor and sensory dysfunctions, spinal cord injury leads to partial or complete disruption of the autonomic nervous system which provides sympathetic and parasympathetic innervation from the brainstem and the hypothalamus to the spinal cord. As a result, the hypothalamus and the brainstem fail to regulate the inter mediolateral cells of the spinal cord. The inter mediolateral cell columns that are located in the spinal cord, in turn, fail to modulate and respond to the various stimuli below the level of lesion. These stimuli usually include pressure, temperature, moisture and shear among others (Brown et al. 2007a). Failure to regulate and respond to various stimuli in addition to being immobile and bedridden for a prolonged period of time often leads to pressure injuries (Brown et al. 2007a). Pressure injuries can occur in acute, subacute as well as chronic stages of SCI (Groah et al. 2015). The risk of pressure injuries were less in the first 10 years of SCI and incidence increased 15-year post injury (Chen, DeVivo, and Jackson 2005).

Pressure injury risk factors in people with SCI include impaired sensation (Caliri 2005), lack of

mobility (Marin, Nixon, and Gorecki 2013), bladder and bowel incontinence (Chen, DeVivo, and Jackson 2005), and impaired autonomic system (Brown et al. 2007b), cognitive status (Horn et al. 2002), poor nutrition (Krishnan et al. 2017; Brurok et al. 2012), functional status post-injury (Groah et al. 2015), and associated medical condition like pneumonia (Krishnan et al. 2017).

C. <u>Pressure injury management</u>

The treatment expenditure for a single full-thickness pressure injury can go up to $ 70,000 and the annual cost for the treatment of pressure injuries comes up to approximately 11 million dollars (Redelings, Lee, and Sorvillo 2005). Pressure injury management is difficult and very expensive. Management of existing pressure injuries include multiple measures such as repositioning the body and early mobilization to cause pressure relief in the areas where the pressure injury developed. The individual must be repositioned continuously even though a pressure redistributing surface is being used (Edsberg et al. 2016a). Inspecting the skin damage regularly to avoid worsening of existing PIs and occurrence of new PIs is an important aspect in the management of pressure injuries ('WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): An Executive Summary' 2017). For existing stage I/II pressure injuries at the heel, it is necessary to "float the heels from off the support surface or the bed or by using heel suspension devices" (Sunn 2014). For the treatment of Stage III/IV and unstageable it is necessary to "place the leg in a device that elevates the heel from the surface of the bed, completely offloading the pressure ulcer. Consider a device that also prevents foot drop"(Taradaj 2017). Wound dressings play a vital role in the treatment of existing pressure injuries and the choice of wound dressings must be based on how deep the wound is and the nature of the biofilm present in the wound (Sunn 2014). Debridement of wound and negative pressure wound therapy is the treatment of choice for the treatment of stage II/IV pressure injuries (Sunn 2014). Surgical

intervention might be necessary for severe pressure injuries that need surgical debridement and wound dressings (Groah et al. 2015). Although there are numerous treatment options for the management of pressure injuries, the treatment of pressure injuries is expensive and also leads to complicated morbidities and could prove fatal for an individual at high-risk of worsening and recurrence of a pressure injury (Edsberg et al. 2016a).

D. Pressure injury prevention

Pressure injuries have always been a great challenge on the entire healthcare industry as well as the health insurance industry (Tran et al. 2016). Due to this, prevention of pressure injuries has become an essential part and the most cost-effective way in dealing with pressure injuries (Reddy, Gill, and Rochon 2006). Management approaches for the prevention of pressure injuries include regular repositioning of the subject, and early mobilization in the form of passive movements or active-assisted movements are recommended (Tran et al. 2016). Minimizing pressure on medical devices such as oxygen tubes, cervical collars, urinary catheters, and casts during acute care settings is an important aspect of preventing medical device related pressure injuries ('WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): An Executive Summary' 2017). In addition, usage of heel suspension devices to offload the pressure from the heels and using ointments such as creams, gels and pastes as incontinence skin barriers in individuals who have bladder and bowel incontinence is recommended in patients at risk of developing pressure injuries ('WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): An Executive Summary' 2017). Preventive strategies such as regular skin assessment, risk-factor assessment, maintenance of proper nutrition, avoidance of risk habits like smoking, alcohol consumption are some of the methods used to prevent the occurrence of a pressure injury (Groah et al. 2015). In addition to the above interventions, prophylactic dressings

are widely used in the past decade for pressure injury prevention (Blenman and Marks-Maran 2017). Overall, a thorough risk assessment, along with regular skin assessment and repositioning in addition to early mobilization, usage of prophylactic dressings on pressure prone areas, administration of appropriate nutrition is mainstay in the prevention of pressure injuries (Taradaj 2017).

E. Emerging techniques for pressure injury prevention

Many new and innovative techniques have emerged in the past few years in an attempt to decrease the incidence of pressure injuries in individuals at risk (Tran et al. 2016). Professional organizations such as National Pressure Ulcer Advisory Panel and Wound, Ostomy and Continence Nurses Society as well as European Pressure Ulcer Advisory Panel recommend the use of support surfaces which help in pressure redistribution and reduction of shear in acute care and home-care settings ('WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): An Executive Summary' 2017; Taradaj 2017). These pressure redistributing surfaces "increase the body surface area that comes in contact with the support surface (to reduce interface pressure) to sequentially alter the parts of the body that bear load, thus reducing the duration of loading at any given anatomical site" (Taradaj 2017).

A support surface is "a specialized device for pressure redistribution designed for management of tissue loads, micro-climate, and/or other therapeutic functions (i.e. any mattresses, integrated bed system, mattress replacement, overlay, or seat cushion, or seat cushion overlay)" (Edsberg et al. 2016a). Although the cost of these support surfaces varies widely, Medicare only covers a small variety of support surfaces that are efficient in pressure redistribution at the bony prominences (Harris et al. 2020). Previously, support surfaces were categorized on the basis of the

material they were made (Brienza, Geyer, and Jan 2005) and then as static surfaces and dynamic surfaces on the basis of their mechanism of action (Liu et al. 2012). More recently, support surfaces are categorized as reactive support surfaces, active support surfaces, integrated bed systems, non-powered/ powered surfaces, overlays and mattresses (Shi, Dumville, and Cullum 2018). A systematic review on the use of support surfaces for treatment and prevention of pressure injuries suggested that a structured foam mattress is more effective than the hospital mattress in preventing pressure injuries, and low air-loss mattress is more effective than the mixed-pulsating type to avoid pressure injuries at the heel, and alternating pressure type to be superior than the visco-elastic type for heel ulcers (Colin et al. 2012). An alternating pressure overlay is considered to be an efficient measure to reduce pressure and prevent the occurrence of preoperative and postoperative pressure related injuries (Colin et al. 2012). Another review suggested that though the efficacy of AP mattresses over constant-low pressure mattresses were unclear, AP mattresses "were associated with an 80% probability of reducing costs owing to a delay in pressure ulcer formation and reduced length of hospital stay when they were used" (Stannard 2012).

F. Ischemic conditioning

Since tissue ischemia and reperfusion injury are the main etiological factors of pressure injury, strategies that could potentially minimize either pathways are warranted for further investigation (Epps and Smart 2016). Due to the feature of AP overlay, we suspect that AP may be used to prevent pressure injuries by preconditioning the skin before long-term ischemia. Ischemic preconditioning is a strategic phenomenon that is implemented for preventing tissue or organ damage due to ischemia and reperfusion, done by subjecting the same tissue or organ with a single or multiple non-lethal episode of ischemia (Benstoem, Nahrstedt, et al. 2017). It has been

proven to be effective in prevention of myocardial infarction and stroke in previous studies (Hausenloy and Yellon 2008). Regional ischemic preconditioning demonstrated positive effects on the prevention of subsequent coronary artery occlusion by providing brief periods of ischemia in dogs (Przyklenk et al. 1993). It was suggested that brief periods of ischemia results in endogenous activation of protective mechanistic pathways such as the neural, humoral and systemic inflammatory pathway, which leads to the prevention of recurrence and reduction of infarction (Hausenloy and Yellon 2008). This has led to recent advanced research in induction of non-cardiac organ/ tissue ischemia, showing significant effects in preventing cerebral and renal injuries in individuals undergoing renal transplantation (Pickard et al. 2015).

Other studies also showed the benefits of ischemic post-conditioning in preventing complications post cardiac surgeries and recurrence of ischemic stroke (Nayak and Kerr 2013; Benstoem, Stoppe, et al. 2017). One study showed that repetitive ischemia was induced during early reperfusion period post-ischemia (post myocardial infarction) proved to be beneficial in reducing the myocardial infarct size and also was found to be useful post cardiovascular surgery by reducing the risk of recurrence or secondary complications. When post-conditioning (60 min occlusion and 3 hours reperfusion) was compared to pre-conditioning (5 min occlusion and 10 min reperfusion), both groups demonstrated similar effects in reducing the infarct size and preserving endothelial function. Although post-conditioning has better and realistic clinical expectations in terms of myocardial infarction (Délot et al. 2003), pre-conditioning could prove of better help in preventing long-term damage and morbidities.

A recent study showed that repeated ischemic conditioning had beneficial effects on diabetic ulcer healing in individuals with unhealed diabetic foot ulcer (Epps, Dieberg, and Smart 2016). The diabetic state is one of the conditions that is highly prone to ischemic-reperfusion injuries (Shaked et al. 2015). Ischemic conditioning was applied repeatedly in the form of

ischemia for 3-5min and reperfusion led to an improvement in endothelial function, improvement in the skin microcirculation and also release of systemic modulatory inflammatory markers (Epps, Dieberg, and Smart 2016) as per the proposed neural, humoral and systemic theories (Hausenloy and Yellon 2008; Loukogeorgakis et al. 2005). Another study applied ischemic conditioning on individuals with aseptic or infected diabetic foot ulcers for 3 times every 2 weeks and followed- up over 6 weeks (Shaked et al. 2015). They found that repeated ischemic conditioning caused significant healing of the foot ulcers (Study group: 41% healed ulcers, Control group: 0%). These results suggested that ischemic condition is a simple, inexpensive, and safe approach that helps with wound healing (Boghossian et al. 2017).

3. METHODS

A. Study Design

We performed a repeated measures study design in which every participant acted as his/her own control. Each participant underwent two protocols: lying on top of the OR pad with and without the alternating pressure overlay. Figure 1 illustrates the research protocols.

Figure 1: Illustration of the full course of experimental protocols. The top panel is the experimental protocol: alternating pressure (AP) followed by a lying on top of OR pad only (APOR), and the bottom panel is control protocol: lying on top of OR pad only

The order of the two protocols was randomized to avoid crossover effect. A washout period of 30 minutes was provided in between the AP and OR (control) and a 15-minute washout period was provided between the AP and AP-OR protocol. Seven participants underwent regular operating room protocol (OR) first, and 8 underwent alternating pressure overlay (AP) first.

During both protocols, the participants were in side-lying position for 10 minutes (baseline), followed by 40 minutes of supine position to simulate the bed lying condition in the clinic.

Figure 2 demonstrates the subject positions and test setting.

Figure 2: Subject position. (Left) Subject in side-lying position for baseline measurements, (right) subject in supine position for simulated bed-lying conditions during the AP, OR and APOR.

For the AP protocol, a 5-minute-inflation-deflation cycle was applied during the 40 minutes of supine position, in other words there were four inflation/deflation cycles for each participant during AP. A session of AP-OR protocol was implemented after the AP protocol to test the potential immediate effect of AP overlay on SBF and interface pressure while lying on an OR pad as an indicator of potential ischemic preconditioning. A washout period of 30 minutes was provided between AP and AP-OR.

B. <u>Study Participants</u>

Fifteen adults between 18-64 years were recruited. All participants had SCI for more than

one year at level T10 and above. They were not ambulatory and required wheelchair for mobility. Individuals with a current pressure injury, history of cardiovascular or pulmonary diseases, diabetes mellitus, and hypertension were excluded from the study. All procedures of this study were approved by the institutional review board of the University of Illinois at Chicago. All participants signed written consent for this study before any procedures were implemented.

C. Instrumentation

Low-profile AP overlay was designed to redistribute pressure across the skin by alternating multiple rows of inflatable/de-flatable air cells in the overlay. The alternating pressure overlay (Dabir Surface Inc.) used in our research study is thin, portable, flexible, and designed to be placed over standard operating room pad. During peak inflation, the thickness of the alternating pressure overlay does not exceed 1 inch. A single inflation-deflation cycle lasts 10 minutes with inflation and deflation each being 5 minutes long. This time period is short enough to avoid ischemia-reperfusion injury (Jan et al. 2011). Interface pressure was measured by using X-sensor pressure mapping system (Xsensor X2, Xsensor Technology Corp., Alberta, Canada). It was sampled at 1Hz using the X3 Medical software (version 6). Pressure mapping system is a relatively new technology as compared to load cell or other devices used to record and interpret the amount of pressure applied by any object (Stinson, Porter-Armstrong, and Eakin 2003). The X-sensor pressure mapping system is a thin, flexible pressure mapping mat with multiple pressure sensors in the form of force sensing arrays. It consists of 1600 pressure sensors in a two-dimensional force sensing array which are situated at a distance of 12mm apart (Bain 2011). It is placed directly in contact with the object or subject to be tested. In this research study, participants lay down over the pressure mapping system in addition to the AP overlay and OR pad depending on the protocol

(see figure 1 and 2).

The data collected through a pressure mapping system was computed and represented on the computer screen in three different forms: numerical data, three-dimensional contour grid and a color-coded contour map of the body (Stinson, Crawford, and Porter-Armstrong 2008b). Figure 3 demonstrates an example of pressure map readings from one of our subjects lying supine on the OR pad. As highlighted in the figure, when the cursor was moved on top of the highest-pressure point at the sacrum area, a small box indicating 3x3 cells of interface pressure reading is presented for actual readings of the nine cells at the selected location.

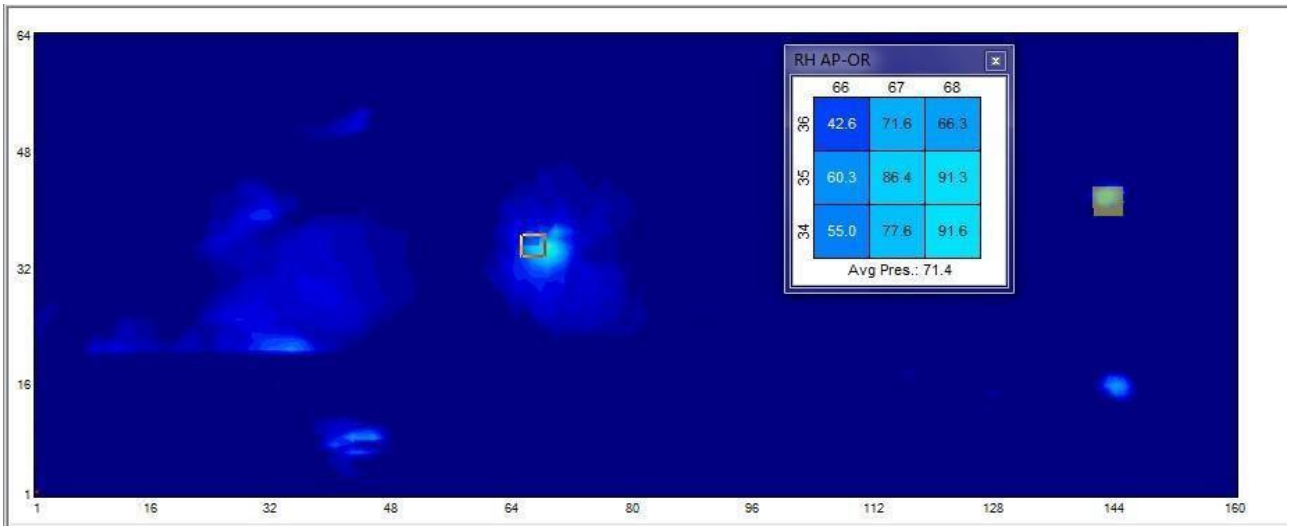

Figure 3: Example of pressure map readings. This is the interface pressure reading during supine on the OR pad on one of our subjects. The small box on the upper right is the 3x3 cells showing actual pressure readings around the area of interest.

There are several advantages of using a pressure mapping system over other pressure sensing devices, including visual clear representation of pressure, real-time feedback of the pressure recordings, thin and flexible sensor allowing minimum interference between the system and the body, and sensors being able to withstand high temperatures and humid climate conditions (Stinson,

Crawford, and Porter-Armstrong 2008a; Vanderwee, Grypdonck, and Defloor 2008). In order to capture the interface pressure readings for this study, the pressure mapping system was calibrated at 50-200 mm Hg as per the manufacturer's instructions. Figure 3 demonstrates the arrangement of pressure mapping system; OP overlay and the OR pad in this study.

Figure 4: Arrangement of study equipment.

Skin blood flow was measured by using the Laser Doppler flowmetry (LDF) equipment system (moor instrument, Wilmington, DE). It is a non-invasive tool used for measuring skin microcirculation by "utilizing the Doppler shift of laser light as the information carrier" (Tagawa 2011). LDF has been proved as a reliable tool for measuring tissue perfusion and also investigating the risk of occurrence of pressure injuries (Herrman et al. 1999a; Schubert and Fagrell 1991). The SBF was collected at the highest pressure point around the sacrum area in this study with the flat probe (VP11SC) head located at the center of the 3x3 indicated in the pressure map image. The

SBF was sampled at 20Hz using Power Lab/Lab Chart (version 8). Synchronization of the SBF and interface pressure data was done by recording time frames of data during baseline, change in position and at the beginning of each inflation-deflation cycle during AP protocol.

D. Data Analysis

The two primary outcome measures of this study were **SBF** and **interface pressure**. Both measures were recorded continuously throughout both procedures. The interface pressure number used for statistical analyses includes peak and averaged interface pressure among the nine sensors (area of interests demonstrated in figure 3. The **peak interface pressure** is the highest pressure among the nine sensors, and the **averaged interface pressure** is the averaged value among the nine sensors. Both values were used as parameters to quantify interface pressure across the highest-pressure points around the sacrum in previous literatures (Higer and James 2016; Arias et al. 2015). For the purpose of statistical analysis, mean values of SBF, peak interface pressure, and averaged interface pressure were calculated for the following time periods: the middle 38 minutes out of the 40 minutes supine lying for OR and AP-OR protocols, and the middle 3 minutes out of the 5 minutes of each inflation/deflation cycle for AP protocol. We did not include the first and last minute of the 40 minutes supine lying on OR pad and the first and last minute of 5 minutes inflation/deflation since we wanted to eliminate the artifact caused by transition between different postures or inflation/deflation cycle. The secondary outcomes were demographic information (age, gender, body mass index (BMI)), self-reported injury history and medical history, ASIA impairment scale (AIS), and the International Standards to Document Remaining Autonomic Function after Spinal Cord Injury (Cragg and Krassioukov 2012). Descriptive analysis was first computed for primary outcomes, and it suggested that interface pressure and SBF were deviated

from normal distribution. Therefore, non-parametric tests were used for analysis and comparison of SBF and interface pressure recorded during alternating pressure (AP) protocol and regular operating room (OR) protocol and AP-OR. Non-parametric tests are assumption-free tests used when the population distribution fails to follow the normal distribution curve which could be due to a smaller small size. Friedman's test for K-related samples (equivalent to one-way ANOVA for repeated measures) was used to compare means of peak interface pressure, average interface pressure and skin blood flow during the inflation and deflation cycles of AP protocol, the OR protocol and the AP-OR protocol. Bonferroni correction was made, and the P-value was set to <0.0125 as being significant for post hoc pair-wise comparison. Wilcoxin-signed rank test was used to compare peak as well as average interface pressure and skin blood flow recorded at sacrum during inflation and deflation cycles of AP protocol, OR and AP-OR protocols. Pearson's correlation was used to find out any correlations between BMI, medical history, autonomic scale with changes in interface pressure and SBF during various protocols. All the statistical analysis was performed using IBM SPSS statistics version 24.

4. RESULTS

A. Subject characteristics

Fifteen participants with chronic SCI were included in this study. The age of the participants ranged between 21-63 years. (Mean ± SD: 41.87± 14.58 years). BMI of the participants ranged between 22-37 kg/m^2 (Mean ± SD: 26.81±4.12 kg/m^2) with a weight of 79.6 ± 15.7 kilograms (kg) and height of 1.73±0.156 meters. The duration of injury of the participants ranged from one year to 48 years. (17± 14.62 years). Table 1 shows the subject characteristics of 15 participants recruited for the research study. Five participants had complete SCI (AIS A), and 10 participants had incomplete SCI (AIS B). Out of 15 participants, 13 participants had previous history of pressure ulcers located either at sacrum or at heels. Eleven participants used power wheelchair for mobility, and four participants used manual wheelchair for mobility.

Table 1: Subject characteristics.

Subject ID	Age (years)	Gender (M=male, F=female)	BMI (kg/m^2)	Duration of injury (years)	Level of injury	AIS	Previous pressure injury history	Wheelchair
1	55	M	25.1	13	T10	A	Yes	Manual
2	29	M	23.1	11	T4	B	Yes	Power
3	62	F	30.9	48	T10	B	Yes	Power
4	55	F	27.1	13	T10	B	Yes	Power
5	33	M	30.3	8	T6	B	Yes	Power
6	28	M	23.7	7	T7	B	Yes	Power
7	48	M	24.5	25	T1	B	Yes	Power
8	54	M	36.7	25	T10	A	Yes	Manual
9	48	M	31.4	20	T7	A	No	Manual
10	37	F	22.6	4	T10	A	Yes	Manual
11	51	M	29.2	27	T8	B	Yes	Power
12	21	M	21.7	4	T10	B	Yes	Power
13	60	F	24.1	45	T4	A	Yes	Manual
14	27	M	26	4	T5	B	No	Power
15	20	M	25.7	1	C3	B	No	Power

B. <u>Primary aim: AP vs Crtl protocols</u>

Figures 5 and 6 demonstrate sample snapshots of pressure map readings from one subject during AP protocol and OR protocol respectively.

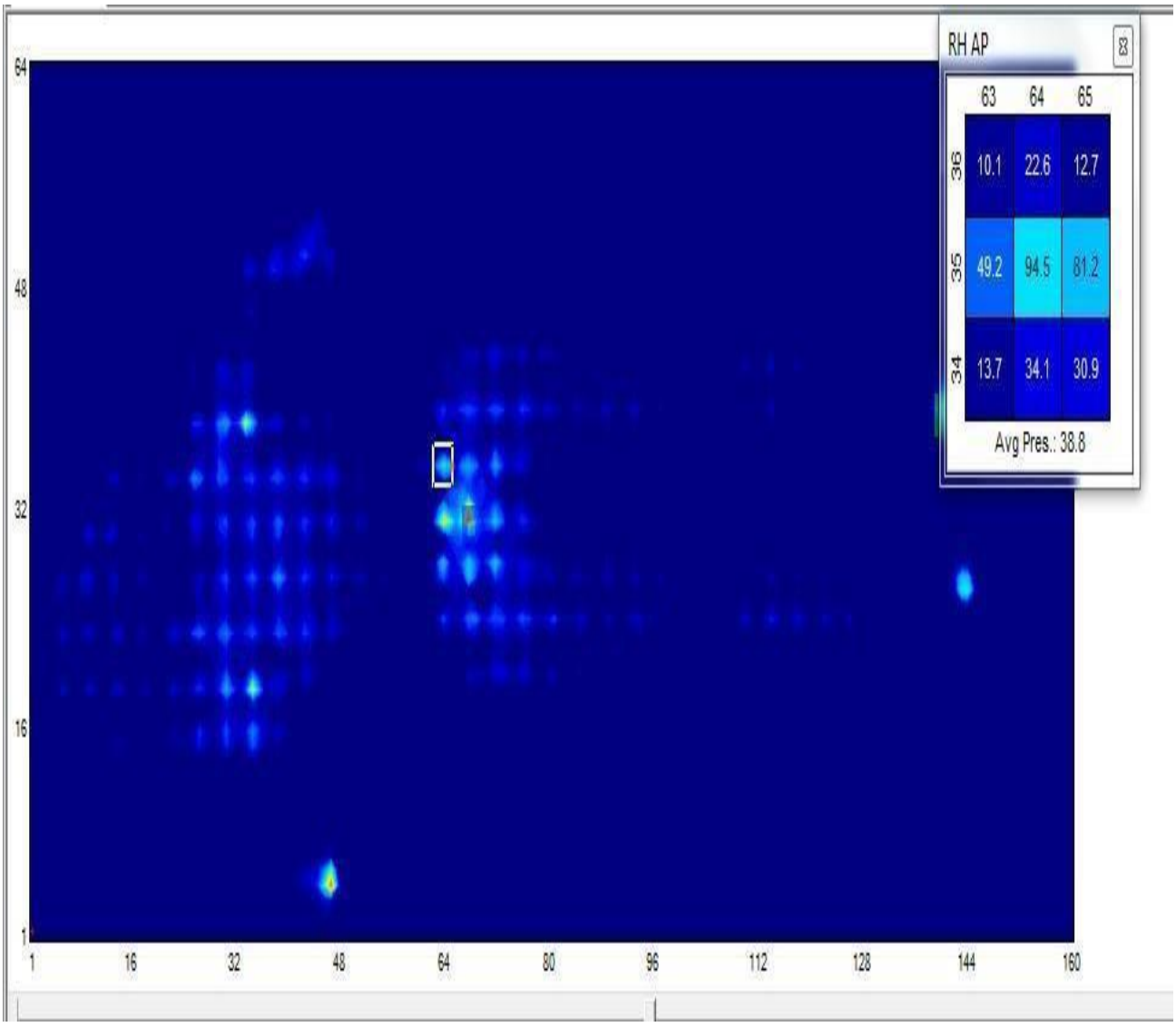

Figure 5: Pressure map reading snapshot during AP-protocol. The subject is in supine lying position, where his head is located at the left side of the pressure map, and heels located at the right side of the pressure map figure.

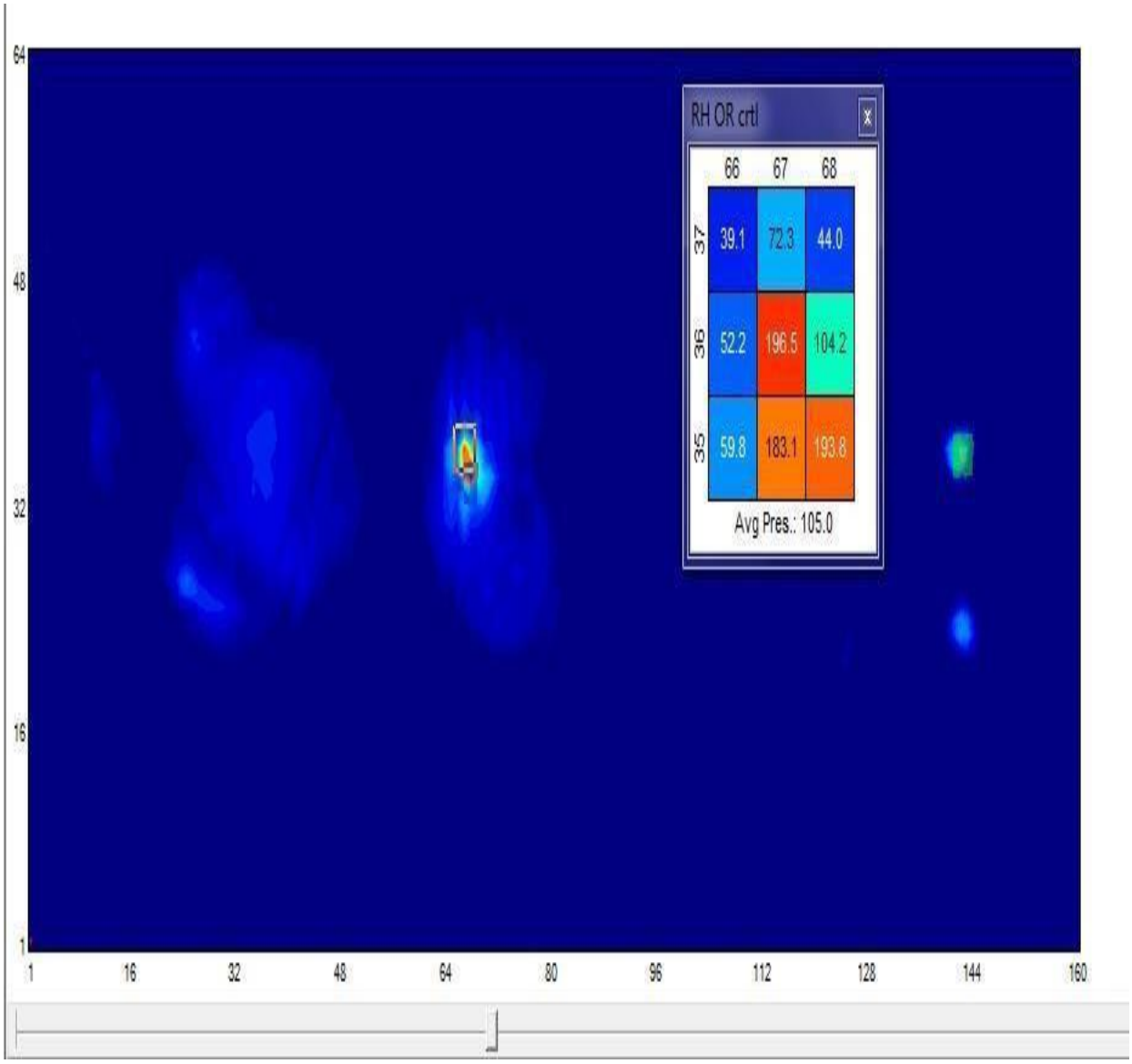

Figure 6: Pressure map reading snapshot during OR-protocol. The subject is in supine lying position, where his head is located at the left side of the pressure map, and heels located at the right side of the pressure map figure.

Figure 7 and 8 demonstrate sample SBF data from one subject during AP protocol and OR protocol respectively.

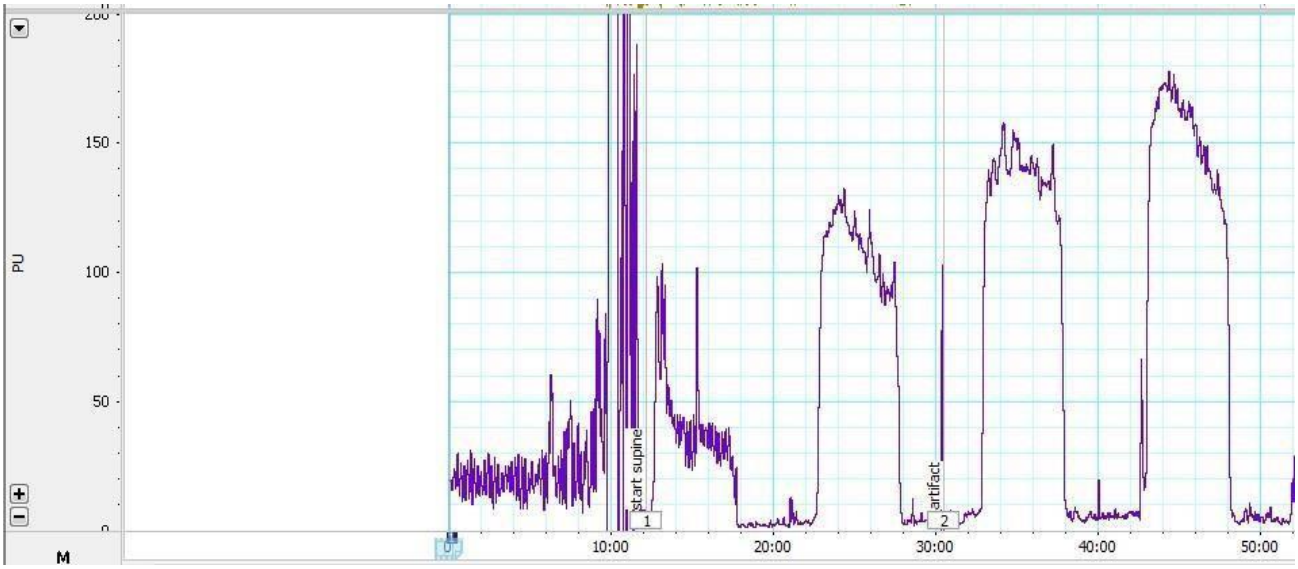

Figure 7: SBF during AP protocol for a subject.

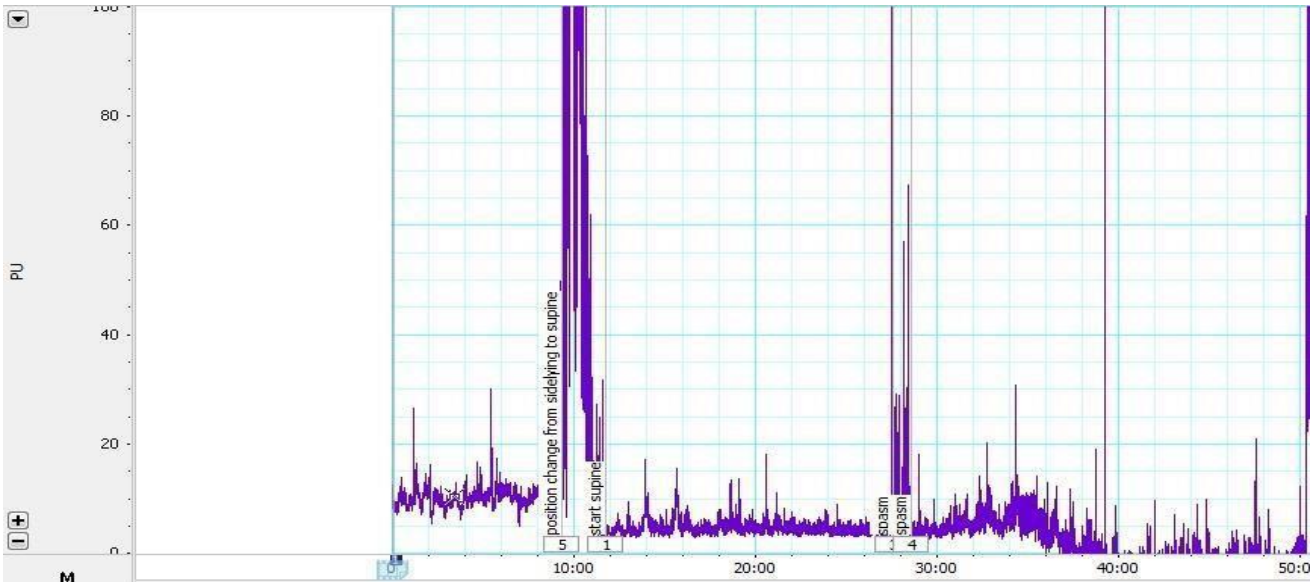

Figure 8: SBF during OR protocol for the same subject as figure 7.

Table 2 shows the mean and one standard deviation of the peak, averaged interface pressure and SBF during AP inflation, deflation and OR. Figures 9, 10 and 11 are boxplots of the peak, averaged interface pressure and SBF of all subjects during the three conditions respectively. Wilcoxon signed rank tests showed that the peak and averaged interface pressure during deflation were significantly lower those during inflation. (p<0.001 for both peak and average interface pressure, r=0.87 and $r=0$.87 for peak and averaged interface pressure respectively. We also found that the peak and averaged interface pressure during deflation were significantly lower than that during OR protocol (p=0.004, r=0.76 for peak pressure, and p=0.003, r=0.76 for averaged pressure). When comparing peak and averaged interface pressure between AP-inflation and OR, there was no statistical significance (p=0.173, r=0.35 for peak pressure, and p=0.1, r=0.42 for averaged pressure).

For the SBF, we found that it was significantly greater during deflation as compared to inflation (p=0.002, r=0.79), and as compared to OR protocol (p=0.023, r=0.56). When comparing SBF during inflation and OR protocol, there was no significant difference (p=0.023, r= 0.38).

Table 2: Mean ± one standard deviation of peak, averaged interface pressure, and SBF during AP and OR

Outcome Measures	ALTERNATING PRESSURE (Experimental)		OR (Control)
	Inflation	Deflation	
Peak interface pressure (mmHg)	89.27±53.92	51.47±30.18	114.13±60.97
Average interface pressure (mmHg)	57.21±28.54	36.16±18.47	81.50±46.39
Skin Blood flow (AU)	12.65±12.45	15.54±15.33	11.96±10.26

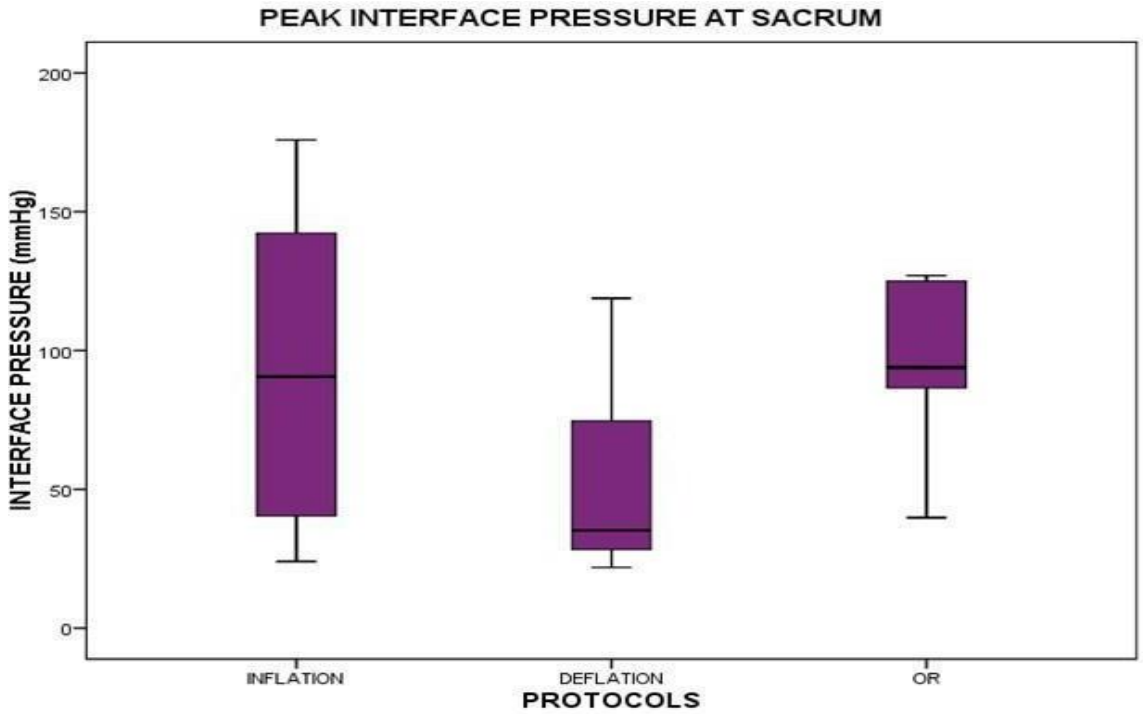

Figure 9: Peak Interface pressure at sacrum during AP inflation, AP deflation and OR (Control)

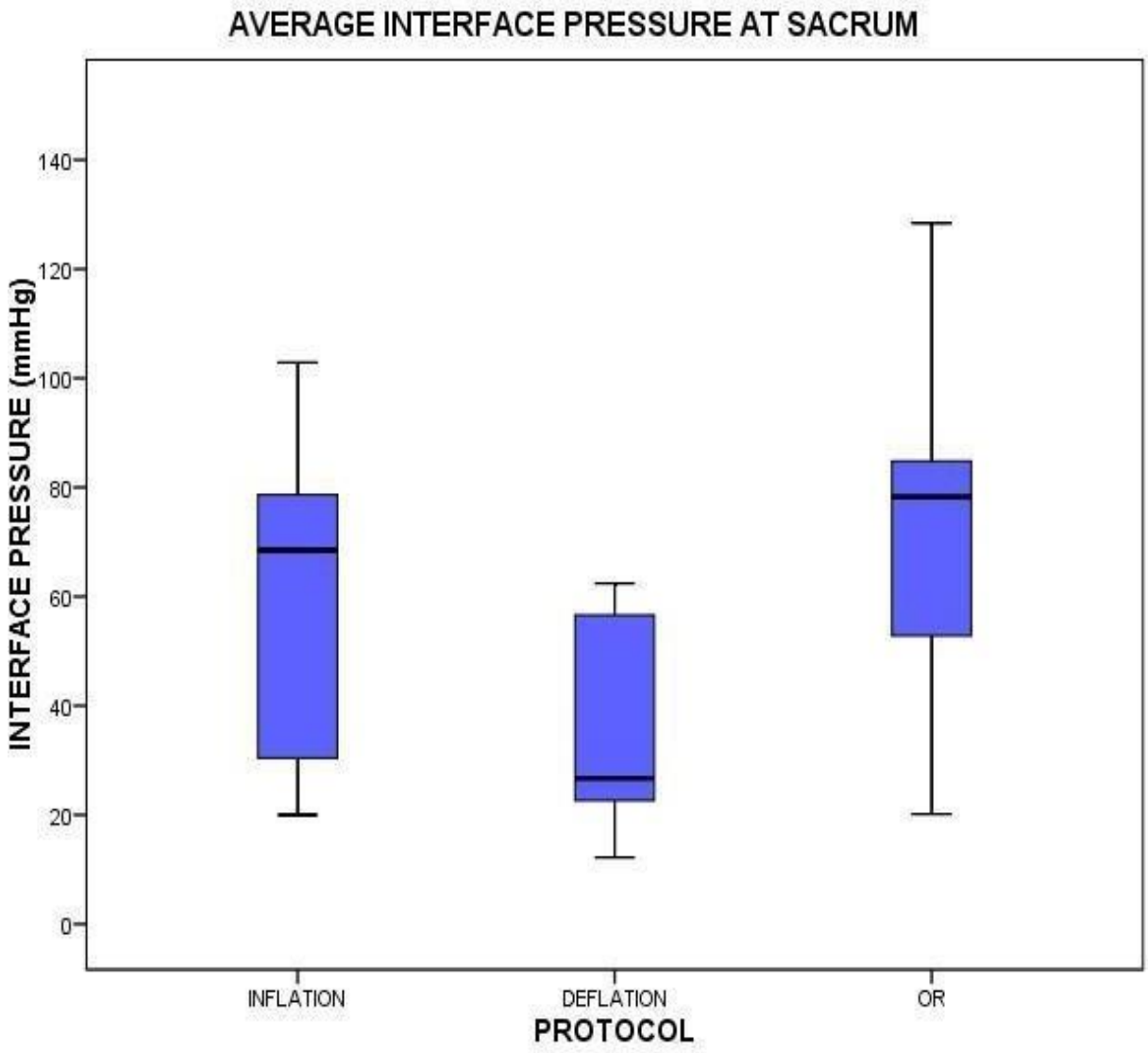

Figure 10: Average interface pressure at sacrum during AP inflation, AP deflation and OR (Control)

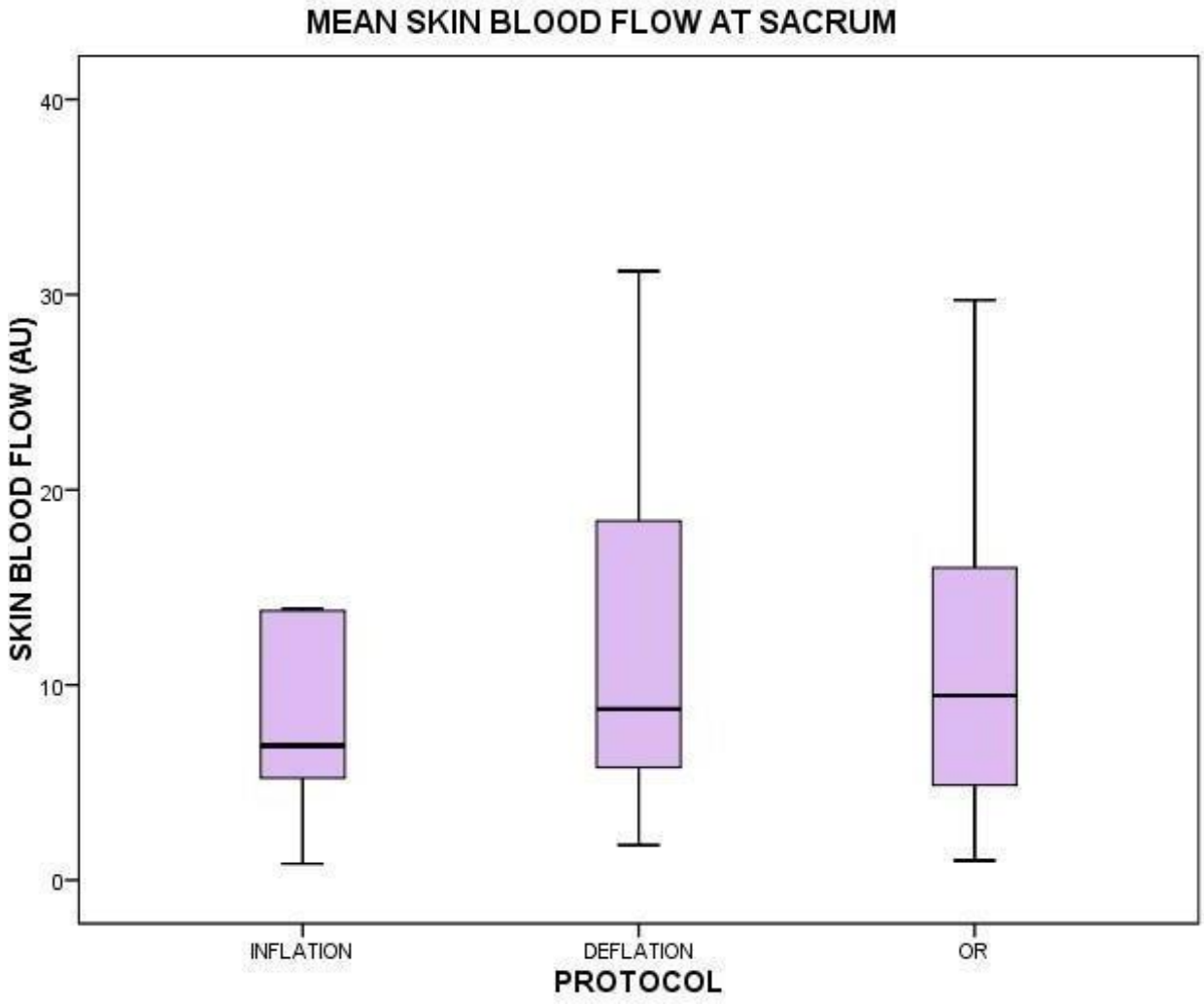

Figure 11: Mean skin blood flow at sacrum during AP inflation, deflation and OR (Control).

C. Secondary aim:AP vs Crtl protocols

Results shown in this section contains the interface pressure and SBF data from AP-OR protocol and the OR protocol presented in previous section. Figure 12 is a sample snapshot of pressure map reading from the same subject as figure 5 and 6 during AP-OR protocol. Figure 13 is a sample SBF data from one subject during AP-OR protocol.

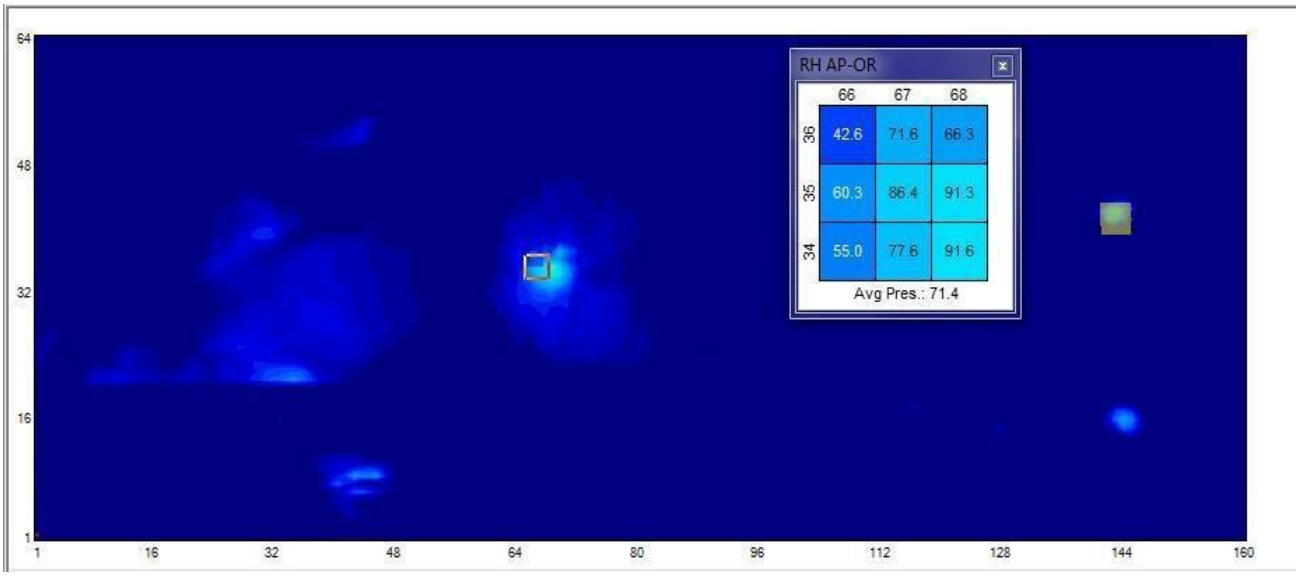

Figure 12: Pressure map reading snapshot during AP-OR-protocol. The subject is in supine lying position, where his head is located at the left side of the pressure map, and heels located at the right side of the pressure map figure

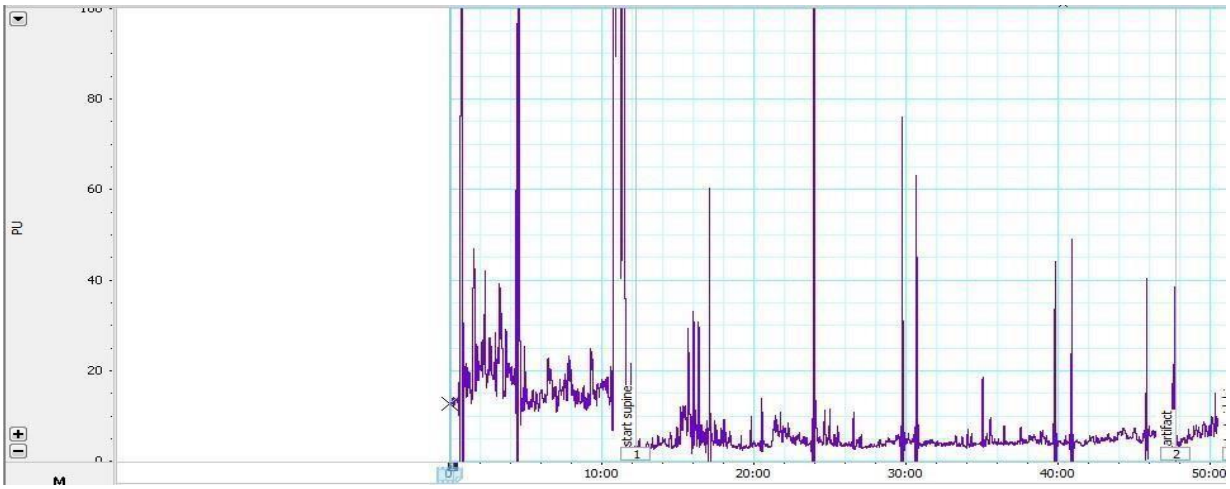

Figure 13: SBF during AP-OR protocol for the same subject as figure 7

Table 3 shows the mean and one standard deviation of the peak, averaged interface pressure and SBF during AP-OR and OR. Wilcoxon signed rank tests results are also included.

Figures 14, 15 and 16 are boxplots of the peak, averaged interface pressure, and SBF of all subjects during the two conditions respectively. We found that the peak interface pressure during AP-OR was significantly lower than that of OR ($p<0.005$, $r=0.70$). There was no statistical significance in averaged interface pressure between AP-OR and OR. We also found that the SBF during AP-OR was significantly higher than that of OR ($p<0.01$, $r = 0.64$).

Table 3: Mean ± one standard deviation of peak, averaged interface pressure, and SBF between OR and AP-OR.

Outcome Measures	OR	AP-OR	*P*
Peak interface pressure (mmHg)	114.13±60.97	104.62±58.17	0.005
Average interface pressure (mmHg)	81.50±46.39	81.07±48.62	0.955
Skin Blood flow (AU)	11.96±10.26	15.78±15.82	0.01

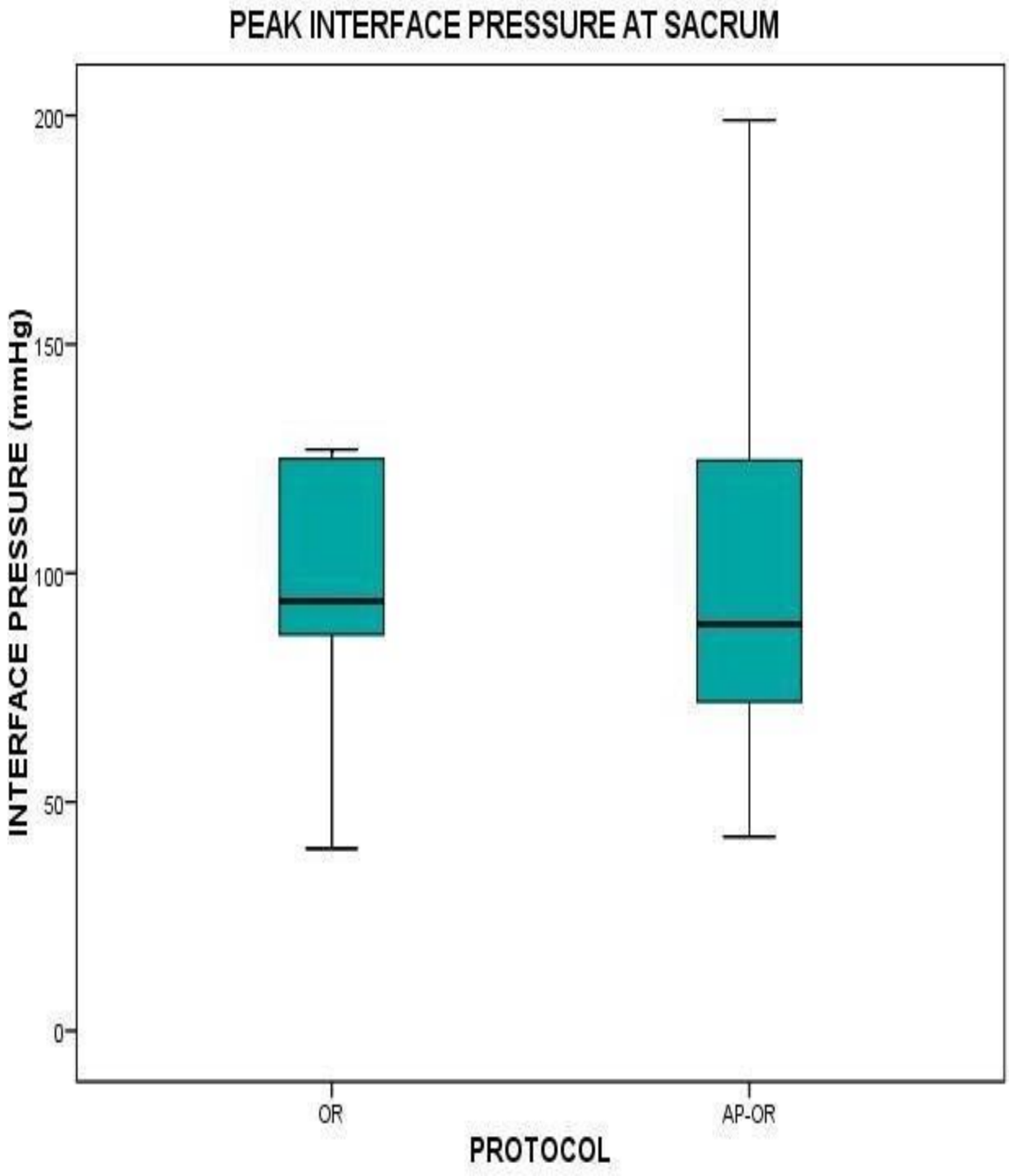

Figure 14:Peak interface pressure during OR and AP-OR.

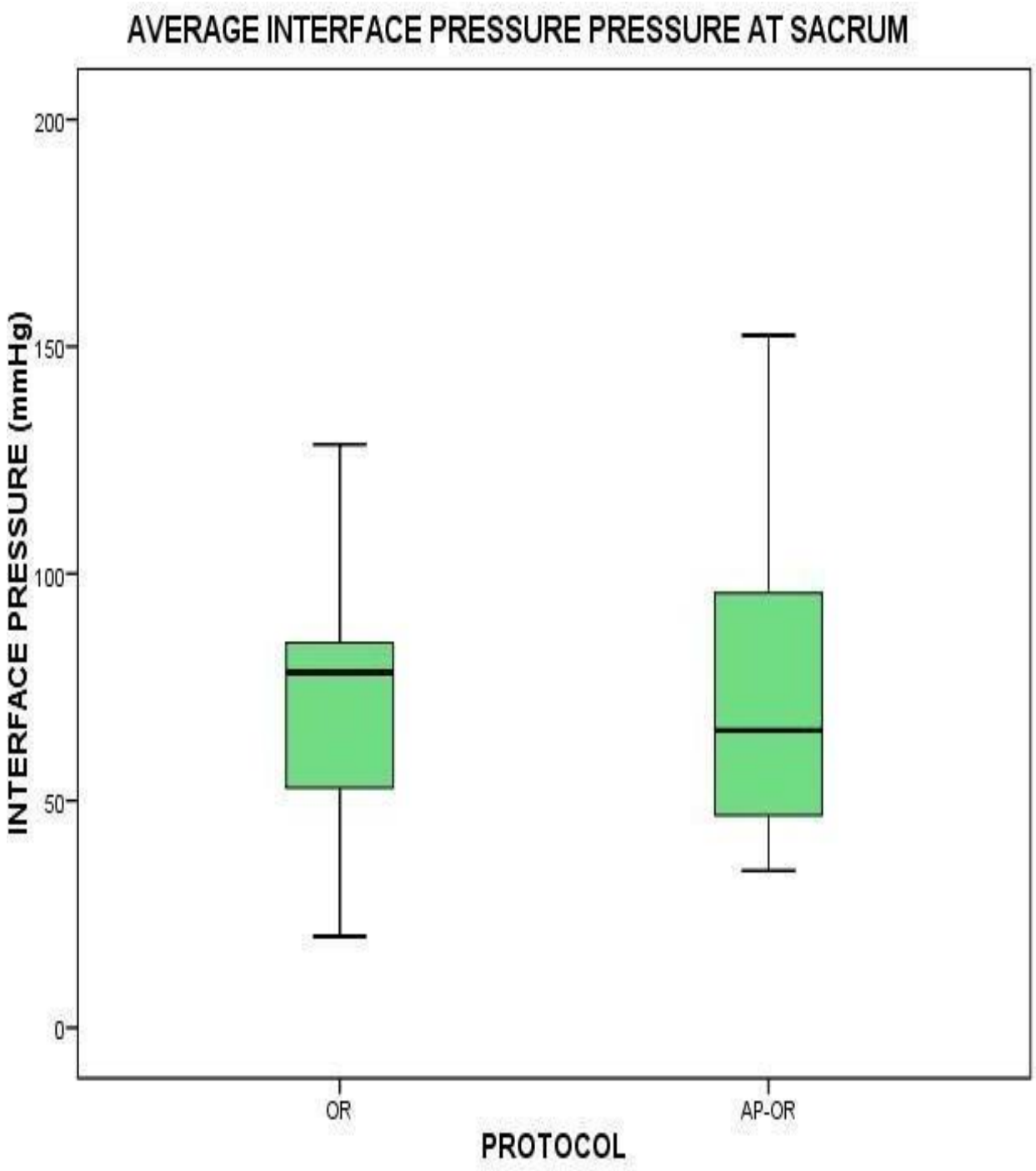

Figure 15: Average interface pressure during OR and AP-OR.

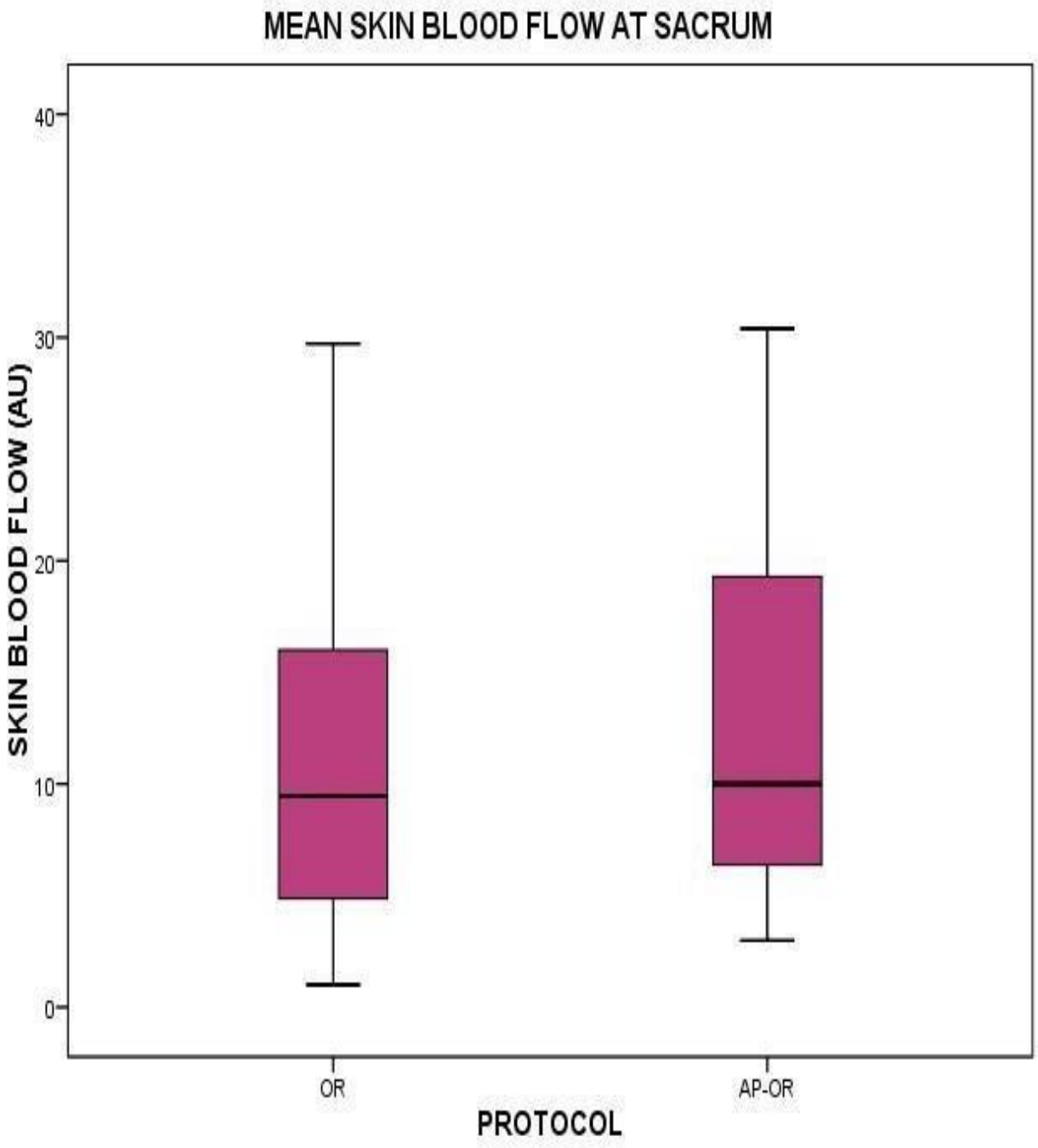

Figure 16: Mean skin blood flow during OR and AP-OR.

5.DISCUSSION

Majority of the previous studies on effects of AP mattresses or overlays used pressure injury risk or incidence as outcome measures. A literature review done by Kottner and colleagues (Kottner 2011) included RCTS comparing AP mattresses with constant-low pressure mattresses showed no clear proof of excellence of one type of mattress over the other. Anderson et al. (1982) showed that the incidence of pressure injuries decreased with the usage of AP mattresses as compared to regular OR mattress. Another study evaluated the effectiveness an AP mattress in critically ill ICU patients and found that the mattress successfully reduced the risk of developing pressure injuries by redistributing pressure and offloading the heels (Masterson and Younger 2014a). Despite the favorable results demonstrated in previous studies, when AP mattress was compared to other pressure redistribution mattresses or overlays (e.g. air mattress or water overlays), there was no clear evidence if AP mattresses outperform other special support surfaces (Vanderwee, Grypdonck, and Defloor 2008). A meta-analysis study also suggested that there is a deficiency of evidence of the effects of constant alternating pressure overlays on preventing pressure injuries (McInnes et al. 2012). In addition, the potential protective mechanisms of AP on pressure injury is not fully understood.

There were only a handful of studies that investigated the potential underlying mechanism of AP strategy. Previous studies suggested that AP mattresses or overlays prevent pressure injuries by reducing the interface pressure. Another study done (Hickerson et al. 2004) showed that AP mattresses are most effective in reducing interface pressure at hips, torso and feet as compared to with other mattresses on healthy adults. The other study done by Goetz et.al (2002) conducted on veterans with SCI compared the interface pressures between AP overlay and the dynamic floatation

system. They found that the interface pressure the interface pressure using AP overlay was lower than that using dynamic floatation system. Our findings that interface pressure reduced with deflation cycle of AP protocol was consistent with previous studies. In addition, even though we demonstrated that interface pressure increases during inflation cycle of AP, this value was not greater than that during OR. This suggested that the low-profile AP overlay used in this study could successfully redistribute pressure at the sacrum area on people with chronic SCI in an OR setting.

Other potential protective mechanisms of AP strategies were investigated previously. One study (Jakobsen and Christensen 1987) compared the transcutaneous oxygen tension ($tcPO_2$) at sacrum with healthy adults using AP overlay, hollow fiber mattress, standard hospital mattress, water mattress and AP mattress. They found that $tcPO_2$ was higher with AP overlay, mattress as compared to that with standard hospital mattress. Another study conducted (Rithalia, Heath, and Gonsalkorale 2000) also demonstrated that APAMs with multiple-air cell showed great $tcPCO_2$ as compared to double layered as well as single air-cell. Rithalia and colleagues also conducted a study on APAMs in a laboratory setting where Low APAM was compared to the Nimbus 3 mattress (Rithalia 2004). Results showed both decrease in IP with an increase in $tcPCO_2$ over both sacrum and heel as well as an increase in the laser Doppler perfusion recorded at the heel. Findings in previous study measuring transcutaneous oxygen tension was backed up by studies investigated SBF non-invasively. One study by Jan and colleagues (Jan et al. 2011) tested the effect of AP strategy versus constant low pressure on sacrum in healthy and SCI individuals. In this study, localized pressure was applied by a computer-controlled indenter at sacrum (indenter head diameter 36mm) and the results demonstrated that alternating pressure caused a significant increase in the SBF as compared to constant low pressure in both individuals with SCI and healthy

controls. The main difference between our study and that of Jan et al. was how alternating pressure was induced. We used an alternating pressure overlay which modulates the whole-body interface pressure to simulate a hospital or a clinical setting, whereas Jan et al. used the application of predetermined localized amount of alternating high (60 mmHg) and low pressure (0 mmHg) and compared SBF with that during constant low pressure (30 mmHg). In addition, the AP induced in Jan's study was at a single spot which its surrounding area is pressure free; whereas the AP induced in our study covered the whole body in contact with the AP overlay. Despite the difference in study design and the amount of interface pressure presented at the site of measurement, our results were consistent with that of Jan's, which SBF increases during the phase of deflation of AP protocol as compared to inflation and constant pressure in people with SCI. Our results also showed the SBF could increase during deflation even if it's not pressure free, this is encouraging for implementing AP in the clinical setting.

One unique finding from our study was the preconditioning effect of AP on weight bearing sacral tissue. Our findings suggested that 40 minutes of preconditioning reduced the peak interface pressure and increase the SBF during the subsequent weight-bearing condition at the sacrum. This is the first study that investigated the effect of preconditioning on weight bearing tissue protection. The reason for decrease in peak interface pressure during the AP-OR session is still unclear as both the OR pad used for control protocol and AP-OR was the same. The closest research study we could find using ischemia conditioning on skin was a recent research done (Shaked et al. 2015). They tested effectiveness of ischemic conditioning on diabetic foot ulcer healing. Ischemic conditioning in this study was provided in the form of a pressure cuff to the lower extremity repeatedly for three sessions every 2 weeks for a period of six weeks. Each session consisted of three 5-minute cycles of providing ischemia by inflating the cuff to 200 mmHg. They found that

the wound heals faster with ischemic conditioning as compared to the control group with standard treatment. Our study implemented similar protocol by providing three bouts of ischemic-like procedure (inflation cycle of AP protocol). Our findings complement previous studies that ischemic conditioning is beneficial to the skin by increasing perfusion, which is crucial to wound healing. Other explanation of the protective mechanism of ischemic conditioning include the neural hypothesis, the humoral and the systemic hypothesis mentioned in chapter. A research previously done (Loukogeorgakis et al. 2005) mentioned the importance of an intact autonomic system for the effect of the neural hypothesis of ischemic conditioning. Although an intact autonomic system is necessary for preconditioning the remove tissues, the study also suggested that in the absence of an intact autonomic system, the humoral system acts by releasing endogenous opioids for the improvement in microcirculation and endothelial function. Hence, though our study population showed disruption in the autonomic system, AP overlay was still successful in inducing preconditioning by increasing the tissue perfusion at sacrum in individuals with chronic SCI.

We did not find any significant injury factor in our study that contributes to the changes in interface pressure or skin blood flow. Our research study did not have a specific inclusion criterion for BMI of participants with SCI. Hence, range of BMI of the participants that were recruited for the study was 21.70 -31.40 (normal to obese). We found a weak negative correlation between BMI and change in SBF during AP-Inflation and AP-deflation, Deflation and OR at sacrum. This means that individuals with higher BMI were associated with smaller change in skin blood flow between AP-inflation- deflation and OR- AP-OR. These findings were consistent to the findings in previous studies where individuals with a lower BMI demonstrated greater improvements in pressure distribution and skin blood flow as compared to individuals with a higher BMI (Vanderwee, Grypdonck, and Defloor 2008). In another study done (Chai and Bader

2013) found that at the time of assessment, the maximum internal pressures at the sacrum were dependent on the BMI of the subjects. However, in this study, the alternating pressure mattress has a special feature called SAALP (Self-adjusting low-pressure sensor) which was used to adjust pressures according to the BMI of the able-bodied subjects. In such research studies, recommendations were made to change the alternating pressure settings according to the BMI of the individuals to avoid "bottoming out" phenomenon (Hampton 2016). The weak correlation found in our study indicates the necessity of adjusting the settings of the alternating pressure overlay to adjust optimal effect of AP overlay on individuals with higher BMI.

A. Clinical applications

The results of our experimental study could be extrapolated into not only clinical and home health-care settings but also in surgical settings. Alternating pressure overlays can be utilized in long-term care or home care settings in addition to repositioning to prevent the incidence of pressure injuries in high-risk populations like spinal cord injuries. Alternating pressure overlays could also be utilized during prolonged surgeries during which the incidence of developing a pressure injury is high due to immobilization for several hours. In such situations, AP overlays can be useful in redistributing pressure from pressure injury prone bony prominences like the sacrum. Although the cost of AP overlays is higher than the cost of regular OR pad or standard hospital mattress, by reducing the risk of pressure injuries; AP overlays may help save medical costs and human resources that are required to manage of pressure injuries.

B. Limitations

There are several limitations to this study. This study included 15 individuals with chronic spinal cord injury with very strict inclusion criteria, therefore the findings could not be generalized to the spinal cord injury population that has other comorbidities or during the acute phase of spinal cord injury. Our study also included individuals with both AIS A, B i.e individuals with complete and incomplete spinal cord injury. Further research is needed to study the influence of completeness of injury on the efficacy of alternating pressure overlay. An OR pad was utilized as control protocol in comparison with the alternating pressure overlay in individuals with chronic SCI. Therefore, our findings could not be generalized to home care or long-term care settings.

6. CONCLUSION

This study investigated the potential protective mechanism of AP overlay on weight bearing sacral tissue simulating an OR setting. We found that AP overlay is low profile yet effective in redistributing the interface pressure on bony prominence during supine lying, and subsequently increase skin blood flow during deflation cycle of AP. In addition, AP has a short-term lasting effect on interface pressure and blood flow, which could potentially be a strategy to precondition tissue before long term ischemia. Further studies in OR, and hospital setting is warranted to investigate the protective effect of AP overlay on other high-risk population of pressure injuries.

7. BIBLIOGRAPHY

Allman, Richard M. 1989. 'Pressure ulcers among the elderly', *New England Journal of Medicine*, 320: 850-53.

Amlung, Stephanie R, Wendy L Miller, and Linda M Bosley. 2001. 'The 1999 national pressure ulcer prevalence survey: a benchmarking approach', *Advances in skin & wound care*, 14: 297-301.

Andersen, K. E., O. Jensen, S. A. Kvorning, and E. Bach. 1983. 'Decubitus prophylaxis: a prospective trial on the efficiency of alternating-pressure air-mattresses and water-mattresses', *Acta Derm Venereol*, 63: 227-30.

Arias, S., E. Cardiel, L. Garay, H. Sanada, T. Mori, H. Noguchi, G. Nakagami, and P. Rogeli. 2015. 'Effects on interface pressure and tissue oxygenation under ischial tuberosities during the application of an alternating cushion', *J Tissue Viability*, 24: 91-101.

Bader, Dan L, Carlijn Bouten, Denis Colin, and Cees WJ Oomens. 2005. 'Pressure ulcer research: current and future perspectives'.

Bain, D. 2011. 'Laboratory performance of alternating pressure air mattresses component and sequelae', *Br J Nurs*, 20: S29-34.

Bansal, Cheryl, Ron Scott, David Stewart, and Clay J Cockerell. 2005. 'Decubitus ulcers: a review of the literature', *International journal of dermatology*, 44: 805-10.

Benstoem, C., C. Stoppe, O. J. Liakopoulos, J. Ney, D. Hasenclever, P. Meybohm, and A. Goetzenich. 2017. 'Remote ischaemic preconditioning for coronary artery bypass grafting (with or without valve surgery)', *Cochrane Database Syst Rev*, 5: Cd011719.

Benstoem, F., A. Nahrstedt, M. Boehler, G. Knopp, D. Montag, H. Siegrist, and J. Pinnekamp. 2017. 'Performance of granular activated carbon to remove micropollutants from municipal wastewater-A meta-analysis of pilot- and large-scale studies', *Chemosphere*, 185: 105-18.

Bergstrom, Nancy, Susan D Horn, Randall J Smout, Stacy A Bender, Maree L Ferguson, George Taler, Abby C Sauer, Siohban S Sharkey, and Anne Coble Voss. 2005. 'The National Pressure Ulcer Long-Term Care Study: outcomes of pressure ulcer treatments in long-term care', *Journal of the American Geriatrics Society*, 53: 1721-29.

Bethell, E. 1994. 'Pressure sores. Alternating pressure systems are useful', *Bmj*, 309: 1436.

Black, Joyce, Mona Mylene Baharestani, Janet Cuddigan, Becky Dorner, Laura Edsberg, Diane Langemo, Mary Ellen Posthauer, Catherine Ratliff, and George Taler. 2007. 'National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system', *Advances in skin & wound care*, 20: 269-74.

Blenman, J., and D. Marks-Maran. 2017. 'Pressure ulcer prevention is everyone's business: the PUPS project', *Br J Nurs*, 26: S16-s26.

Bliss, M. R. 1995. 'Preventing pressure sores in elderly patients: a comparison of seven mattress overlays', *Age Ageing*, 24: 297-302.

Bliss, M. R., and J. M. Thomas. 1993. 'An investigative approach. An overview of randomised controlled trials of alternating pressure supports', *Prof Nurse*, 8: 437-44.

Boghossian, J. A., B. Joseph, M. J. Slepian, and D. G. Armstrong. 2017. 'Remote Ischemic Conditioning(Promising Potential in Wound Repair in Diabetes?)', *J Am Podiatr Med Assoc*, 107: 313-17.

Bouten, C. V., M. M. Knight, D. A. Lee, and D. L. Bade. 2001. 'Compressive deformation and damage of muscle cell subpopulations in a model system', *Ann Biomed Eng*, 29: 153-63.

Breuls, RGM, CVC Bouten, CWJ Oomens, DL Bader, and FPT Baaijens. 2003. 'Compression induced cell damage in engineered muscle tissue: an in vitro model to study pressure ulcer aetiology', *Ann Biomed Eng*, 31: 1357-64.

Brienza, D. M., M. J. Geyer, and Y. K. Jan. 2005. 'A comparison of changes in rhythms of sacral skin blood flow in response to heating and indentation', *Arch Phys Med Rehabil*, 86: 1245-51.

Brown, R., S. Engel, B. G. Wallin, M. Elam, and V. Macefield. 2007a. 'Assessing the integrity of sympathetic pathways in spinal cord injury', *Auton Neurosci*, 134: 61-8.

Brown, Rachael, Stella Engel, B Gunnar Wallin, Mikael Elam, and Vaughan Macefield. 2007b. 'Assessing the integrity of sympathetic pathways in spinal cord injury', *Autonomic Neuroscience*, 134: 61-68.

Brurok, B, T Tørhaug, G Leivseth, T Karlsen, J Helgerud, and J Hoff. 2012. 'Effect of leg vascular occlusion on arm cycling peak oxygen uptake in spinal cord-injured individuals', *Spinal Cord*, 50: 298-302.

Byrne, DW, and CA Salzberg. 1996. 'Major risk factors for pressure ulcers in the spinal cord disabled: a literature review', *Spinal Cord*, 34: 255-63.

Caliri, Maria Helena Larcher. 2005. 'Spinal cord injury and pressure ulcers', *Nursing Clinics*, 40: 337-47.

Chai, C. Y., and D. L. Bader. 2013. 'The physiological response of skin tissues to alternating support pressures in able-bodied subjects', *J Mech Behav Biomed Mater*, 28: 427-35.

Chan, B. C., N. Nanwa, N. Mittmann, D. Bryant, P. C. Coyte, and P. E. Houghton. 2013. 'The average cost of pressure ulcer management in a community dwelling spinal cord injury population', *Int Wound J*, 10: 431-40.

Chen, Yuying, Michael J DeVivo, and Amie B Jackson. 2005. 'Pressure ulcer prevalence in people with spinal cord injury: age-period-duration effects', *Arch Phys Med Rehabil*, 86: 1208-13.

Colin, D., J. M. Rochet, P. Ribinik, B. Barrois, Y. Passadori, and J. M. Michel. 2012. 'What is the best support surface in prevention and treatment, as of 2012, for a patient at risk and/or suffering from pressure ulcer sore? Developing French guidelines for clinical practice', *Ann Phys Rehabil Med*, 55: 466-81.

Conine, T. A., D. Daechsel, and M. S. Lau. 1990. 'The role of alternating air and Silicore overlays in preventing decubitus ulcers', *Int J Rehabil Res*, 13: 57-65.

Cox, Jill. 2017. 'Pressure Injury Risk Factors in Adult Critical Care Patients: A Review of the Literature', *Ostomy/wound management*, 63: 30-43.

Cragg, J., and A. Krassioukov. 2012. 'Autonomic dysreflexia', *Cmaj*, 184: 66.

Defloor, T. 2000. 'The effect of position and mattress on interface pressure', *Appl Nurs Res*, 13: 2-11.

Defloor, T., and J. D. De Schuijmer. 2000. 'Preventing pressure ulcers: an evaluation of four operating-table mattresses', *Appl Nurs Res*, 13: 134-41.

Délot, E. C., M. E. Bahamonde, M. Zhao, and K. M. Lyons. 2003. 'BMP signaling is required for septation of the outflow tract of the mammalian heart', *Development*, 130: 209-20.

Edsberg, L. E., J. M. Black, M. Goldberg, L. McNichol, L. Moore, and M. Sieggreen. 2016a. 'Revised National Pressure Ulcer Advisory Panel Pressure Injury Staging System: Revised Pressure Injury Staging System', *J Wound Ostomy Continence Nurs*, 43: 585-97.

Edsberg, Laura E, Joyce M Black, Margaret Goldberg, Laurie McNichol, Lynn Moore, and Mary Sieggreen. 2016b. 'Revised national pressure ulcer advisory panel pressure injury staging system: revised pressure injury

staging system', *Journal of Wound, Ostomy, and Continence Nursing*, 43: 585.

Epps, J. A., and N. A. Smart. 2016. 'Remote ischaemic conditioning in the context of type 2 diabetes and neuropathy: the case for repeat application as a novel therapy for lower extremity ulceration', *Cardiovasc Diabetol*, 15: 130.

Epps, J., G. Dieberg, and N. A. Smart. 2016. 'Repeat remote ischaemic pre-conditioning for improved cardiovascular function in humans: A systematic review', *Int J Cardiol Heart Vasc*, 11: 55-58.

González-Méndez, M. I., M. Lima-Serrano, C. Martín-Castaño, I. Alonso-Araujo, and J. S. Lima-Rodríguez. 2018. 'Incidence and risk factors associated with the development of pressure ulcers in an intensive care unit', *J Clin Nurs*, 27: 1028-37.

Goossens, RHM, R Zegers, GA Hoek van Dijke, and CJ Snijders. 1994. 'Influence of shear on skin oxygen tension', *Clinical physiology*, 14: 111-18.

Groah, S. L., M. Schladen, C. G. Pineda, and C. H. Hsieh. 2015. 'Prevention of Pressure Ulcers Among People With Spinal Cord Injury: A Systematic Review', *Pm r*, 7: 613-36.

Hampton, S. 2016. 'The use of alternating mattresses in the management and prevention of pressure ulcers in a community setting', *Br J Community Nurs*, 21 Suppl 9: S19-25.

Harris, R. P., M. Helfand, S. H. Woolf, K. N. Lohr, C. D. Mulrow, S. M. Teutsch, and D. Atkins. 2020. 'REPRINT OF: Current Methods of the U.S. Preventive Services Task Force: A Review of the Process', *Am J Prev Med*, 58: 316-31.

Hausenloy, D. J., and D. M. Yellon. 2008. 'Remote ischaemic preconditioning: underlying mechanisms and clinical application', *Cardiovasc Res*, 79: 377-86.

Herrman, E. C., C. F. Knapp, J. C. Donofrio, and R. Salcido. 1999a. 'Skin perfusion responses to surface pressure-induced ischemia: implication for the developing pressure ulcer', *J Rehabil Res Dev*, 36: 109-20.

Herrman, Eric C, Charles F Knapp, James C Donofrio, and Richard Salcido. 1999b. 'Skin perfusion responses to surface pressure-induced ischemia: implication for the developing pressure ulcer', *J. Rehabil. Res. Dev*, 36.

Hickerson, W. L., G. M. Slugocki, R. L. Thaker, R. Dunkan, J. F. Bishop, and J. K. Parks. 2004. 'Comparison of total body tissue interface pressure of specialized pressure-relieving mattresses', *J Long Term Eff Med*

Implants, 14: 81-94.

Higer, S., and T. James. 2016. 'Interface pressure mapping pilot study to select surfaces that effectively redistribute pediatric occipital pressure', *J Tissue Viability*, 25: 41-9.

Horn, Susan D, Stacy A Bender, Nancy Bergstrom, Abby S Cook, Maree L Ferguson, Holly L Rimmasch, Siobhan S Sharkey, Randall J Smout, George A Taler, and Anne C Voss. 2002. 'Description of the national pressure ulcer long-term care study', *Journal of the American Geriatrics Society*, 50: 1816-25.

Horn, Susan D, Stacy A Bender, Maree L Ferguson, Randall J Smout, Nancy Bergstrom, George Taler, Abby S Cook, Siobhan S Sharkey, and Anne Coble Voss. 2004. 'The National Pressure Ulcer Long-Term Care Study: pressure ulcer development in long-term care residents', *Journal of the American Geriatrics Society*, 52: 359-67.

Jakobsen, J., and K. S. Christensen. 1987. 'Transcutaneous oxygen tension measurement over the sacrum on various anti-decubitus mattresses', *Dan Med Bull*, 34: 330-1.

Jan, Y. K., D. M. Brienza, M. L. Boninger, and G. Brenes. 2011. 'Comparison of skin perfusion response with alternating and constant pressures in people with spinal cord injury', *Spinal Cord*, 49: 136-41.

Jan, Yih-Kuen, and David Brienza. 2006. 'Technology for pressure ulcer prevention', *Topics in spinal cord injury rehabilitation*, 11: 30-41.

Jaul, Efraim. 2010. 'Assessment and management of pressure ulcers in the elderly', *Drugs & aging*, 27: 311-25.

Joseph, J., D. McLaughlin, V. Darian, L. Hayes, and A. Siddiqui. 2019. 'Alternating Pressure Overlay for Prevention of Intraoperative Pressure Injury', *J Wound Ostomy Continence Nurs*, 46: 13-17.

Kosiak, Michael. 1961. 'Etiology of decubitus ulcers', *Arch phy Med Rehab*, 42: 19-29.

Kottner, J. 2011. 'Preventing additional ischemic injury?', *J Wound Ostomy Continence Nurs*, 38: 221.

Krishnan, S., P. E. Karg, M. L. Boninger, and D. M. Brienza. 2017. 'Association between presence of pneumonia and pressure ulcer formation following traumatic spinal cord injury', *J Spinal Cord Med*, 40: 415-22.

Kruger, E. A., M. Pires, Y. Ngann, M. Sterling, and S. Rubayi. 2013. 'Comprehensive management of pressure ulcers in spinal cord injury: current concepts and future trends', *J Spinal Cord Med*, 36: 572-85.

Li, C, ND DiPiro, Y Cao, Y Szlachcic, and J Krause. 2016. 'The association between metabolic syndrome and

pressure ulcers among individuals living with spinal cord injury', *Spinal Cord*, 54: 967-72.

Liu, Hong, Dimitri Leray, Stéphane Colin, P. Pons, and Adrien Broué. 2012. *Finite Element Based Surface Roughness Study for Ohmic Contact of Microswitches*.

Loukogeorgakis, S. P., A. T. Panagiotidou, M. W. Broadhead, A. Donald, J. E. Deanfield, and R. J. MacAllister. 2005. 'Remote ischemic preconditioning provides early and late protection against endothelial ischemia-reperfusion injury in humans: role of the autonomic nervous system', *J Am Coll Cardiol*, 46: 450-6.

Manzano, F., M. Colmenero, A. M. Pérez-Pérez, D. Roldán, M. Jiménez-Quintana Mdel, M. R. Mañas, M. A. Sánchez-Moya, C. Guerrero, MÁ Moral-Marfil, E. Sánchez-Cantalejo, and E. Fernández-Mondéjar. 2014. 'Comparison of two repositioning schedules for the prevention of pressure ulcers in patients on mechanical ventilation with alternating pressure air mattresses', *Intensive Care Med*, 40: 1679-87.

Manzano, Francisco, Manuel Colmenero, Ana María Pérez-Pérez, Delphine Roldán, María del Mar Jiménez-Quintana, María Reyes Mañas, María Angustias Sánchez-Moya, Carmen Guerrero, María Ángeles Moral-Marfil, and Emilio Sánchez-Cantalejo. 2014. 'Comparison of two repositioning schedules for the prevention of pressure ulcers in patients on mechanical ventilation with alternating pressure air mattresses', *Intensive Care Med*, 40: 1679-87.

Marin, J, J Nixon, and C Gorecki. 2013. 'A systematic review of risk factors for the development and recurrence of pressure ulcers in people with spinal cord injuries', *Spinal Cord*, 51: 522-27.

Masterson, S., and C. Younger. 2014a. 'Using an alternating pressure mattress to offload heels in ICU', *Br J Nurs*, 23: S44, s46-9.

Masterson, Sarah, and Caroline Younger. 2014b. 'Using an alternating pressure mattress to offload heels in ICU', *British Journal of Nursing*, 23: S44-S49.

McInnes, E., A. Jammali-Blasi, S. Bell-Syer, J. Dumville, and N. Cullum. 2012. 'Preventing pressure ulcers--Are pressure-redistributing support surfaces effective? A Cochrane systematic review and meta-analysis', *Int J Nurs Stud*, 49: 345-59.

Nayak, P. K., and D. S. Kerr. 2013. 'Low-dose GYKI-52466: prophylactic preconditioning confers long-term neuroprotection and functional recovery following hypoxic-ischaemic brain injury', *Neuroscience*, 232:

128-38.

Nixon, J., G. Cranny, and S. Bond. 2005. 'Pathology, diagnosis, and classification of pressure ulcers: comparing clinical and imaging techniques', *Wound Repair Regen*, 13: 365-72.

Peirce, Shayn M, Thomas C Skalak, and George T Rodeheaver. 2000. 'Ischemia-reperfusion injury in chronic pressure ulcer formation: a skin model in the rat', *Wound repair and regeneration*, 8: 68-76.

Pickard, J. M., H. E. Bøtker, G. Crimi, B. Davidson, S. M. Davidson, D. Dutka, P. Ferdinandy, R. Ganske, D. Garcia-Dorado, Z. Giricz, A. V. Gourine, G. Heusch, R. Kharbanda, P. Kleinbongard, R. MacAllister, C. McIntyre, P. Meybohm, F. Prunier, A. Redington, N. J. Robertson, M. S. Suleiman, A. Vanezis, S. Walsh, D. M. Yellon, and D. J. Hausenloy. 2015. 'Remote ischemic conditioning: from experimental observation to clinical application: report from the 8th Biennial Hatter Cardiovascular Institute Workshop', *Basic Res Cardiol*, 110: 453.

Przyklenk, K., B. Bauer, M. Ovize, R. A. Kloner, and P. Whittaker. 1993. 'Regional ischemic 'preconditioning' protects remote virgin myocardium from subsequent sustained coronary occlusion', *Circulation*, 87: 893-9.

Reddy, M., S. S. Gill, and P. A. Rochon. 2006. 'Preventing pressure ulcers: a systematic review', *Jama*, 296: 974-84.

Redelings, M. D., N. E. Lee, and F. Sorvillo. 2005. 'Pressure ulcers: more lethal than we thought?', *Adv Skin Wound Care*, 18: 367-72.

Reswick, JB, and JE Rogers. 1976. 'Experience at Rancho Los Amigos Hospital with devices and techniques to prevent pressure sores.' in, *Bed sore biomechanics* (Springer).

Rithalia, S. V. 2004. 'Evaluation of alternating pressure air mattresses: one laboratory-based strategy', *J Tissue Viability*, 14: 51-8.

Rithalia, S. V., and M. Gonsalkorale. 1998. 'Assessment of alternating air mattresses using a time-based interface pressure threshold technique', *J Rehabil Res Dev*, 35: 225-30.

———. 2000. 'Quantification of pressure relief using interface pressure and tissue perfusion in alternating pressure air mattresses', *Arch Phys Med Rehabil*, 81: 1364-9.

Rithalia, S. V., G. H. Heath, and M. Gonsalkorale. 2000. 'Assessment of alternating-pressure air mattresses using a time-based pressure threshold technique and continuous measurements of transcutaneous gases', *J Tissue*

Viability, 10: 13-20.

Schubert, V., and B. Fagrell. 1991. 'Postocclusive reactive hyperemia and thermal response in the skin microcirculation of subjects with spinal cord injury', *Scand J Rehabil Med*, 23: 33-40.

Shaked, G., D. Czeiger, A. Abu Arar, T. Katz, I. Harman-Boehm, and G. Sebbag. 2015. 'Intermittent cycles of remote ischemic preconditioning augment diabetic foot ulcer healing', *Wound Repair Regen*, 23: 191-6.

Shi, C., J. C. Dumville, and N. Cullum. 2018. 'Support surfaces for pressure ulcer prevention: A network meta-analysis', *PLoS One*, 13: e0192707.

Stannard, D. 2012. 'Support surfaces for pressure ulcer prevention', *J Perianesth Nurs*, 27: 341-2.

Stinson, M. D., S. A. Crawford, and A. P. Porter-Armstrong. 2008a. 'Interface pressure measurements: visual interpretation of pressure maps with MS clients', *Disabil Rehabil*, 30: 618-24.

Stinson, M. D., A. Porter-Armstrong, and P. Eakin. 2003. 'Seat-interface pressure: a pilot study of the relationship to gender, body mass index, and seating position', *Arch Phys Med Rehabil*, 84: 405-9.

Stinson, MD, SA Crawford, and AP Porter-Armstrong. 2008b. 'Interface pressure measurements: Visual interpretation of pressure maps with MS clients', *Disability and Rehabilitation*, 30: 618-24.

Sunn, Gabriel. 2014. 'Spinal cord injury pressure ulcer treatment: an experience-based approach', *Physical Medicine and Rehabilitation Clinics*, 25: 671-80.

Tagawa, T., Tamura, T.,& Oberg, P.A. 2011. ' Biomedical Sensors and Instruments ', *CRC Press.* *https://doi.org/10.1201/b10775*.

Taradaj, Jakub. 2017. 'Prevention and treatment of pressure ulcers by newest recommendations from European Pressure Ulcer Advisory Panel (EPUAP): practical reference guide for GPs', *Family Medicine and Primary Care Review*, 19: 81-83.

Tran, Jacquelynn P, Jillian M McLaughlin, Ramon T Li, and Linda G Phillips. 2016. 'Prevention of pressure ulcers in the acute care setting: new innovations and technologies', *Plastic and Reconstructive Surgery*, 138: 232S-40S.

Vanderwee, K., M. Grypdonck, and T. Defloor. 2008. 'Alternating pressure air mattresses as prevention for pressure ulcers: a literature review', *Int J Nurs Stud*, 45: 784-801.

Walton-Geer, Patina S. 2009. 'Prevention of pressure ulcers in the surgical patient', *AORN journal*, 89: 538-52.

'WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): An Executive Summary'. 2017. *J Wound Ostomy Continence Nurs*, 44: 241-46.

Xiong, Can, Xinglian Gao, Qiong Ma, Ying Yang, Zengyan Wang, Wenjing Yu, and Lei Yu. 2019. 'Risk factors for intraoperative pressure injuries in patients undergoing digestive surgery: A retrospective study', *J Clin Nurs*, 28: 1148-55.

Yarkony, Gary M. 1994. 'Pressure ulcers: a review', *Arch Phys Med Rehabil*, 75: 908-17.

VITA

Name	Rudri Purohit
Education	Bachelor of Physiotherapy, Sancheti Institute College of Physiotherapy; Pune. India. (2012-2015) MS in Rehabilitation Sciences, University of Illinois at Chicago. USA. (2017-present)
Internship	Sancheti Orthopedic and Rehabilitation Hospital.Pune. India. Aug2015-March 2016
Clinical/Practical Experience	Consultant Physical Therapist (Home Health Care) Nightingales- The Home Health Specialist, Mumbai, India. (Sep 2016 - June 2017) Physical therapist, SPARC Clinic (sports and aquatic rehabilitation clinic), HOD Dr.Ketan Bhatikar (M.I.A.P, Secretary, MPT Sports and orthopedic rehabilitation, Margao, Goa, India. (March 2016-October 2016) Physical therapy assistant, LAST clinic, under Dr.Gajanan Bhalerao (MPT Neurosciences).Pune.Maharashtra. (August 2015-Feb 2016)
Awards/Recognition	Recipient of 2019 AHS Achievement Award Recipient of 2018 Van Doran Scolarship, for outstanding achievement. Recipient of WOCN poster presenter award, 2019, Recipient of BOT scholarship for spring 2018, UIC. Recipient of Academic Excellence in Bachelors in Physiotherapy 2015-2016 by the police commissioner of India. Recipient of outstanding contribution to research and academics at Sancheti Institute-2015-2016
Professional Membership	Licensed Physical Therapist in the state of New York from 23rd August 2019. Licensed PT in the state of Indiana, 23rd August 2019. Licensed PT in India (2016).

	Member of Indian Association of Physio-Therapist (2016).
Extra-curricular	Conferences: • Scientifica , (Research conference for physiotherapists) Feb-2015 • CME on Matrix Rhythmus therapy. Oct-2014 Workshops: • Sports Medicine and Emergency Services with 29th Pune International Marathon Dec-2014 • Motor Relearning Program for stroke rehabilitation; a practical approach (8 credit hours). Sep-2014 • Exercises with the gym ball, Sancheti healthcare academy. Sep-2014 Supplementary Activities: • Kinesiology taping level, April 2016 • Proprioceptive Neuromuscular Facilitation (P.N.F Techniques), May 2015. • Motor Relearning Program (stroke rehabilitation), May 2015. • 1 st Knee Rehab Course, March 2015. • I.C.U training, August 2015. • Exercises with the gym ball, November 2014. • CME Programme -Matrix Rhythm Therapy, July 2013. • BLS and ACLS, June 2011 (basic and advanced life support) • Application of Yoga and Meditation in neuromuscular as well as geriartric and obstetric patients,2011-2012