

From the Department of Molecular Medicine and Surgery  
Karolinska Institutet, Stockholm Sweden

# PRESSURE ULCERS - ROLE OF THE NURSE TO IMPROVE PATIENT SAFETY

– prevalence, risk factors, classification and  
documentation in patients undergoing hip surgery

Eila Sterner



**Karolinska  
Institutet**

2012

All previously published papers were reproduced with permission from the publisher.

Published by Karolinska Institutet.

Illustrations: Kamilla Andersson. With kind permission to use from the illustrator.  
[www.kamillaandersson.se](http://www.kamillaandersson.se)

Layout Ringvor Hägglöf

© Eila Sterner, 2012

ISBN 978-91-7457-690-0

Printed by



[www.reproprint.se](http://www.reproprint.se)

Gårdsvägen 4, 169 70 Solna

*It is no pleasure to have pressure.*



# CONTENTS

---

|  |    |
|--|----|
| ABSTRACT.....  | 7  |
| LIST OF PUBLICATIONS.....                                  | 9  |
| LIST OF ABBREVIATION.....                                  | 11 |
| INTRODUCTION.....  | 13 |
| Pressure ulcers.....                                       | 13 |
| BACKGROUND.....  | 15 |
| Definition of pressure ulcers.....                         | 15 |
| Differentiation of skin erythema.....                      | 17 |
| Blanching.....   | 17 |
| Reactive hyperaemia.....                                   | 17 |
| Non-blanching.....   | 17 |
| Erythema.....  | 17 |
| Incidence and prevalence of pressure ulcers.....           | 17 |
| Risk factors.....  | 19 |
| Extrinsic risk factors.....                                | 19 |
| <i>Pressure</i> .....                                      | 19 |
| <i>Shear</i> .....   | 21 |
| <i>Friction</i> .....                                      | 21 |
| <i>Microclimate</i> .....                                  | 22 |
| Intrinsic risk factors.....                                | 22 |
| <i>Architecture of the skin</i> .....                      | 22 |
| <i>Vasculatory and circulation diseases</i> .....          | 24 |
| <i>Tissue perfusion</i> .....                              | 24 |
| <i>Cognitive impairment</i> .....                          | 25 |
| Prevention.....  | 25 |
| <i>Pressure relief</i> .....                               | 25 |
| <i>Support surface</i> .....                               | 26 |
| <i>Nutrition</i> .....                                     | 26 |
| Morbidity, mortality and suffering.....                    | 27 |
| Economic impact.....                                       | 27 |
| Risk assessment subjective /objective risk assessment..... | 28 |
| <i>Risk assessment scale</i> .....                         | 28 |
| <i>Perception of colour</i> .....                          | 29 |
| <i>Detection of pressure ulcer</i> .....                   | 29 |
| <i>Finger press test</i> .....                             | 30 |
| <i>Non invasive measure of skin erythema</i> .....         | 30 |
| <i>Reflectance spectrophotometer</i> .....                 | 31 |
| <i>Pilot examples from functional tests of DS</i> .....    | 32 |
| Femoral neck fractures.....                                | 32 |
| <i>Epidemiology</i> .....                                  | 32 |
| <i>Pathophysiology</i> .....                               | 32 |

|  |    |
|--|----|
| Pressure Ulcers and femoral neck fractures.....              | 33 |
| <i>Epidemiology</i> .....                                    | 33 |
| Nursing assessment.....                                      | 34 |
| <i>Good nursing care</i> .....                               | 35 |
| <i>The Nursing process</i> .....                             | 35 |
| <i>Nursing documentation</i> .....                           | 36 |
| Patient safety .....   | 37 |
| Quality indicator.....                                       | 39 |
| Government initiative.....                                   | 40 |
| AIMS OF STUDIES.....   | 41 |
| MATERIALS and METHODS.....                                   | 43 |
| Design.....  | 43 |
| Setting.....   | 43 |
| Data collection.....   | 44 |
| Study I.....   | 46 |
| Study II.....  | 47 |
| Study III.....   | 48 |
| Study IV.....  | 49 |
| Ethical consideration.....                                   | 50 |
| SUMMARY OF RESULTS .....                                     | 51 |
| Study I.....   | 51 |
| Study II.....  | 51 |
| Study III.....   | 51 |
| Study IV.....  | 52 |
| DISCUSSION.....  | 53 |
| CONCLUSIONS AND CLINICAL IMPLICATIONS.....                   | 59 |
| Patient benefit and generalisability.....                    | 59 |
| FURTHER RESEARCH.....  | 61 |
| Summary in Swedish - POPULÄRVETENSKAPLIG SAMMANFATTNING..... | 63 |
| ACKNOWLEDGMENTS.....   | 69 |
| REFERENCES.....  | 73 |
| APPENDIX .....   | 89 |
| PAPER I-IV   |    |

# ABSTRACT

---

Pressure ulcer is a common complication in hip fracture surgery and convalescence. Earlier prevalence studies have demonstrated a lower prevalence in Southern Europe than in Northern Europe. In patients with hip fractures, specific risk factors for developing pressure ulcers, apart from those included in standardised risk assessment are not fully understood. Correct classification of Category I pressure ulcers is a prerequisite for planning preventive measures. It is also mandatory for the reliability of prevalence studies. Until now subjective tests such as finger-press test and visual assessment have been utilised in clinical practice. An objective method has hitherto been lacking. Planning and delivering good nursing care to patients who are at risk of, or already have, manifest pressure ulcers should be built on proper documentation. Degree of documentation of pressure ulcer prevalence and risk factors in patients with hip fractures versus elective hip replacement surgery has hitherto not been investigated. Scrutiny of medical records for these diagnoses and identification of potential differences should therefore be of interest.

*Aim:*

- To investigate prevalence and incidence of pressure ulcers upon arrival and at discharge from hospital, and to identify potential intrinsic and extrinsic risk factors for the development of pressure ulcers in patients admitted for hip fracture surgery.

To establish the inter-rater reliability between blanching and non-blanching erythema, assessed by two independent assessors. The secondary purpose was to investigate potential correlations between risk factors and pressure ulcers.

- To explore if a non-invasive objective method could differentiate between blanching/non-blanching erythema in the sacral area of patients undergoing hip fracture surgery.

- To investigate the degree of documentation regarding risk assessment, preventive measures taken, prevalence and severity of pressure ulcers, in patients undergoing surgery for hip fractures or elective hip replacements at admission and during hospital care at an orthopaedic unit.

*Results:* The prevalence of pressure ulcers in Southern Europe was lower compared to Northern Europe. Specific risk factors such as dehydration ( $p=.005$ ), moist skin ( $p=.004$ ), pulmonary disease ( $p=.006$ ) and diabetes ( $p=.005$ ) were identified. The finger-press test and visual assessment of Category I pressure ulcers were both unreliable methods with low inter-rater reliability. The proportion of patients with persistent discoloration differed significantly between the assessors from Day 1 to Day 5 ( $p = .013$ ). Reflectance spectrophotometer used was proven to deliver high precision regarding classification of non-blanchable erythema (Category I pressure ulcers). Documentation of pressure ulcers, risk assessment, body mass index and prevention at admittance was unsatisfactory in patients undergoing hip surgery.

**Keywords:** Hip fracture, pressure ulcers, classification, reflectance spectrophotometer, nursing documentation



# LIST OF PUBLICATIONS

---

This thesis is based on the following papers, which will be referred to in the text by their Roman numerals.

- I **Hip fractures and pressure ulcers – the Pan European Pressure Ulcer Study – intrinsic and extrinsic risk factors.**  
Lindholm C, Sterner E, Romanelli M, Pina E, Torra y Bou J, Hietanen H, Livainen A, Gunningberg L, Hommel A, Klang, B, Dealey C  
*Int. Wound Journal*, 2008, vol 5, no 2, sid. 315 - 327
  
- II **Category I pressure ulcers – how reliable is clinical assessment?**  
Sterner E., Lindholm C, Berg E, Stark A, Fossum B  
*Orthop Nurs.* 2011 May/June;30(3):194-205
  
- III **Objective evaluation by reflectance spectrophotometry can be of clinical value for verification of sacral category I pressure ulcers**  
Sterner E, Fossum B, Lindholm C, Berg E, Stark A  
Submitted
  
- IV **Poor documentation of risk factors and prevention strategies for pressure ulcers in orthopedic inpatients.**  
Sterner E, Unbeck M, Lindholm C, Stark A, Gordon M, Fossum B  
In manuscript



# LIST OF ABBREVIATIONS

---

|         |  |
|---------|--|
| A&E     | Acute & Emergency Department                         |
| BMI     | Body Mass Index                                      |
| DS      | DermaSpectrometer                                    |
| E-Index | Erythema Index                                       |
| EPUAP   | European Pressure Ulcer Advisory Panel               |
| MNS     | Modified Norton Scale                                |
| NPUAP   | National Pressure Ulcer Advisory Panel               |
| PU      | Pressure Ulcers                                      |
| RSM     | Reflectance Spectrophotometry/Meter                  |
| SALAR   | Swedish Association of Local Authorities and Regions |
| SPMSQ   | The short portable mental status questionnaire       |
| THR     | Total Hip Replacement                                |



# INTRODUCTION

---

## Pressure ulcer

Pressure ulcers (PUs) have affected humans throughout history. In the past, they have been called “pressure sores,” “bedsores” and “decubitus ulcers” (from the Latin word *decumbere*, which means “to lie on one’s side”). The term “pressure ulcer” has been established by European Pressure Ulcer Advisory Panel (EPUAP) and is the term used in this thesis.<sup>1</sup> PUs are still a major problem in many healthcare settings for a number of reasons. Today, they constitute a global healthcare issue, as well as an economic burden.<sup>2-6</sup>

Preventing PUs has been a concern in the field of nursing for many years, beginning with Florence Nightingale and her assumption that if a bedsore develops, it is caused by nursing problems (Notes of nursing).<sup>7,8</sup> Some clinicians believe that PUs are caused by poor quality of care or limited knowledge and access to prevention.<sup>8-12</sup> This may give rise to feelings of guilt among nursing staff. The problem might be swept under the carpet, and staff might neglect the pressure ulcers but blame other caregivers.

Others believe that PUs are a direct result of a poor healthcare system.<sup>13</sup> Maintaining a high standard of patient safety requires a good organization and leadership, effective routines and a knowledgeable and interested staff.<sup>14</sup> A high level of skilled nurses is the most effective weapon, which at the same time is hard to achieve in times marked by big staff turnover and reduced number of nurses.<sup>15,16</sup> Difficulties in finding aids and equipment to prevent PUs are often reported.<sup>15</sup> A low prevalence and incidence of PUs is a quality indicator.<sup>17,18</sup> However, it is difficult to compare prevalence and incidence data without clear understanding of the definitions.<sup>19</sup> It is important to identify patients at risk and implement effective preventive strategies, with a goal of decreasing the incidence of PUs. PUs cause considerable harm to the patient, extend the length of hospital stay and increase mortality.<sup>2,20,21</sup> There should be zero tolerance of PU development<sup>22</sup> and if the prevention of PUs is a major nursing task it is also a multidisciplinary responsibility.<sup>23</sup>

Research in the area of PUs needs to focus on prevention as well as treatment. Inadequate intervention in category I PUs may lead to PUs deteriorating into a more severe category.<sup>24</sup> Since occurrence of PUs often is related to quality of care, it is important to investigate the effectiveness of prevention and to evaluate epidemiological studies to monitor changes over time.<sup>25,26,27-30</sup> It is also important to understand the mechanisms of pressure, shear stress, friction and microclimate in the development of PUs.<sup>31,32-34</sup>

Although most PUs are avoidable and steps are being taken to raise awareness of PUs in the areas of nursing care, medicine, surgery, and even if self-care education is improved, PUs remain a cause of morbidity and mortality.<sup>32,35,36</sup> This is mostly true for patients who have difficulties in changing positions due to pain, acute illness, loss of sensation or unconsciousness, or who are suffering from cognitive impairment.<sup>37-39</sup> Advanced age, malnutrition, incontinence and co-morbidities related to circulatory problems are reported to be related to the development of PUs.<sup>33,40-44</sup>



# BACKGROUND

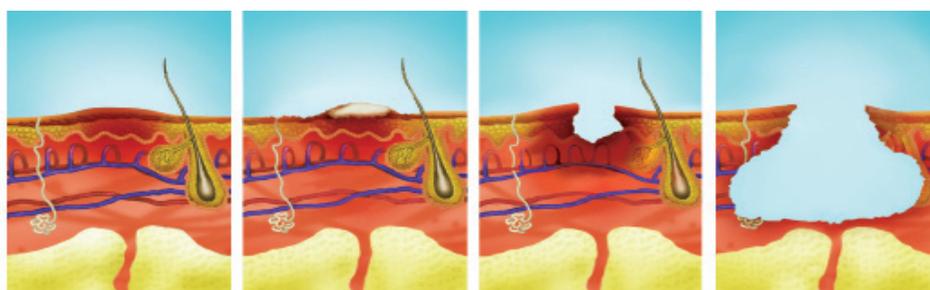
---

PU occur commonly over bony prominences although soft tissue can also be affected, resulting in ischemia, cell death, and tissue necrosis. Severity of illness and co-morbidity can influence the circulation in the skin, so that less pressure is needed for PUs to develop. A PU can develop in as little as 2-6 hours<sup>45</sup> have been reported in the medical literature, such as physiological factors, related to impairment of the microcirculatory system,<sup>46</sup> but other risk factors still remain unknown.<sup>47</sup> PUs have previously been categorised by stage (still used in United States) and grade. Since 2010 the severity of PUs has been classified as Category I-IV.

## Definition of pressure ulcers

“A pressure ulcer is a localised injury to the skin and/or underlying tissue usually over bony prominences, as a result of pressure, or pressure in combination with shear. A number of contributing or confounding factors are also associated with pressure ulcers; the significance of these factors is yet to be elucidated”.<sup>1, 48</sup> The severity of pressure ulcers can be classified in 4 different categories from non-blanching erythema (Category I), to full thickness tissue loss with exposed bone, tendon or muscle (Category IV). Two levels are used predominantly in the United States: unstageable and suspected deep tissue injury, where levels of the depth are unknown.<sup>25</sup> Certain clinical signs can indicate the development of a PU, such as tissue with different colour (blue/red), or more painful, firmer, softer, warmer or cooler than surrounding skin and tissue (Table 1). The true time span for development of PUs is yet unclear but it has been suggested that it can develop within 1-2 hours<sup>49</sup> or 2-6 hours.<sup>50, 45, 51</sup>

Illustration to Table 1



Category I

Category II

Category III

Category IV

**Table 1.** International EPUAP-NPUAP Pressure ulcer classification system

| Category   | Description  |
|--|--|
| <b>Category/Stage I:<br/>Non-blanchable erythema</b>   | Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area. The area may be painful, firm, soft, warmer or cooler compared to adjacent tissue. Category I/Stage I may be difficult to detect in individuals with dark skin tones. May indicate "at risk" persons (a heralding sign of risk).   |
| <b>Category/Stage II:<br/>Partial thickness</b>  | Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled or serosanguinous-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising.* This Category/Stage should not be used to describe skin tears, tape burns, incontinence associated dermatitis, maceration or excoriation. *Bruising indicates deep tissue injury.   |
| <b>Category/Stage III:<br/>Full thickness skin loss</b>  | Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscles are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling. The depth of a Category/Stage III pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have (adipose) subcutaneous tissue and Category/Stage III ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Category/Stage III pressure ulcers. Bone/tendon is not visible or directly palpable.          |
| <b>Category/Stage IV:<br/>Full thickness tissue loss</b>   | Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present. Often includes undermining and tunneling. The depth of a Category/Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have (adipose) subcutaneous tissue and these ulcers can be shallow. Category/Stage IV ulcers can extend into muscle and/or supporting structures (e.g., fascia, tendon or joint capsule) making osteomyelitis or osteitis likely to occur. Exposed bone/muscle is visible or directly palpable<br>(www.epuap.org, www.npuap.org) |
| Category   | Description  |
| <b>Additional Categories/<br/>Stages for the United States<br/>Unstageable/ Unclassified:<br/>Full thickness skin or tissue<br/>loss – depth unknown</b> | Full thickness tissue loss in which the actual depth of the ulcer is completely obscured by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed. Until enough slough and/or eschar are removed to expose the base of the wound, the true depth cannot be determined; but it will be either a Category/Stage III or IV. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as "the body's natural (biological) cover" and should not be removed.  |
| <b>Suspected Deep Tissue<br/>Injury – depth unknown</b>  | Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful or firm, or softer, warmer or cooler as compared to adjacent tissue. Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment (www.npuap.org 2010).           |

## Differentiation of skin erythema

### Blanching

Blanch means “to become white”.<sup>52</sup> Blanching hyperaemia is the distinct erythema caused by reactive hyperaemia. When pressure is applied e.g. finger-press test, the blood is evacuated and the skin blanches temporarily before the blood returns. This indicates that the patient’s microcirculation is intact.<sup>53</sup>

### Reactive hyperaemi

Reactive hyperaemia is the characteristic bright flush of the skin associated with an increased return of blood to an area after the release of an occlusion (pressure).<sup>54</sup> This is mainly noted in the epidermis and dermis as blanching hyperaemia.<sup>46,55</sup> If a reactive hyperaemia lasts for more than half an hour and is not resolved completely within 2 hours, it demonstrates microcirculatory disruption.<sup>45,46,49</sup> Reactive hyperaemia can occur after 30 minutes or less and generally this redness resolves within 1 hour.<sup>51</sup> The ischemia will develop after 2-6 hours<sup>45,50</sup> of continued pressure and after 6 hours the tissue may be necrotic.<sup>51</sup>

### Non-blanching hyperaemia

Non-blanching hyperaemia is when there are no changes in skin colour after the release of pressure. This indicates a degree of microcirculatory disruption. Other clinical signs may also be found, such as blistering, indurations and oedema. Non-blanching erythema (PUs Category I) should not be confused with blanching, reactive hyperaemia or erythema.<sup>46</sup>

### Erythema

Erythema can be defined as a non-specific redness of the skin. This can be as a result of an infection, cellulitis<sup>56</sup>, prolonged pressure or reactive hyperaemia.<sup>53</sup>

### Incidence and prevalence of pressure ulcers

PUs is still a problem in healthcare. All categories of patients can be affected and PU prevalence has been reported to range between 0.4%-66% depending on care settings and study design.<sup>57</sup> Prevalence and incidence in different studies are presented in Table 2. Certain reports indicate that the number of PUs is going to increase substantially, related to the increasing age.<sup>58</sup> More than 80% of all PUs occur in “classic” PU locations such as sacrum, ischial tuberosities, heels and ankles. Russo (2006) reported from 1993 to 2006 a 63% increase in PUs was found.<sup>59</sup> During the same period there was only an 11% increase in hospitalisations. Patients with PUs were 65 year or older in 73.3%, however nearly 28% were younger than 65 years.<sup>59</sup>

**Table 2** Prevalence or incidence of pressure ulcers as reported in different studies and care settings

|                                     |   |
|-------------------------------------|---|
| Hospital care/hip fracture patients | 3.8% <sup>60</sup> , 7% <sup>61</sup> , 8,8% <sup>62</sup> , 12.9% <sup>63</sup> *, 13.2% <sup>42</sup> , 18% <sup>64</sup> , 22.1% <sup>24</sup> , 23.9% <sup>42</sup> , 29% <sup>65</sup> , 30% <sup>66</sup> , 32% <sup>67</sup> , 36% <sup>68</sup> , 55% <sup>18</sup> , 66% <sup>69</sup> |
| Hospital care                       | 2,2% <sup>77</sup> , 3.9% <sup>71</sup> , 6.7% <sup>72</sup> *, 10,5% <sup>73</sup> *, 14.9% <sup>71</sup> , 20.0% <sup>74</sup> , 21.2% <sup>75</sup> , 33.3% <sup>76</sup> , 53.5% <sup>77</sup>  |
| Long term care/nursing home         | 4.1% <sup>70</sup> , 5.4% <sup>78</sup> , 8.7% <sup>24</sup> , 11.6% <sup>79</sup> , 11.7% <sup>79</sup> , 18% <sup>80</sup> ND, 18.7% <sup>40</sup> *, 20% <sup>42</sup> , 27% <sup>81</sup> ND , 29% <sup>82</sup>  |

\* Category I PUs not included. ND = no data

To measure PUs at admission and discharge gives information about development of pressure ulcers on a group level, but does not represent the true incidence unless the same patients are studied longitudinally.<sup>43</sup> Incidence of hospital acquired pressure ulcers (HAPUs) can only be studied if the same patients are followed during the care episode.<sup>83</sup>

People with spinal cord injury (SCI) are at great risk for develop PUs.<sup>84</sup> The incidence of PUs in this population has been estimated to between 30% and 46%<sup>85,86,87</sup>, and 7-10% of all deaths are reported to be attributed to PUs in this group of patients.<sup>88, 89</sup> Young children comprise another risk group for PU development. In one study, 27% of young children had PUs, including Category I PUs<sup>90</sup>. Prematurity, malnutrition, immobility (depending on age) and degree of unconsciousness are risk factors predisposing for pressure ulcer development in children.

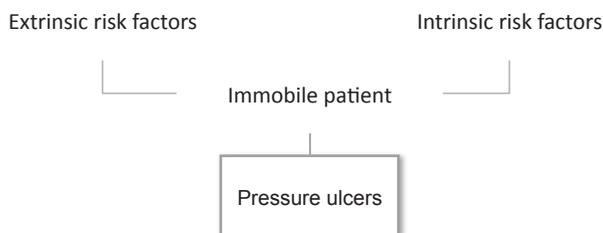
Research has shown that the risk of developing PUs at least doubles in operations lasting more than 4 hours.<sup>75, 91-93</sup> It is important to remember that the whole period the patient lies on an operating table can affect the tissue, including waiting for the surgery to start. If a patient has a PU prior to being admitted, there is an increased risk that it will deteriorate during the hospital stay.<sup>94</sup> A study from the Netherlands found that over 22% of pre-existing PUs in acute care and 8% in geriatric care deteriorate.<sup>24</sup>

Non-Caucasian patients tend to develop more severe PUs, which is attributable to difficulties in detecting areas affected by pressure, shear stress and friction. One early warning sign of a PU is reactive hyperaemia, which develops due to pressure.<sup>54</sup> The resulting change in colour is not as visible in darker skin, which can result in failure to take preventive measures before the skin has a change to rupture.<sup>95, 96</sup> More women than men suffer from rheumatoid arthritis in which the complications of medication can lead to extra skin sensitivity (thinning). This means that the bone is closer to the skin surface (because less padding) and there is an increased risk of developing a PU due to pressure. More women than men suffer from PUs due to advanced age, low weight and changes in the sensitivity of the skin to pressure and other influences. There is also a slight anatomical difference between the female and male sacrum. Os sacrum is more cupped in women which might lead to stretching of the tissue. The bony prominence per se also is less padded.

Certain under- or over-reporting of PUs can occur and this should be taken into consideration when comparing different settings of healthcare. The Swedish Association of Local Authorities and Regions (SALAR)<sup>97</sup> conducted two national point prevalence studies in Sweden (2011). PU rate declined from 17% to 14.4% in hospitals whereas the same prevalence (14%) was reported from the communities. More than 50% of the PUs were Category I. The two national point prevalence studies were performed in spring and autumn 2011 and will be repeated 2012. A total of 12,397 persons were investigated.<sup>97</sup> Achieving a goal of zero PUs during hospitalization.<sup>22</sup> requires expertise, and the opportunity to learn and use new evidence-based knowledge.<sup>98</sup> However, a zero-vision for PUs might be unrealistic in acute medical emergencies where life-saving actions might be prioritized. The development of PUs in the dying patient cannot always be avoided due to the physiological skin changes at life's end.<sup>99, 100</sup>

## Risk factors

Numerous risk factors for development of PUs have been reported.<sup>33, 69, 101, 102</sup> These can be classified as extrinsic or pathomechanical and intrinsic or pathophysiological risk factors (Figure 1). Tissue tolerance is one important intrinsic factor and stands for the skin and supporting structure ability to tolerate the effect of pressure without adverse effect.<sup>31</sup>



**Figure 1**

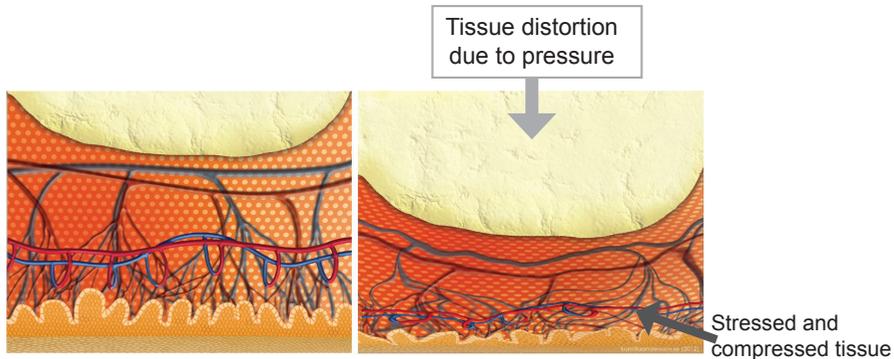
*Prolonged pressure due to a combination of immobility and extrinsic and intrinsic factors might lead to development of PUs.*

## Extrinsic risk factors

### *Pressure*

The most important factor in the development of PUs is unrelieved pressure. Pressure is defined as “the amount of force applied perpendicular to a surface per unit area of application”.<sup>31</sup> Healthy people and alert patients feel signals from the areas under pressure. These signals lead to movements even during sleep and re-establishing the tissue perfusion.<sup>103-106</sup> In an early study, Exton-Smith (1961) reported that if a patient moves fewer than 25 times during one night, the risk of developing PUs increases.<sup>104</sup>

Signals for position changes initiating spontaneous body movements may not function optimally in sick patients, leading to unrelieved pressure to vulnerable areas for example bony prominences as sacrum and heels. PUs arises from prolonged tissue ischemia caused by pressure that exceeds the tissue capillary pressure.<sup>55, 107-108</sup> (Figure 2) This pressure decrease oxygenation of the tissues and the supply of essential nutrients, and the skin appears pale.<sup>46, 109, 31</sup> When the pressure is relieved, healthy skin quickly becomes red due to the physiological response and appearance returns to normal.<sup>46, 53, 54</sup> In vulnerable skin, e.g. aged skin, and prolonged ischemia compression and blocking of the capillaries can occur. The blood cells aggregate and the capillary walls can become damaged. Accumulation of red blood cells and fluid leakage into the interstitial space can lead to non-blanchable erythema.<sup>46, 110</sup> This will aggravate the ischemia and cause necrosis of the tissue and ulceration. Reactive hyperemia should be distinctly separated from non blanchable erythema.<sup>42, 73, 111</sup>



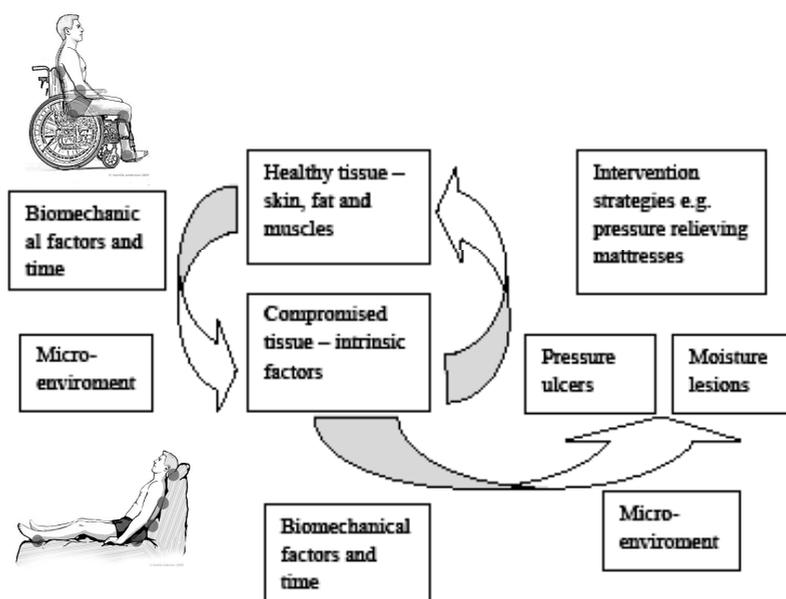
**Figure 2**

*When external pressure is applied over a bony prominence, the tissue is affected by compression and shear stress. This decreases oxygenation and nutrient supply to the tissue.*

Pressure and shear act synergistically in development of PUs. Pressure distorts the skin and underlying soft tissue especially near bony prominences.<sup>31, 109, 112</sup> Pressure on any part of the body, especially over a bony prominence, may cause reactive hyperaemia for up to 48 hours and still have normal dermal response.<sup>45, 113-115</sup> It can take several days before it is possible to detect if the skin begins to break down or recover to normal tissue.<sup>46, 116</sup>

There is a relationship between intensity of pressure and duration however; this can vary between different individuals as well as within the same individual. Normal blood pressure within capillaries has been reported to range between 20-40 mmHg with a average of 32 mmHg.<sup>117</sup> In 1930, Landis used a microinjection method to cannulise the arteriolar limb of capillaries in human fingernail beds to study capillary blood pressure.<sup>117</sup> He reported an average pressure of 32 mmHg in the arteriolar limb, but this may not be a relevant value for capillary pressure in areas at risk for PUs.<sup>118</sup> Kosiak (1959) described results from studies in canine and rat.<sup>45</sup> He found microscopic pathological changes in the tissue after as little as 60 mmHg pressure for one hour. Other authors have reported that high pressure over a short period of time can cause damage and that low pressure applied for longer time does not cause damage to the same extent.<sup>119-121</sup>

While interface pressure of less than 32 mmHg is assumed to be safe by many clinicians, and pressure in excess of 32 mmHg is thought to lead to closure of capillaries, the importance of tissue ischemia still remains to be more investigated. Tissue can withstand pressure more or less, depending on other patient characteristics. Effects of stress deformation has been demonstrated in computer modelling.<sup>96</sup> The complexity of factors leading to development of PUs includes not only local factors but also general risks depending on the health status of the patient. A combination of intrinsic and extrinsic risk factors in the individual patient will decide whether a PU develops or not.<sup>118</sup> (Figure 3)



**Figure 3.**

*A combination of intrinsic and extrinsic risk factors. The figure shows biomechanics at the loaded interface - extrinsic factors. With kind permission from Dan Bader, presentation at EPUAP's 14<sup>th</sup> annual conference in Portugal September 2011.*

### *Shear*

Shear is mechanical stress parallel to the surface. Shear stress deforms and changes the shape of the tissue and occurs usually in combination with pressure.<sup>122</sup> (Figure 2) Shear and friction are often mentioned together and sometimes inaccurately presumed to be interchanged. The synergies of shear and pressure lead to the formation of PUs. Shear stress is caused by friction e.g. when a patient is sliding down in bed or chair.<sup>123, 124</sup> In this situation the shear stress keeps the skin against the surface while the rest of the body is moved downwards.<sup>122</sup> It is logical to conclude that the force applied downward when the patient is in the Semi-Fowler position in bed tends to distort the tissues and blood vessels near the sacrum, placing this region at risk for tissue breakdown. Furthermore, in a research project focused on assessing the pressure-reducing effects of operating table mattresses, Defloor (2000) concluded that elevation of the top end of the operating table to 30° might cause high pressure and shearing force on the sacrum during head and neck surgery.<sup>125</sup>

### *Friction*

Friction is the force of two surfaces moving or rubbing against one another.<sup>122</sup> Microscopic or macroscopic tissue trauma can occur when one surface is moving against another with enough of friction force and weight to the surface.<sup>126, 127</sup> In the study on swine Dinsdale (1973) reported that friction removed the stratum corneum and separated the epidermis from the basal cell layer.<sup>126</sup> This situation can appear when the patient is sliding down across bed sheets or is rubbing heels or elbows to the sheets. Dinsdale also demonstrated that pressure and friction together decrease the tissue tolerance for pressure force.

Moisture, maceration, and tissue breakdown increase the surface tension of the skin and the support surface. Increased skin moisture or incontinence may lead to maceration of the skin, which in turn makes it more predisposed to pressure, shear, and friction damage.<sup>128, 129</sup> It has also been suggested that the use of soap is affecting the aging skin because exposure to water, soap, or other irritants as well as bacteria can irritate skin and potentiate the effects of friction.<sup>130, 131</sup>

### *Microclimate*

In the early 1970's Roaf reported factors contributing to PUs and proposed how to avoid them, specifically by promoting optimal circulation and avoiding long periods of pressure, abrasions, extreme heat or cold, skin irritants and infections as well as by maintaining an optimal microclimate.<sup>132</sup> Moisture of the skin surface can dissolve areas between the collagen in dermis and stratum corneum.<sup>133</sup> This may increase the risk for maceration and can increase the sensitivity of the blood vessels for pressure, shear and friction.

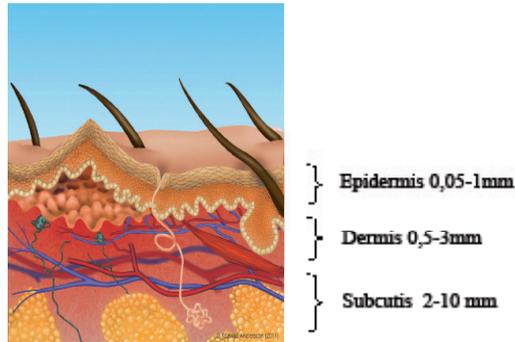
Increasing body temperature is also a risk factor for developing PUs.<sup>134</sup> If the body temperature increases with 1° C, the metabolic activity also increases<sup>39, 135</sup> Subsequently, the need of oxygen and energy in the skin rises by approximately 10%<sup>136</sup> When metabolic needs increase, the resulting diminished tissue perfusion can lead to ischemia. It is suggested that this leads to quicker development of PUs because less pressure and shear force are needed to affect the tissue.<sup>137</sup> Nixon (2000) found that low body temperature during surgery was associated with PU development.<sup>39</sup> Scott (2001) tried to prove this theory and found that the risk for PUs was lower if the patients received warming but this result was not statistically significant<sup>138</sup>. This indicates that further studies are required to establish if skin temperature is a reliable predictor for the development of PUs.

Today the term “microclimate” is used to describe the environment between the skin and the surface of the mattress or cushion. Temperature and humidity are factors of importance for optimal prevention of PUs.<sup>128, 139</sup> Repositioning the patient is one way to control the microclimate under the patient and decrease the heat accumulated between the mattress or support surface and the patient. Appropriate support surfaces as well as use of barrier creams are reported to solve some of these problems.<sup>131</sup>

## **Intrinsic risk factors**

### *Architecture of the skin*

The skin is the largest organ of the body and has a dynamic structure where cellular replacement and modification respond to local needs.<sup>116, 140, 141</sup> This is a continuous process throughout life.<sup>130</sup> The skin offers protection from mechanical disruption and the tissue beneath the skin is protecting the underlying structures. Although the skin has these properties, PUs can occur as a result of the disruption of the vascular network of arteries, arterioles and capillaries after prolonged pressure in vulnerable areas.<sup>107</sup> The skin consists of three layers: epidermis, dermis and subcutis (see figure 4)



**Figure 4**

*The skin consists of three layers: epidermis, dermis and subcutaneous tissue*

Older patients are more vulnerable to PUs because of age-related skin changes.<sup>116, 130, 140</sup> The pathology and aetiology show wide individual variations in how skin responds to pressure. Elasticity of the skin and loss of muscle (strengths and mass) and subcutaneous fat is decreased in the aging skin. This increases vulnerability for pressure and shear forces. The dermal layer becomes thinner, and epidermis vascularisation, proliferation and thickness decreases.<sup>116</sup> The skin plays an important role in the regulation of the body temperature. If body temperature rises, dermal vasodilatation cools the skin by increasing blood flow and perspiration.<sup>39, 135</sup> The properties of the skin, such as the ability to feel pain, sensibility, inflammatory response, decreased sensitivity to cold and heat, making it more vulnerable to injury.<sup>142, 143</sup> Aging leads to a reduced ability to regulate hyperthermia due to decreased peripheral circulation. Increased perspiration increases the risk of excess moisture on the skin surface. Old age and severity of illness and co-morbidity can influence the circulation in the skin, so that less pressure is needed for PUs to develop. (Figure 5)

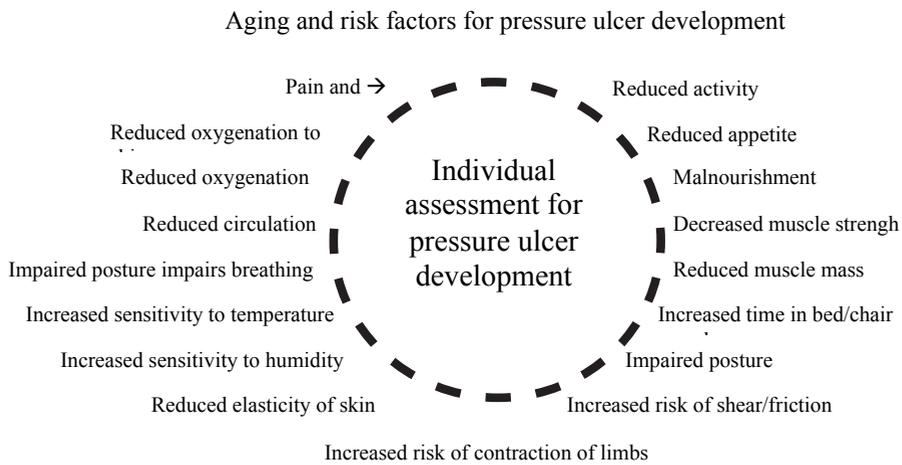
*The epidermis* is arranged in four layers: the stratum corneum, granular layer, stratum spinosum and basal layer (or stratum germinativum) which separates it from the underlying dermis.<sup>140</sup> The stratum corneum is continuously replaced by cells from deeper layers. The changes that occur in the skin during aging affect the epidermis, which becomes thinner.<sup>130</sup>

*The dermis* consists of two layers: the papillary dermis and reticular dermis. Collagen and elastin allow the skin to recover from stretching after pressure.<sup>102, 140</sup> In aged skin the dermis has a reduced number of sweat glands and it produces less sebum. The lack of sebum can lead to dryness of the skin which can affect how the skin reacts to pressure.<sup>118, 130, 144, 145</sup> Dryness can be exacerbated when the skin is cleansed with soap and water. Dry skin has more crevices, and bacterial invasion can be detrimental.<sup>129, 130</sup>

*The subcutaneous* layer consists of fat and is separated from the dermis and deeper structures such as the fascia, muscle and bone. The thickness varies depending on the location on the body and body type and weight as well as the number of fat cells and gender.<sup>140</sup>

### Vasculatory and circulation diseases

Impaired mobility, leads to reduced activity in all ages and in addition to impaired mobility<sup>146</sup>, risk factors such as vascular disease, diabetes and pulmonary disease<sup>43, 147</sup> as well as spinal cord injury (SCI) and other neurological conditions increase the risk of developing PUs.<sup>148</sup> A decrease in peripheral arterial circulation, as measured by Ankle-Brachial Pressure Index, has also been shown to contribute to heel sores.<sup>149</sup> Ischemia, hypoxia, hypotension, anaemia and increased metabolic demands may be potential factors for PU development.<sup>46, 150, 151</sup> In addition malnutrition may also increase the risk of infection and PU development.<sup>151-154</sup> Pain and pain-relieving drugs may also be factors affecting the peripheral circulation since the patient tend to be more immobile both from pain and analgesia.<sup>155</sup> Drugs used perioperatively may also affect the peripheral circulation.<sup>156</sup>



**Figure 5**

*The vicious circle is modified from first version presented in SLL, Vårdprogram, 2010.<sup>157</sup> Old age and severity of illness and co-morbidity can influence the circulation in the skin, so that less pressure is needed for PUs to develop.*

### Tissue perfusion

Superficial PUs - Category I and II PUs - are caused by compromised peripheral circulation. The capillaries are compressed by the external pressure which leads to tissue ischemia, capillary thrombosis and occlusion of lymphatic vessels.<sup>110</sup> When the pressure is relieved, the area is reperfused. The combination of periods of ischemia followed by reperfusion, results in an increased number of reactive agents that cause inflammation in the tissue.<sup>102, 109, 158</sup> The length of time at which tissues can survive without oxygen varies. Ischemia- reperfusion damage is reported to be a significant mechanism in early stages of PU development.<sup>159, 160</sup>

Animal studies show that reperfusion damage is higher in older animals.<sup>161</sup> Older patients may have a similar reduction in tissue reperfusion speed leading to prolonged or absent reactive hyperaemia.<sup>46, 110, 116</sup> This makes it difficult to assess if the skin is insufficiently supplied with oxygen and nutrients or if it is healthy and non-affected by pressure damage. It is also thought that repeated episodes of ischemia and reperfusion injury may lead to failure of healing of established chronic wounds such as PUs.<sup>162</sup>

Tissue perfusion may also be affected by changes in the body temperature<sup>134, 139</sup>, vasoactive drugs<sup>163</sup>, vascular diseases including diabetes and cerebrovascular accident.<sup>39, 91, 164</sup> All these factors, demonstrate the complex interacting cascade of events leading to development of PUs where no single component can be isolated.

### *Cognitive impairment*

Cognitive impairment measured by SPMSQ is reported to be a risk factor for PUs.<sup>165-167</sup> Cognitive impairment defined as severe on the SPMSQ scale led to significantly increased risk of developing PUs.<sup>166</sup> (SPMSQ scale (Appendix 4) Cognitive impairment is common in patients with hip fracture.<sup>168</sup> This is sometimes due to temporary confusion, but in other cases caused by dementia.<sup>156</sup> Known intrinsic and extrinsic risk factors affecting pressure ulcer formation is presenting in table 3

**Table 3**

Known intrinsic and extrinsic risk factors affecting pressure ulcer formation

| Extrinsic  | Intrinsic   |
|--|---|
| <ul style="list-style-type: none"> <li>• Pressure on the tissue</li> <li>• Shear</li> <li>• Friction</li> <li>• Immobility</li> <li>• Maceration, skin irritations</li> <li>• Support surface under the patient</li> </ul> | <ul style="list-style-type: none"> <li>• Architecture of the skin</li> <li>• Age</li> <li>• Acute trauma e.g hip fracture (or illness)</li> <li>• Vasculatory and circulation diseases</li> <li>• Systolic blood pressure</li> <li>• Cognitive impairment</li> <li>• Vasoactive drugs</li> <li>• Anemia, ischemia, hypoxemia</li> <li>• Increased or decreased body and peripheral temperature</li> <li>• Infection</li> <li>• Malnutrition, decreased body mass index</li> <li>• Spinal cord injury</li> <li>• Other neurological conditions</li> <li>• Increased metabolic demands</li> </ul> |

## **Prevention**

### *Pressure relief*

The overall goal of pressure relief is to prevent persistent erythema. The easiest way to reduce pressure is to reposition the patient in either a lying or sitting position together with redistributing the pressure by appropriate support surfaces.<sup>2</sup> However, a systematic review found insufficient evidence to support specific repositioning regimes.<sup>2</sup> The traditional two-hour interval usually serves as a starting point, but it is important to individualise the interval depending on the patient's condition.<sup>169</sup> and support surface. The use of pressure redistributing support surfaces in lying and seating positions does not eliminate the need for repositioning.<sup>170</sup> The overall goal is to establish a regimen in which pressure is completely relieved on all areas of the body. Small shifts and movements both in lying and seating positions have been proven efficient.<sup>104, 171</sup> Tilting the patient between 30-degree positions has been documented to be optimal.<sup>172, 173</sup>

### *Support surfaces*

In selecting an appropriate support surface, the body weight of the patient must be considered. Different support surfaces support patient weights differently. There are two important principles regarding support surfaces: immersion and envelopment.<sup>25, 174</sup> Immersion allows the patient to sink into the support surface. Here it is important that the material is not too soft and that the patient's weight does not cause him or her to "bottom out" resulting in resting on the underlying structure of the bed. Envelopment refers to how well the support surface embraces the whole body, that is, how the surface redistributes pressure. Support surfaces that provide immersion or envelopment allow the patient to be more independent and mobile, for example, it is easier for the patient to raise from a lying to a standing position.<sup>25, 174</sup> Foam mattresses that adhere to these principles are easy to use, but their life span may be short depending on the duration of which they have been used as well as the amount of weight applied.<sup>174</sup> The choice of support surfaces should be directed by individual needs; however, in practice this often depends on the reimbursement practice.<sup>175</sup> Most importantly, all decisions and preventive measures must be evaluated based on the extent to which they achieve the goal that is best for the patient – no development of PUs. Documentation of selection of surface is an important part of the nurses' responsibility. If a person is a high-risk patient or already has a PU, the choice may be an alternating active support surface with cyclical infiltration or an air mattress with constant low pressure to provide immersion and envelopment to redistribute pressure.<sup>174</sup>

Choice of support surfaces should be based on central questions:

- 1) Is the patient mobile and able to get out of bed?
- 2) Can the patient feel discomfort from pressure and shear stress and accordingly change position frequently by him/herself?
- 3) Can the patient ask others to move him/her if unable to move him/her self?

The most commonly used interventions are related to support surfaces (mattress overlays on operating tables, specialised foam overlays), optimising nutritional status and keeping the skin free from humidity.<sup>2</sup> Repositioning is still the most common preventive measure but it is still not known whether it has an advantage over other preventive strategies. The technique used for repositioning of the patient can also influence the development of pressure ulcers, since if the skin is macerated and the patient is drawn incorrectly in the bed or chair both friction and shear forces can contribute to increased vulnerability for pressure.<sup>176</sup> Further well-designed clinical studies are needed.

### *Nutrition*

Optimizing nutritional status is one of the appropriate strategies to prevent PUs and to minimise other complications and death.<sup>2, 177</sup> Malnutrition is common in hospital settings.<sup>178</sup> Eating difficulties and loss of weight together with actual weight (Body Mass Index (BMI) weight in kg/height in m<sup>2</sup>) are recommended to be documented and acted upon (SKL).<sup>97</sup> Malnourishment is defined as patients < 70 years with a BMI score <20 or patients >70 years with BMI <22.<sup>179</sup> (Table 4) A low BMI does not per se mean that there is a nutritional problem. Overweight is not usually a risk factor for PUs in patients with hip fractures in the Nordic countries.<sup>43</sup> Elderly patients with a hip fracture have however been reported often to

be a risk of malnutrition.<sup>44, 180</sup> Prior to surgery it is important to maintain a good nutritional status because patients who are in a catabolic phase have high risk for complications.<sup>181</sup> Beside BMI and identification of eating abilities, Minimal Nutritional Assessment (MNA) is a tool designed to identify nutritional problems for patients over 65 years.<sup>182</sup> For all ages, Subjective Global Assessment (SGA) is commonly used.<sup>183</sup>

**Table 4**

Body Mass Index (BMI - weight in kg/height in m<sup>2</sup>). The international classification according to WHO.

| Classification    | Cutoff points |
|-------------------|---------------|
| Underweight       | < 18.50       |
| Severe thinness   | <16.00        |
| Normal range      | 18.50 – 24.99 |
| <b>Overweight</b> | ≥25.00        |
| Obesity           | ≥30           |

## Morbidity, mortality and suffering

Patients predisposed to PUs have a higher risk of mortality.<sup>20, 35, 38, 80, 184-186</sup> In a follow-up study of patients with PUs in Uppsala<sup>187</sup>, a three-month mortality was reported for 35% of the entire patient group who had PUs. Infection is the most severe complication of PUs. In another study a mortality rate of 48% in 4-year was reported in patient with hip fracture.<sup>180</sup>

Approximately 60,000 people die each year from complications of PUs.<sup>188</sup> Development of PUs has been associated with up to 4.5-times greater risk of death than that for persons with the risk factors but without PUs.<sup>189, 190</sup> Wound-related bacteraemia with sepsis can increase the risk of mortality to 55%.<sup>191</sup> Elderly patients are a vulnerable group since they often have several co morbidities.<sup>147</sup> For the group of SCI patients it has been estimated 7-10% will die as a result their ulcers.<sup>88</sup> Landi (2007) reported from a home care project in Italy that 26% of patients who develop PUs died within 1 year follow up.<sup>80</sup>

Manifest PUs lead to suffering, affecting the lives of patients emotionally, physically and socially.<sup>192-194</sup> They may also contribute to isolation and depression.<sup>195, 196</sup> Patients have also described that pain, discomfort and distress caused by PUs was not recognised by nursing staff.<sup>192, 197, 198</sup> Those affected by PUs also described a sense of changed body image, sleeping difficulties, inadequate treatment and care as well as isolation and need for knowledge and understanding.<sup>199</sup>

## Economic impact

PUs constitute one of the most expensive conditions in health care. In the United States, cost for treatment alone is estimated to be USD 2,000 to 90,000 per PU depending on the severity of the ulcer.<sup>189</sup> This adds up to annual costs of some USD 11 billion dollars.<sup>2, 22</sup> It has also been calculated to an average charge per stay of \$37,800.<sup>59</sup> In Great Britain the cost is estimated at GBP 1,4 to 2,1 billion yearly, representing 4% of the total NHS expenditure.<sup>200</sup> Most of the cost is nurses' time.<sup>200-202</sup> In the Netherlands the cost of treating PUs is the third highest

expenditure after cardiovascular disease and cancer, estimated to between USD 362 million and USD 2,8 billion, a total health care cost of 1% (based on the lowest estimation).<sup>203</sup> In the Swedish county council of Jönköping with 4,200 admissions to the hospital in 2005, 8% had a PUs (mainly Category I-II), the treatment of which was estimated to be SEK 53 million.<sup>204</sup> A PU can become infected, leading to sepsis and amputation. This creates additional costs by re-admission to hospital, antibiotics and prolonged hospital stay and death.<sup>2, 3, 21, 192</sup> This cost is likely to rise due to the fact that an aging population has an increased risk of falling and getting a hip fracture. Prevention of PUs cost less than treatment.<sup>4</sup> It has been estimated to 2,5 times less than cost for treatment.<sup>82</sup> However the largest part of the cost is for nursing time due to turning, mobilization and wound care.<sup>201</sup> Early prevention with technologically advanced equipment has been estimated to be more cost effectiveness than standard mattresses.<sup>201, 205</sup> These costs alone, without the cost of human suffering, demonstrate the importance of preventing PUs and of cost-effective treatment practices.

## **Risk assessment Subjective /objective risk assessment**

### *Risk assessment scales*

A number of PU risk assessment instruments have been developed to identify risk factors in specific risk groups. Sharp (2005) reported that nurses in general do not use tools for assessment of PUs but rely on clinical practice and knowledge of risk factors.<sup>146</sup> Studies focused on risk assessment instruments have not proved reduction of PU incidence rate.<sup>206-208</sup> The implementation of risk assessment according to an instrument is often part of a quality improvement program with focus on reduction of the number of pressure ulcers in a unit.<sup>209, 210</sup> However, use of risk assessment instruments is recommended<sup>211, 212</sup>, even if usage of such instrument has been questioned.<sup>41, 206, 213-215</sup> It is however obvious that regular usage of such instruments enhances awareness of different risk factors, improves continuity of care, decreases the development of PUs and contributes to significantly improved documentation.<sup>216</sup>

NICE guidelines suggest that patients at risk must be assessed within 6 hours of admission.<sup>217</sup> SALAR recommends risk assessment, within a couple of hours, in all individuals over 70 years as well as on those who are bedbound or expected to be so, or who are wheelchair bound or sit most of the day.<sup>218</sup>

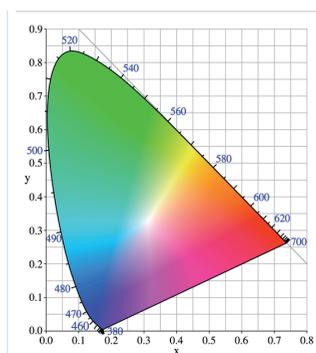
The most commonly used risk assessment instruments in Europe are Braden Scale.<sup>211, 219-221</sup>(Appendix 1), Waterlow<sup>222</sup> and Norton<sup>223</sup> (Appendix 2). Risk assessment instruments are designed to be a complement to general medical examination and clinical assessment built on experience. It is important that all members of the care team (regardless of area of responsibility) use the risk assessment tool in the same way and interpret classification in the same way. The MNS is recommended by the Stockholm County Council and is most widespread in Sweden. It assesses 7 different areas (Appendix 3). The MNS was developed, validated and reliability tested by Ek.<sup>224</sup> It is based on the Norton (1975) risk assessment tool developed for geriatric patients.<sup>223</sup> The MNS differs from the original Norton scale in two areas, nutritional intake and fluid intake, which have been added to the MNS. Lindgren (2002) further developed the MNS into an instrument called RAPS which stands for Risk Assessment Pressure Sore.<sup>225</sup> Risk assessment instruments available today have mainly been

developed for elderly patients. Risk assessment instruments are also developed for patients with spinal damage<sup>87</sup> and for newborns<sup>226</sup> but whether the risk assessment scales reduces the incidence in paediatric care is unknown.<sup>207</sup>

### *Perception of color*

The primary step in the decision-making process when assessing patients' skin is observation of changes in skin colour and detecting early signs of deeper tissue damage. Interpretation of colours is subjective: different colours are interpreted in different ways by different people.

The spectrum that the human eye can see is called the visible spectrum or "visible light" and consists of wavelengths of approximately 400 – 700 nm (Figure 6) One nanometer (nm) is a billionths of a meter. Light, no matter how complex the composition of wavelengths, is reduced by the eye into three colour components. The human eye has rods and cones that process the light in the retina for subsequent processing in the brain. Rods see black, white, and shades of gray and discern the form or shape of an object. They cannot distinguish between colours, but are supersensitive and allow the human eye to see even in the dark. Cones sense color and are most helpful in normal or bright light. The retina has three types of cones and each cone type is sensitive to either red, green, or blue. Combinations of red, green and blue is called RGB.<sup>228</sup>



*Figure 6*  
The spectral colour of the "visible light" of and the combination of  
RGB = Red/Green/Blue.<sup>227</sup>

### *Detection of pressure ulcers*

The observation of a change in skin colour is the first step of the procedure to assess and detect early PUs. The human perception of colour is, however, subjective and based upon the varying sensitivity of the different cells in the retina to light of different wavelengths. Because of external conditions, colour can be perceived in different ways so that a colour can look different in bright sunlight and at dawn, or indoors under a light bulb, lamp or fluorescent light. Humans perceive colour variations in different ways, which means that light red to one person is not the same as the same colour to another. The perception of colour is also heavily dependent on the way that it contrasts with its surroundings. These factors might hazard the subjective visual perception of erythema.

### *Finger press test*

Identification and classification of pressure ulcers Category I still remain unreliable. A finger-press test is most commonly used in order to differentiate Category I PUs from reactive hyperaemia.<sup>37</sup> If the area under the pressure of the finger blanches and then becomes red again once the pressure is released, then it is classified as an area with satisfactory circulation.<sup>46, 53</sup> Blanching erythema indicates pathological changes in the tissue, with inadequate circulation. PUs can develop in a matter of hours.<sup>24, 33, 106, 229</sup> though it can take from 3-5 days before an incipient PU becomes visible.<sup>37, 63, 75, 115</sup> Other signs of an incipient PU include temperature differences in the surrounding tissue; indurations or softness of the tissue and painful, burning or pricking sensations.<sup>1, 230, 231</sup> The latter symptoms may be the only identifiable symptoms for people with dark skin tones on which different skin hues are not visible.

Once the reactive hyperaemia has disappeared the absence of redness does not necessarily indicate that serious damage to tissue no longer exists.<sup>24, 53, 146</sup> In clinical practice, neglected or incorrectly classified reactive hyperaemia may occur because of the delayed body response to pressure.<sup>24, 232-234</sup>

Many questions can be raised related to the finger-press test methodology. So far it is not clear for how long the skin should be relieved of pressure before the test is performed.<sup>235, 50, 141</sup> The precise duration of the finger press before removal of the pressure, is yet unknown. Since the finger-press methodology is of a subjective character a method to standardise the performance was demonstrated by Vanderwee (2006) who used a transparent plastic disc to assess blanching/non blanching erythema.<sup>236</sup> The disc was recommended to be pressed on to the erythema for three seconds but the amount of pressure was not decided.

### *Non invasive measurement of skin erythema*

Reactions from pressure on the skin has interested researchers for several decades and different methods have been tested for its verification, e.g. Laser Doppler, different spectrophotometry methods and transcutaneous gas tension.<sup>95, 237-241, 134, 242-244</sup> Spectrometry and spectrography are terms used to refer to the measurement of radiation intensity as a function of wavelength and are often used to describe experimental spectroscopic methods.<sup>227</sup> Spectral measurement devices are referred to as spectrometers, spectrophotometers, spectrographs or spectral analysers. Colours that can be produced by the “visible light” of a narrow band of wavelengths (monochromatic light) are called pure spectral colours.<sup>227</sup> (Figure 7)

Recognition of the early signs of pressure damage is a major objective for nurses and reactive hyperemia is often detected during daily nursing care.<sup>141, 235, 245, 246</sup> Current methods are, however subjective and may in some cases be unreliable.<sup>83</sup> An objective assessment to complement the human eye to assess early changes in the skin may be of help in clinical situations. In order to determine if a skin area is at risk of developing a PU the ability of the skin to absorb light of different wavelengths can be used.<sup>247-249</sup> This phenomenon is used to analyse the haemoglobin in blood. The same mechanism could be used to quantify minor changes in the colour of the skin.<sup>237, 239, 250</sup>

As Dawson (1980) described that surface colour can be quantified.<sup>251</sup> To do this calculation comprehensive mathematical formulae have been developed. Since the skin consists of

several layers, the surface of the skin can transmit or reflect the light sent out from the conducted light source. This can be calculated mathematically, as previously described in Dawson and Diffey.<sup>251, 252</sup> The mathematical formula has been adapted to the instrument in use to describe the light absorption and scattering from the surface. This makes quantification of for example, the red value in the skin possible.

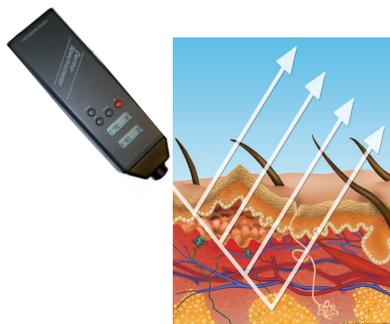
#### *Reflectance spectrophotometer*

The reflectance spectrophotometer (RSM) used in Study III was an early version of the Deraspectrometer a simplified narrow-band reflectance spectrophotometer (Figure 7). Instead of using a white source of light alone, and a monochromator to obtain a narrow reflectance spectrophotometry, a red and a green light diode is used. A blue strengthened, silicone based photo diode is used as a detector to measure the reflected light from the skin. With an increasing proportion of blood in an erythema, a larger amount of green light will be absorbed, i.e. less light is reflected to the detector and the digital readout presents a higher value. The simplified formula<sup>251, 252</sup> used for calculation of the value of reflecting light to the instrument is as follows:

$$\text{Erythema: } 100 * \text{Log } 10 \frac{(\text{intensity of reflecting red light})}{\text{intensity of reflecting greenlight}}$$

$$\text{Melanin: } 100 * \text{Log } 10 \frac{(1)}{\text{intensity of reflecting red light}}$$

The amount of red light does not change noticeably under the same circumstances. The erythema is measured in an area with a diameter of 7 millimeters. A glass protects the diodes and the detector from the skin and provides a flat surface on which to measure. The instrument compensates automatically for surrounding light so that this does not affect the results. Disturbances that affect the measurement results are labelled as “error” in the display, and the measurements must be repeated. Calibration is carried out against a completely white reflecting surface, as well as against a black non-reflecting surface. The method thereby offers a quantitative and objective measurement of a specific aspect regarding inflammatory conditions of the skin. Skin colour varies spontaneously during the day and it is suggested that repeated measurements should therefore be carried out at the same time of day, at the same room temperature and after the skin has been uncovered for at least 5 minutes.<sup>253</sup> It is important to attempt the same measuring circumstances on each occasion of measurement in order to hinder factors that can affect the measurement.



*Figure 7.*

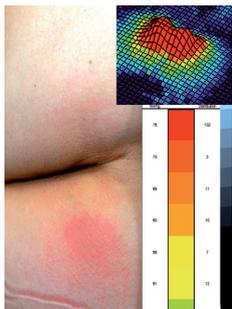
*This illustration is modified from the original presented in Dawson for how the amount of light is transported differently in different human tissues. The non-invasive instrument can quantify minor changes in the colour of the skin.*

### *Pilot examples from functional tests of DS*

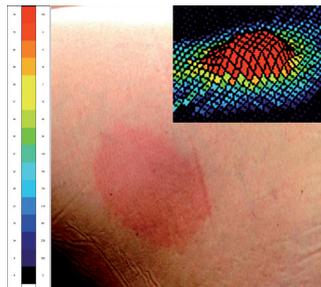
Two pre-tests were performed. The first was to test if spinal anaesthesia would affect the perfusion over the sacral area. The pre-test demonstrated no influence on perfusion following spinal anaesthesia i.e. no increased or decreased values were registered by RSM due to the anaesthesia.

The second test was to determine if time have an influence on erythematic (red) area after pressure relief (Figure 8 and 9). The test show a strong correlation in decreasing Erythema Index (E-Index), with longer time of pressure relief i.e. area less reddish when E-Index was decreasing.

**Figure 8**



**Figure 9**



**Figure 8 and 9**

*Photos reveal different shapes of non-blanching erythema depending on different tissue structure affected. Buttock area to the left and hip on the right.*

*Photos : Maria Amnell, with kind permission*

## **Femoral neck fractures**

### *Epidemiology*

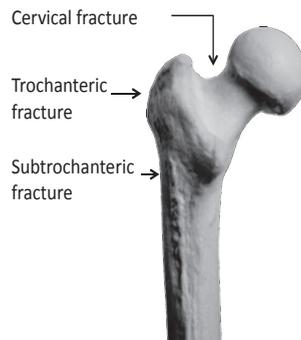
Each year 18,000 people are treated for hip fractures in Sweden. The mean age of patients with a hip fracture was 81 years in the mid-1990s. In the 2010 the age has increased to 83 years (RIKSHÖFT 2010 Annual report). The percentage of men has increased slightly, from 28% in 1996 to 31% in 2008.<sup>254, 255</sup> The number of elderly people in Sweden is on the rise and over the past 20 years, the number of people over 80 years of age who have sustained hip fractures has doubled.<sup>58</sup> Hip fractures among the elderly are expected to increase with age due to an increased risk of falls<sup>256</sup> Scandinavia, North America and Asia have reported the highest frequency of hip fractures.<sup>257</sup> In the United States the incidence of hip fracture has been estimated to be 80 per 100,000 inhabitants<sup>258</sup> It is estimated that by 2050, the number of patients with hip fractures around the world will have increased from 1.66 million (1991) to 6.3 million<sup>168, 259, 260</sup>

### *Pathophysiology*

Hip fractures are classified into three general groups: femoral neck fractures (56%), pertrochanteric fractures (36%) and subtrochanteric fractures (8%) (RIKSHÖFT 2010 Annual report). (Figure 10) Proximal femoral fractures are then sub-classified according to level of complexity. There are more women than men with femoral neck fractures requiring a total hip replacement (THR), the reason for which is not fully understood.

Patients with trochanteric fractures have often previously experienced an osteoporosis-related fracture.<sup>58</sup> These patients are often older and they spend more time indoors than patients with femoral neck fractures. The risk of non-union of the fracture is higher for patients with displaced femoral neck fractures. This carries a risk that surgery will be more complicated, which increases the risk for re-operation.<sup>156, 261</sup> One type of complication that may occur is osteonecrosis of the femoral head. As most of the patients are elderly, it is important to perform surgery as quickly as possible to prevent complications and death.<sup>262, 263</sup> It is also of high importance to mobilise the patient as quickly as possible to avoid complications.<sup>156, 264</sup>

Hip replacement surgery, also called total hip arthroplasty, involves removing a diseased hip joint and replacing it with an artificial joint. Joint replacement is generally carried out to relieve arthritis pain or a damaged joint as part of hip fracture treatment. THR can be performed as a total replacement or a hemi (half) replacement. A THR consists of replacing both the acetabulum and the femoral head while hemiarthroplasty generally only involves replacing the femoral head. Hip replacement is currently the most common orthopaedic operation. Patient satisfaction in both short and long term varies widely.<sup>265</sup>



**Figure 10**

*Pathophysiology - Hip fractures are classified into three general groups: femoral neck fractures (56%), pertrochanteric fractures (36%) and subtrochanteric fractures (8%).*

*Photo: Anna Larsson, Medical illustration, Karolinska University Hospital*

## Pressure Ulcers and femoral neck fractures

### *Epidemiology*

Patients suffering from hip fractures constitute a vulnerable group and therefore may have a higher prevalence and incidence rate of PUs. The prevalence and incidence of PUs in patients with hip fractures varies from 3% to over 66%. Table 2. They become immobilised during the preoperative period at the hospital, before and during surgery and remain immobile until arrival in the recovery room. The length of stay is short and the mean time in hospital in 2010 was 9.4 days (median 8 days RIKSHÖFT 2010 Annual report). After surgery, the patients often need analgesics or sedation over several days, which can mask the signals for position changes.<sup>156</sup> The nurse has an important role in the mobilization of patients post surgery.

In clinical practice it is difficult to determine if a patient with a hip fracture is at risk of developing a PU if the patient is admitted acutely and if the medical history of the patient is not available. Delayed body response to hyperaemia and misinterpretation of erythema are factors which can obscure the detection of PUs.<sup>24, 42, 232, 233</sup> Apart from being a health

risk per se, PUs increases the risk of other complications such as secondary infection of the endoprosthesis. An infection may also lead to sepsis, amputation and death.<sup>266, 267</sup> PUs are particularly problematic in the case of hip fractures, where elderly patients with inadequate circulation have fractures of the neck of femur. Studies have shown that the displaced femoral neck fractures should be treated by hemi or total hip prosthesis (THR).<sup>156, 268-270</sup> The patient's cognitive function has to be considered for the optimal choice of surgical method.<sup>270</sup> The development of PUs in this patient category is particularly serious, as a deep infection in the prosthesis can have very serious consequences for the patient and society.<sup>265</sup>

## **Nursing assessment**

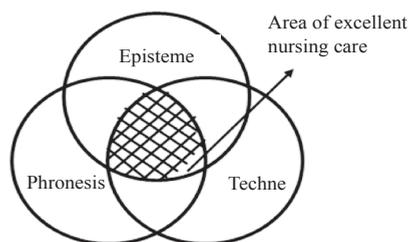
The role of the nurse in the nursing care situation is to identify the patient's needs. In the medical setting, each person has the right to be treated in an ethical and respectful manner. The nursing profession is one (amongst many other relationship-based professions) that requires an ability to deal with patients professionally in a variety of situations and to provide good care to the whole patient; however, nursing also demands particular professionalism so as not to base decisions on emotions.<sup>271</sup> Nursing is in some respects based on practical knowledge like other relationship-based professions such as policing, teaching and healthcare in general. This practical knowledge is based on several sets of skills, as well as on science and intuition.<sup>272</sup>

Assessment is based on a practical knowledge and this concept is sometimes used synonymously with examination and inspection in medical procedures. It most often refers to 1) the evaluation of a condition 2) the process of carrying out such an evaluation 3) an examiner's evaluation of the disease or condition based on the patient's subjective report of the symptoms and course of the illness or condition. In this thesis, assessment is used to refer to the process of identifying risks for developing PUs and includes the visual inspection and examination of the skin and tissue. Assessment of the risks for complications that can arise during the care period is the first step in establishing a care plan in the nursing process. In this process the nurse carries out a complete and holistic assessment. Problems are identified as either actual or potential, as an effort to understand "what she sees" and "get inside" the problem for solving it. The nurse determines how difficult a problem may be to solve and how great the risk is that an identified problem will arise.

### *Good nursing care*

In the nursing profession a capability to meet other humans, to perform correct analyses of observations and to listen is crucial. Much of the practical knowledge and skills is part of the nurse's experience and acts as a hidden source of competence which, even if not used on a daily basis, remains intact and can be practiced intuitively. Good practical knowledge is based on good judgment – phronesis – which means – "knowing when". Phronesis can, together with science - episteme - "to know that" – and theoretical understanding make the difference between good and suboptimal nursing care. The practical knowledge – techne - "knowing how" - is sometimes difficult to prove. In the process of assessing patients at risk for PUs, there are gaps regarding phronesis, episteme and techne. (Figure 11) There is no description exactly as how to assess skin with the finger-press test and no method of measurement or instrument to guarantee accuracy. Such practical knowledge can be affected

by attitudes and level of knowledge<sup>273,11</sup> by the nurses, as well by as how important nurses consider the task and their commitment to their work. Ultimately, it can mean the difference between good and bad nursing care.



**Figure 11**

*Synthesis between Phronesis, Episteme and Techne is a base for professional nursing. In the process of assessing patients at risk for PUs, there are gaps regarding phronesis, episteme and techne.*

Correct decisions can only be made based on knowledge and accuracy when carrying out an assessment. Attitudes and knowledge by nurses are reported to be important factors for professional prevention of PUs.<sup>11,273</sup> The role of the nurse in pressure ulcer prevention is thus to understand and be positive to the importance of risk and skin assessment and prevention strategies.

## The Nursing process

Nursing involves assessing the physiological, social and spiritual needs of a patient.<sup>274</sup> This results in two different processes which should be monitored simultaneously by the nurse in order to give patients good professional nursing care. The nurse must:

- Assess of objective medical parameters e.g. pain and needs of analgesics, blood pressure and oxygen levels as well as different blood tests. The results lead to a decision about the severity of the patients' condition and the emergence of a physician's consultation. It is then the physician's responsibility to evaluate the medical needs of the patient and to prescribe and act accordingly.
- Decide provision of care to the patients using the nursing process e.g. if patients are at risk for PU's, which preventive strategies that should be implemented as well as how the result will be evaluated. Nurses use to care for several of patients and to meet the needs of all the patients is a challenging task.<sup>15</sup>

The nursing process is a problem-solving model or a tool designed to sort the information about the patient in a structured way.<sup>275</sup> It serves to clarify nursing care and makes it possible to identify and provide an individual structured plan for the patient. The care plan is an important tool in delivering nursing care. It can include primary and secondary evaluations. It also includes identification by a nurse of the needs, preferences and abilities of a patient. In addition, it includes an interview with, and observation of a patient by a nurse who considers the signs and symptoms and interprets the patient's verbal and nonverbal communication, medical and social history and any other available information. Among the physical aspects assessed are vital signs, skin colour and condition, motor and sensory nerve function,

nutrition, rest, sleep, activity level, elimination and consciousness. Among the social and emotional factors included in assessment are religion, occupation, attitude toward hospital and healthcare, mood, emotional tone and family ties and responsibilities.<sup>52</sup>

In Sweden the nursing care is based on a model called VIPS which stands for the Swedish words for well-being, integrity, prevention and security.<sup>276-278</sup> The VIPS model is also based on key words which facilitate structured documentation in a patient's medical records<sup>279</sup> concerning assessment (patient history, status), diagnosis and goals, planned interventions, evaluations of the discharges notes.

#### *Nursing documentation*

Patient records are useful in planning and implementing nursing care.<sup>280</sup> If information is complete, describing the need of nursing care this document can lead to a more secure care and high patient safety. Studies have shown that the patient record sometimes is incomplete, even after digitalization and implementation of electronic records.<sup>281-283</sup> The major gain with digital records is that the medical records are accessible and easily readable. However these electronic systems do not always support nursing practice and clinical decisions.<sup>284, 285</sup> This deficit may affect the quality of nursing documentation.<sup>286</sup>

In Sweden, nursing documentation is regulated by the Swedish National Board of Health and Welfare.<sup>287</sup> The nursing process must be clearly stated in the patient record in terms of the initial assessment of the patient's condition, needs, interventions, goals and care plan. Based upon the information gathered, the nurse then makes decisions regarding the nature of the acute problem for the patient, or determines what may become a problem during the hospital stay. This assessment is the first stage of the nursing process.<sup>279, 288</sup>

According to the NANDA classification, a nursing diagnosis is a current or potential problem that a trained and experienced nurse is capable of and qualified to treat.<sup>289</sup> A nursing diagnosis may describe a problem or a risk for a patient, e.g. the risk of developing a PU, or identify a resource used by the patient to mitigate such a risk. Nursing diagnosis has been used internationally since the start of the 1950s and in Sweden from the mid 1980s<sup>275</sup> and NANDA is the most commonly used nursing diagnostic classification. The use of ICD-10 classification codes for diagnoses and complications is not possible in nursing documentation, because the complication code describes only that a patient has a condition, e.g. a PU, not that the PU already existed at admission. Nor can ICD-10 complication codes (L89.0 – L89.9) be used to describe preventive actions or treatment strategies. NANDA can only specify the individual needs of a patient, provide guidelines to promote patient motivation, generate statistics showing the “burden of care” and costs of care, and form the basis of nursing science.<sup>275</sup>

Evidence-based guidelines for the identification and prevention PUs have been developed<sup>25</sup> Adherence to these guidelines is crucial and may help to decrease stress levels for the nursing staff.<sup>290</sup> These guidelines can also aid in documentation, as well as create a platform for patient and care safety<sup>291, 292</sup> and make it easier to follow up on quality indicators. In addition, good documentation using the correct keywords to describe problems and preventive strategies will make it easier for healthcare staff to retrieve such information. In the nursing process, nursing diagnosis is the part of documentation that contains information about the patient that can be used for follow up and for security.<sup>289</sup>

## Patient safety

Preventing PUs is a goal of patient safety and ultimately the responsibility of the nurses.<sup>293</sup> The National Board of Health and Welfare defines patient safety as freedom from unnecessary harm or potential harm associated with healthcare.<sup>292</sup> According to the National Board of Health and Welfare a PU can be defined as an adverse event or injury and is an unnecessary complication associated with healthcare since most PUs are avoidable.<sup>291</sup> A healthcare facility has the responsibility to implement evidence-based working methods to avoid unnecessary complications, as PUs. It means that all health care staff, independent of workplace use preventive strategies and equipment according to the individual needs of the patient, use risk assessment instruments and routine checkups<sup>294</sup> to prevent PUs.

Health care is a high risk area comparable to nuclear power and aviation.<sup>295</sup> Lives can be saved via advanced medical treatments based on modern technology and –often expensive medicines. Life supporting decisions often need to be taken quickly. The risk for well-known and new complications to occur will be more easily identifiable with increased technological advancements. Health care staff can solve many problems due to their awareness of them and prevent errors but the engagement is needed from the management to redesign the working situation in such way that errors are less likely to occur.<sup>15</sup> By the law<sup>291</sup> adverse events shall be reported, investigated and prevention taken to avoid additional ones. PUs are an adverse event, often possible to avoid. Reporting PUs as an adverse event is not common practice since it can be associated with feelings of guilt. This might complicate analyses of potential events during a care –chain, which has led to the development of PUs. Analyses of adverse events can give important insights leading to optimisation of care.

Understanding the system perspective (the organisation) and the human factors (humans are fallible) relationship is important. Reason sub-divided slips and lapses into three main types as recognition failures, memory failures and attention failures.<sup>296</sup> (figure 12) Decisions on both levels may affect patient safety.<sup>297-299</sup> Serious accidents are usually preceded by periods of different events or changes in the organization that lead to a mistake or failure.<sup>298</sup>

If the preconditions in one area within the organization changes, it leads to changes in several other areas. For example if the unit has high patient and staff turnover, it may temporarily be impossible to work according to instructions, local routines and regulations. In these cases, existing knowledge might be obscured. Newly educated nurses have fewer and less rich mental models to apply to new situations. For this reason, they require more time and mental energy to understand patterns of cues using a process of systematic analysis and comparison with possible solutions.<sup>300</sup> At the same time it is important to have situation awareness to make correct decisions. It is not easy to avoid interruptions such as emergencies, telephone calls and issues to handle for other patients and staff. Losing focus can result in adverse events in an acute situation caused by making the wrong decisions or waiting too long to take action. This can happen to all healthcare staff, but since nurses care directly for the patients within the complex structure of the healthcare system they are often at the “sharp end” of error as the last barrier for the patient.<sup>299</sup> Five types of problem areas for nurses which could lead to failure of care have been identified; insufficient information, equipment, staff, supply and simultaneous demands on nurses time.<sup>15</sup>

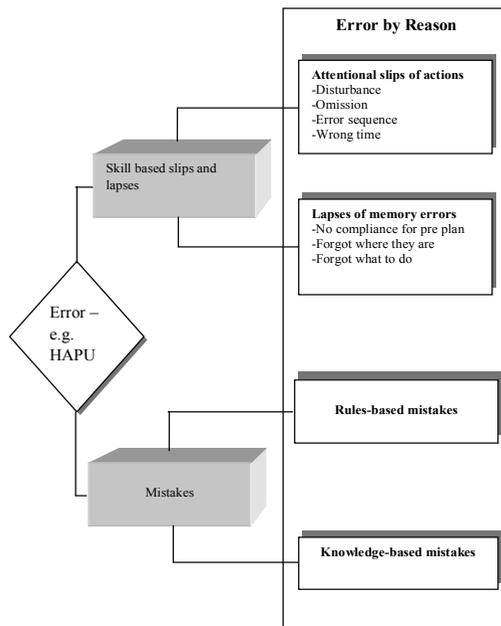


Figure 12 Summary of principal error types by Reason. Model modified with kind permission from Ödegård S, pp 33 (thesis).<sup>301</sup> Reason sub-divided slips and lapses into three main types as recognition failures, memory failures and attention failures.<sup>296</sup>

Cognitive, social and personal resource skills are needed to complement technical skills. New techniques and equipment demands needs to train new technical skills. It is not sufficient to simply use technology and read the results, understanding of how the technology works and how to interpret the results must be continuously learned. This will contribute to safe and efficient task performance.<sup>300</sup> Barrier and support systems can give the nursing staff tools to provide safe care in patient-related work. (Table 5) The role of the nurse in terms of PUs, includes ensuring that the staff follows appropriate evidence-based guidelines for prevention and treatment.<sup>25</sup> This can lead to sustainability in patient safety and prevent PUs.

**Table 5**

A barrier is defined as a physical hindrance, a wall or a borderline that either prevents an act from occurring or reduces the consequence of an action. Below are some of the barriers associated with the prevention of PUs.

| Barrier                       | Example in the prevention o pressure ulcers   |
|-------------------------------|---|
| Tangible or physical barriers | Mattress with preventive functions e.g. in all beds. Friction and shear reducing materials in overlays  |
| Functional barriers           | Special control stations, or a code, for changing the automatic pressure reducing surface mattress function   |
| Symbolic barriers             | Alarm functions of an automatic mattress or an indicator for high pressure at one part of the body which needs to be relieved.<br>Visible mark at the bed for 30 degree elevation of the head |
| Intangible barriers           | Adherence to local and international guidelines aimed at preventing PUs.<br>Rules and checklists for what to do with a patient identified as a risk patient.                                  |

## Quality indicators

In order to ensure patient safety and good quality of care, activities carried out must be measured and monitored. Indicators make it possible to compare between the processes, outcomes and costs over time. Health indicators can be used to define public health problems at a particular point in time, in order to indicate changes over time in the level of the health of a population and/or an individual. Additionally it is important to define differences in the health of populations, and to assess the extent to which the objectives of a program are being attained.<sup>302</sup>

The terms “quality indicator” and “quality measure” are often used synonymously. When using a quality indicator the results from activities in healthcare can be followed.<sup>17</sup> How the results are followed depends upon the area to be investigated. To demonstrate good quality in healthcare, the variable must be measurable. Quality indicators in nursing care include documentation of the risk of developing PUs, malnutrition, falls and nursing documentation.<sup>17</sup> These indicators can be of help in identifying conditions and areas that should be studied more closely in terms of causation and opportunities for improvement. Traditionally, these indicators are structured according to the model from Donabedian.<sup>303</sup> The structure shows the resources that are available, the process describes what is done and the result is the outcome of the structure and process.<sup>17</sup> Measurements should be valid, measurable, influential and possible to interpret, or clear. There is a clear connection between these three parts and quality cannot be presented if any one of them is missing. The process indicators provide information about fields that healthcare providers can change directly e.g. assessment of the risk of developing PUs.

The concept of Good Health was launched in conjunction with the publication of the Swedish National Board of Health and Welfare regulations on the management of quality and patient safety.<sup>304</sup> Six areas have been highlighted as important prerequisites for good health. The meanings of each Good Care area are clarified in the report. The six areas are:

- Knowledge-based and efficient health care
- Safe health care
- Patient-focused health care
- Effective health care
- Equality in health care
- Healthcare delivered in a timely manner.

The government together with National patient safety initiatives SALAR<sup>97, 218</sup> also carry out indicator-based comparisons of healthcare quality and efficiency among the various regions and counties in Sweden. One purpose of the yearly reports is to make the publicly financed healthcare system more transparent. Another purpose is to promote healthcare management and control.

SALAR has introduced several systematic follow ups connected to quality indicators. This includes a more systematic follow-up of outcomes from the entire healthcare field. Areas defined as preventable: PUs, falls, post operative wound infections, urinary tract infections,

infections from central venous and peripheral venous catheters, malnutrition, drug related problems including those in patients transferred between different care levels.

### **Government initiative**

SALAR<sup>97</sup> is a politically- driven organization which has actively addressed adverse events in health care since 2008. One of these adverse events is PUs.<sup>218</sup> The initiative focuses on raised awareness of certain risks in health care and aims to improve a safety culture that emphasises prevention. Risk assessment is recommended to be performed in all individuals over 70 years as well as in those who are bedridden, expected to be so, wheelchair bound or sitting most of the day. SALAR recommends carrying out the initial assessment within a few hours after the arrival of a patient at a hospital, or a resident at a nursing home. The risk assessment should be performed with a reliable instrument and repeated when needed, for example, if the condition of the patient deteriorates, or if the patient undergoes major surgery, as well as before transferring the patient to other caregivers. Skin assessment is another measure that should be carried out upon admission and regularly thereafter. In addition, an individual care plan should be written and communicated.

# AIMS OF THE STUDIES

---

## **Study I**

To investigate prevalence and incidence of pressure ulcers upon arrival and at discharge from hospital, and to identify potential intrinsic and extrinsic risk factors for development of pressure ulcers in patients admitted for hip fracture surgery.

To illuminate potential differences in patient logistics, surgery, pressure ulcer prevalence and incidence and care between Northern and Southern Europe

## **Study II**

The primary purpose of this study was to establish the inter-rater reliability between blanching and non-blanching erythema assessed by two independent assessors. The secondary purpose was to investigate potential correlations between risk factors and pressure ulcers.

## **Study III**

The purpose of this investigation was to explore if a non-invasive objective method could differentiate between blanching/non-blanching erythema in the sacral area of patients undergoing hip fracture surgery.

## **Study IV**

To investigate the degree of documentation regarding risk assessment, preventive measures taken, prevalence and severity of pressure ulcers, in patients undergoing surgery for hip fractures or elective hip replacement at admission and during hospital care at an orthopaedic unit.



# MATERIALS AND METHOD

---

## **Design**

The design used of the different studies was prospective, comparative, experimental and retrospective review (Table 6). In Study I the design was a prospective, descriptive cohort study with the inclusion of 20 consecutive patients from each participating hospital in 6 European countries. A total of 635 patients with hip fractures were followed throughout the care period for a maximum of 7 days. The design in Study II was a prospective comparative observational study to establish the inter-rater reliability between blanching and non-blanching erythema assessed by two independent assessors. The method used for the blanching/non-blanching test was visual observation and finger-press test performed in the sacral area. Seventy eight patients with hip fractures were followed up for maximum of 5 days after surgery and 156 assessments were conducted independently. Study III was an experimental prospective comparative observation study comparing the results from the blanching/non-blanching test performed with finger-press test and the E-Index using a simplified narrow-band reflectance spectrophotometry in the sacral area. Seventy eight patients with hip fracture were followed up for a maximum of 5 days after surgery and daily assessments were conducted using the RSM. Study IV was a retrospective study, a repeated one-day monthly point prevalence survey from January 2007 until October 2010 (46 months) at an orthopaedic wards. Data were collected one day monthly from the computerised patient record system. 2,281 patient records were scrutinised.

## **Settings**

All of the studies were conducted between 2002 and 2010 and the study sample was orthopaedic patients with hip fractures (Study I-IV) and (in Study IV) THR. Study I was initiated by EPUAP and involved 6 European countries. Trustee members of EPUAP conducted the study in their respective countries. The study was supervised by a coordinator in Sweden. Study II and III were initiated and conducted in the Department of orthopedics at the Karolinska University Hospital in Stockholm and Study IV was conducted at Danderyds Hospital in Stockholm. In Study IV, the orthopaedic patients were divided into 4 groups. The groups of patients with hip fractures and THR were investigated in more detail.

### **Data collection**

Study I was based on a protocol specific for the purpose and designed as 3 different parts (see also Methods). The data were transcribed in formats that enabled analyses in statistical programs. In Study II-IV a specific designed data collection tool based on protocols specific for the respective studies, was used to obtain information such as:

- 1) Age and gender (Study II-IV)
- 2) Diagnoses for admittance to hospital on the study day (Study II-IV)
- 3) Documented risk assessment with MNS at admission and/or during hospital stay (Study II and IV)
- 4) Documented presence or absence of pressure ulcers at admission and during hospital stay including categorization of the pressure ulcers (Study II and IV)
- 5) Documentation regarding if PU was hospital-acquired, plus category of pressure ulcers (Study II and IV)
- 6) Documentation of BMI at admission (Study II and IV)
- 7) Prevention at admission and during hospital stay (Study IV)
- 8) Length of hospital stay (Study IV)

The data were transcribed in formats that enabled analysis in statistical programs.

**Table 6** Overview of the four studies

| Study                           | I  | II  | III   | IV   |
|---------------------------------|--|---|---|--|
| Design                          | Prospective, comparative   | Prospective, comparative observational  | Experimental, comparative observation   | Retrospective  |
| Sample                          | 635 patients   | 78 patients, 154 nurses   | 78 patients   | 2,281 patient medical records  |
| Settings<br>Orthopedic patients | Patients with hip fractures in 6 European countries  | Patients with hip fractures and staff in a university hospital  | Patients with hip fractures in a university hospital  | Patients in a university hospital  |
| Data period                     | 2002   | 2005  | 2005  | 2007- 2010   |
| Statistics                      | Crosstabulation<br>Frequency counts and percentages<br>Mean<br>Median<br><br><i>Association:</i><br>Pearson's chi-square test<br>Mann-Whitney<br>U-test<br>T-test for independent sample | Crosstabulation<br>Frequency counts and percentages<br><br><i>Agreement:</i><br>Kappa statistics<br>Weighted kappa<br><br><i>Comparison:</i><br>GENMOD*<br><br><i>Association:</i><br>Pearson's chi-square<br>Fisher's exact test | <i>test-retest reliability:</i><br>ICC*, SEM*<br>Kappa statistics<br><br><i>Association:</i><br>Mixed linear models with one and two factors. Pairwise comparisons with Bonferroni adjusted p-values. Estimates presented as means and CI*<br><br><i>Sensitivity and specificity ROC*</i> | Cross tabulation<br>Frequency counts and percentages<br>Mean<br>Median<br><br><i>Association:</i><br>Pearson's chi-square<br>Logistic regression analysis  |
| Data collection                 | Data collection protocol<br>Risk assessment with Braden Scale<br>Skin assessment, Patient medical record<br>Cognitive function test by Pfeiffer<br>Co-morbidities                        | Data collection protocol<br>Finger- press test<br>Visual observation<br>Patient medical records<br>Skin assessment – PUs<br>Risk assessment by Modified Norton Scale<br>Body Mass Index (BMI)<br>Co-morbidities                   | Data collection protocol conduct for the study<br>Finger-press test – PUs<br>Skin assessment<br>Narrow-band reflectance spectrophotometry   | Patient medical records – check for PUs<br>Documentation of:<br>Risk assessment<br>Skin assessment<br>PUs<br>BMI<br>Prevention strategies<br>Co-morbidity<br>Length of stay<br>Admission acute or elective |
| Cognitive function test         | SPMSQ  |   |   |  |
| PU Risk assessment scale        | Braden   | MNS   |   | MNS  |
| PU skin classification          | EPUAP/NPUAP Classification scale   | EPUAP/NPUAP Classification scale  | EPUAP/NPUAP Classification scale<br>Dermaspectrometer   | EPUAP/NPUAP Classification scale   |

\* SPMSQ=The Short Portable Mental Status Questionnaire

MNS=Modified Norton Scale

EPUAP=European Pressure Ulcer Advisory Panel

NPUAP=National Pressure Ulcer Advisory Panel

GENMOD= a generalised estimating equation model for repeated measurement analysis of binomial outcomes

ICC= Intra-class Correlation Coefficient

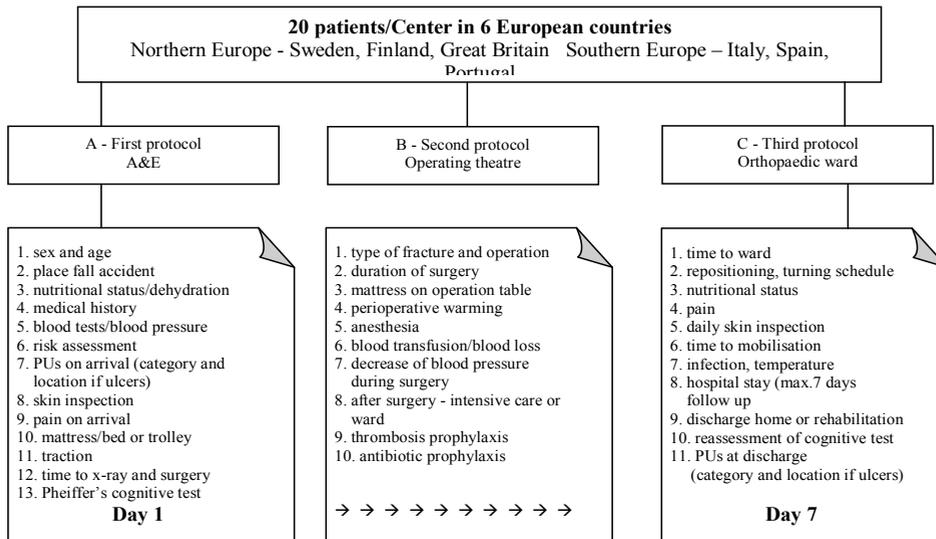
SEM= the Standard Error of Measurement

ROC= Receiver Operating characteristic Curve

CI=95% Confidence Intervals

## Study I

### Flow chart

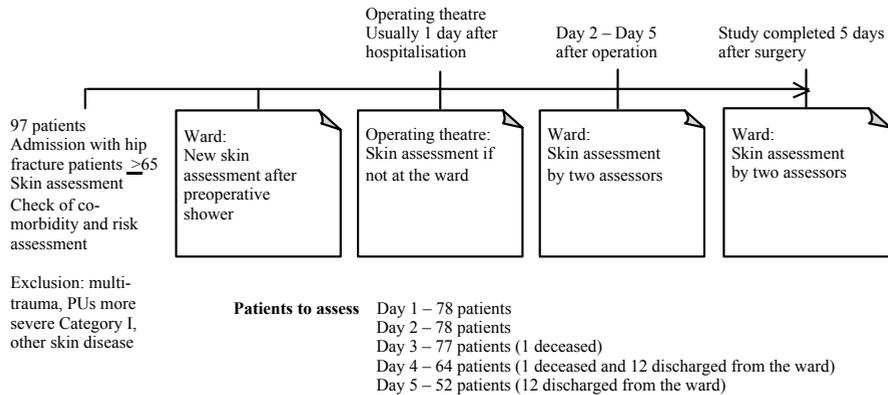


The study was designed as a prospective cohort study with the inclusion of 20 consecutive patients with a radiologically-verified diagnosis of hip fracture, who after providing verbal and written information consented to participate and were included. In cases where the patient was confused, their next of kin gave consent. Each country had an experienced study nurse, who was responsible for the selection of centre's and education of the local investigators and staff. Multi-trauma patients were excluded from the study.

The study protocol agreed upon was divided into 3 main sections. Section A was aimed at collecting patient- and care-related data at the Acute & Emergency Department (A&E). Section B comprised questions related to perioperative care and in section C data regarding postoperative care were recorded. The patients were followed up until discharge or for 7 days, whichever was first. The patients' skin was inspected daily from head to toe and documented on an anatomical drawing. Classification of PUs was standardised and a 'pressure ulcer card' with colour pictures guiding the investigators to the correct classification was used.

## Study II

### Flow chart



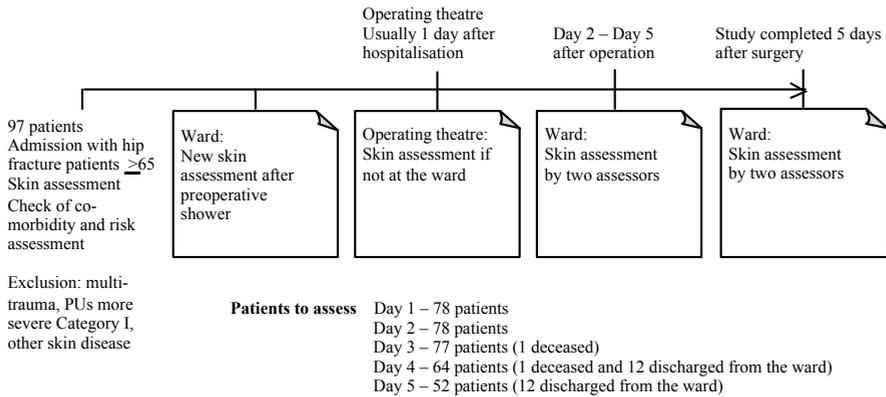
The inclusion criteria for this prospective comparative observation study were patients with hip fractures who were admitted to an orthopaedic ward, aged  $\geq 65$  years. Patients with pre-existing skin dermatoses or pressure ulcers  $\geq$  Category 2 in the sacral area were excluded. No major accident or trauma patients were included. At the time of the study, patients with hip fractures were treated by either an orthopaedic or geriatric ward. If the patients were deemed able to return to their previous style of living, they were treated by the geriatric ward. These patients were excluded from the study. Ninety-seven patients over 65 years with hip fractures were consecutively recruited for this prospective, comparative observation study. The sacral area was visually assessed upon admission, during hospital stay and at discharge. Erythema in the same area was also tested by a light finger-press test by two independent assessors.

From the first postoperative day and on day 5 after surgery—unless the patients were discharged earlier— the sacral area was assessed to determine if the skin was erythematous or not. Prior to the skin assessment, the patient was placed in a lateral position and stabilised using pillows. The skin of the patient was cleansed using tap water (37°C) and mild shower gel. The sacral area was relieved of pressure for 5 minutes before the assessment was carried out. This assessment was performed each morning at breakfast time by two independent assessors. The results from the assessments were documented.

Potential risk factors such as high age, gender, type of diagnosis, low or high BMI, blood loss and transfusion, low haemoglobin, low MNS and diseases were documented. The MNS was used for risk assessment, with a cutoff point  $\leq 20$  for a high risk of developing pressure ulcers. BMI was used to identify patients with low or high body weight. Malnourishment was defined as patients  $< 70$  years with BMI score  $< 20$  or patients  $> 70$  years with BMI  $< 22$ .

### Study III

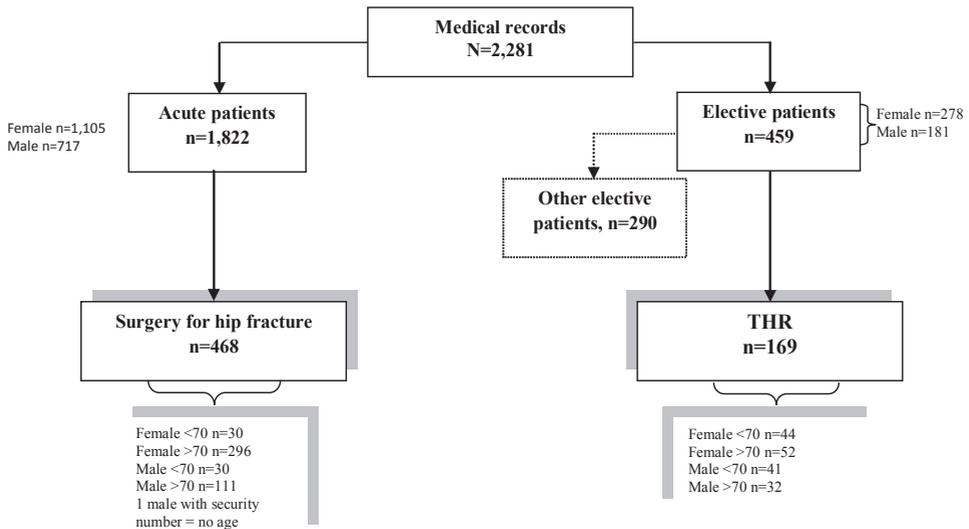
#### Flow chart – Same patients as in Study II



The study design was experimental and the study sample was the same as in Study II. It was only possible to perform the pre-measurement after the spinal anaesthesia in 22 patients since the process was too time consuming and delayed surgery. From the first postoperative day until Day 4 after surgery –unless the patients were discharged earlier - the sacral area was assessed to determine if the skin was erythematous or not. The sacral area was scrutinised using a simplified narrow-band reflectance spectrophotometer (RSM) with a digital reading. This method offers a quantitative and objective measurement of a specific aspect regarding redness of the skin, the E-Index. The instrument measures the amount of light reflected by the skin (green light 568 nm and red light 655 nm) at different structures in the tissue. When the number of erythrocytes increases in the tissue, a greater amount of green light is absorbed and less is reflected. This absorption is registered on the reading as raised amplitude, redder skin and a higher of E-Index value. Prior to registration, a waterproof pen was used to mark the skin with 8 measuring points and 1 reference point that was not loaded by pressure. Each point was measured 3 times. The reference point was located on the side opposite to the hip fracture. Two trained “study nurses” performed the registrations with the RSM.

## Study IV

## Flow chart



This retrospective study was a repeated one-day monthly point prevalence survey from January 2007 until October 2010 (46 months). Data were collected one day monthly from the computerised multi-professional patient medical record system. The days of the point prevalence survey were selected on a rolling schedule so that all days of the week were included. A total of 2,281 patient medical records were scrutinized and divided into 4 groups, hip fractures, THR, other fracture and an elective patient group. The hip fracture and THR group was scrutinized in more detail. Records from patients, who were hospitalised for an extended period, may have risked being included in more than one month's sample but were only counted once in the result. The study was performed at an orthopaedic department at a university hospital in the Stockholm metropolitan area. The Department has 52 beds, performs both emergency and elective surgery and has an annual admittance of approximately 3,500 patients with a dominance of acute care. Patients with hip fractures constitute the major diagnostic group. Data collected from patients' records comprised documentation of age, gender, risk assessment by MNS, nutritional status and BMI. Documentation in the patient record was scrutinised during the hospital stay for pressure ulcers, preventive measures, surgical methods, and medical diagnoses e.g. dementia, but only for the patient group with hip fractures. Patients who had surgery for hip fractures in other settings, patients with pathological fractures periprosthetic fractures and those who had undergone re-operation or has a hip fracture treated conservatively were excluded.

### **Ethical considerations**

The studies were approved by the local ethics committees at each hospitals in each country for Study I (Dnr 01-121) and Studies II, III, IV by the local ethics committees (Dnr 00-423 and Dnr 04-563/2, 2009/1376-32). These were conducted in accordance with the ethical principles of the Declaration of Helsinki (Helsinki Declaration 1989). This thesis also complies with the ICN Code of Ethics for Nurses (ICN 2006).

Patients were informed both orally and in writing about the studies and informed that they were free to withdraw from the study at any time without providing a specific reason. Regular skin inspection is part of normal professional nursing routines, but the extra assessment of the skin with reflectance spectrophotometer (Dermaspectrometer) might have caused the patient mild positional problems. However, the principle of good was deemed to overrule the short period of patient discomfort. The study patients may also have received more individual attention, which was perceived to be positive.

Different ethical considerations had to be considered in the studies. Patients admitted to hospital with hip fractures could have had cognitive impairments prior to admission or could have developed confusion due to various factors such as pain, fear, unfamiliar environment and new people around them. It is therefore particularly important to have an ethical approach when including such patients in studies.

In Study I-III, the patients' skin was inspected on several occasions during the hospital stay. This procedure follows guidelines for detecting established PUs or the signs of developing ones. It is important not to violate patients' integrity and dignity especially since the sacral area might be regarded as a private zone. Actions were taken to minimise this potential discomfort by covering as much of the area under inspection as possible. It is important to inform patients of findings, since they cannot see what is being done behind their back. As an investigator, you need to be sensitive to the patients' body language, especially if they are confused

SPMSQ is a test designed to diagnose impaired cognitive function. Inability to answer certain questions might be embarrassing to some patients. The staff conducting the test might for this reason be tempted to avoid some of the questions. It is thus crucial to give careful instructions to the staff prior to study start.

In Study IV, patient medical records were reviewed retrospectively. This methodology provides important knowledge and the results can serve as a platform for quality improvements on a meta-level. Protection of data regarding individual patients is mandatory, and is regulated by law

# SUMMARY OF RESULT

---

## **Study I**

Of the 635 patients, 10% had pressure ulcers upon arrival at hospital and 22% at discharge (26% North and 16% South). The majority of ulcers were Category I and no Category IV ulcers were detected. Cervical hip fractures were more common in the North while trochanteric were more numerous the South. The waiting time for surgery and duration of surgery was significantly longer in the South. Traction was more common in the South and perioperative warming was more common in the North. Risk factors of statistical significance that correlated to pressure ulcers at discharge were: age  $\geq 71$  years ( $p = .020$ ), dehydration ( $p = .005$ ), moist skin ( $p = .004$ ) and total Braden score ( $p = .050$ ) as well as subscores for friction ( $p = .020$ ), nutrition ( $p = .020$ ) and sensory perception ( $p = .040$ ). Co-morbid conditions of statistical significance for the development of PUs were diabetes ( $p = .005$ ) and pulmonary disease ( $p = .006$ ). Waiting time for surgery, duration of surgery, warming or non-warming perioperatively, type of anaesthesia, traction, and type of fracture was not significantly correlated with the development of pressure ulcers.

## **Study II.**

This prospective, comparative observational study included 78 patients of which 64 were women and 14 men. The mean age for women was 82 years (range 65-100 years) and for men 74 years (range 65-91 years). Fifty-five percent of the patients had pressure ulcers in the sacral area at discharge from the orthopedic ward, 45% had Category I pressure ulcers and 13% had Category II pressure ulcers. No category III and IV PUs were documented. Finger-press tests and visual observation alone were not reliable methods to discriminate between blanching and non-blanching erythema. The proportion of patients with persistent discoloration differed significantly from Day 1 to Day 5 ( $p = .013$ ). Further analysis showed that the probability of non-blanching erythema was higher from Day 2 to Day 5 compared with Day 1 ( $p < .01$ ). When analysing inter-assessor agreement for the subset of patients where the two assessors agreed on the visual assessment of Category I and II ulcers, the strength of agreement at 5 days was poor to moderate.

## **Study III – Same patients as in study II**

In this experimental, comparative observation study 97 patients were recruited on admission to the hospital. Nineteen patients did not complete the study. Reasons for this included the fact that the patient had to wait for more than 24 hours for surgery (of 11 patients, 5 had Category II and III PUs), the patients were referred to another ward (4 patients), or the patient died (1 patient). Seventy-eight patients were included. The sacral area of all patients was assessed by the conventional finger-press test and a digital reading of the E-Index by reflectance spectrophotometer (RSM). The patients were examined at admission and over

4 days post-surgery. RSM measurements proved to discriminate between blanching/non-blanching erythema. The reliability, quantified by the intra-class correlation coefficient, was almost perfect over the measurement period and varied between 0.82 and 0.96 and a significant change was recorded in the areas from Day 1 to Day 5 ( $p < 0.0001$ ). The value from the reference point did not show any significant changes over the period ( $p = 0.32$ ). To analyse the ability of the E-Index to discriminate between the sub-groups “blanching” and “non-blanching erythema,” ROC curves were used one per day. A cut-off value was considered positive if sensitivity and the specificity, respectively, were high.

#### **Study IV**

This retrospective day point prevalence study in an orthopaedic department, at a university hospital, included 2,281 patient records. The patient sample consisted of orthopaedic inpatients. In the total sample of patients ( $N = 2,281$ ), 1,383 (39.4%) were female with a median age of 78 years (interquartile range 66-86 years) and 898 (60.6%) were male in the median age of 68 year (interquartile range 51-80 years). For the 2,281 patients, PUs were documented at admission in 3.3% ( $n = 76$ ). These 76 patients had a total of 119 PUs. Distribution between the acutely admitted and electively admitted patients was 3.4% ( $n = 71$ ) for acute and 1.1% ( $n = 5$ ) for elective respectively. During the hospital stay 10.5% ( $n = 240$ ) had documented PUs. The distribution was 12.4% ( $n = 226$ ) of all acute patients and 3.1% ( $n = 14$ ) of the elective. A total of 355 PUs was documented. Documentation regarding progression of several PUs detected upon admission was lacking. For the 1,822 patients who were admitted acutely, 46.5% ( $n = 848$ ) had a BMI value documented in the patient record, while for patients undergoing elective surgery ( $n = 459$ ) it was 63.6% ( $n = 292$ ). Risk assessment at admission by MNS was documented in the patient record for acutely admitted patients in 38.3% ( $n = 697$ ) versus 39.2% ( $n = 180$ ) of patients admitted for elective surgery. Regression analysis of missing data for BMI and MNS showed a slight tendency to more complete registration during the summer. An age-dependent correlation was found with an increasing number of missing data for both parameters with increasing age. Besides lack of documentation of BMI and MNS it was difficult to follow the development of PUs and prevention strategies taken.

# DISCUSSION

---

## **Patho-physiology of pressure ulcers**

Early investigators focused on pressure as the primary cause of PUs. For example Witkowski and Parish were among the first to publish on the histology of PUs in human skin exposed to pressure, which included vascular infiltrates, thrombosis and oedema.<sup>305</sup> The earliest research was carried out through animal studies. In experiments on rats conducted by Husain (1953) histological changes primarily in muscles of a rat's leg after pressure was applied were studied.<sup>306</sup> Romanus (1977) tested the effects of pressure on rat tail. Kosiak (1959,1961)<sup>45, 115</sup> investigated the relationship between amount of pressure, duration of application and development of tissue damage on canine and rat. Dinsdale (1973)<sup>126</sup> showed that a combination of friction and pressure produced lesions in the epidermis of swine. Salcido (1994) studied application of pressure for 6 hours to the skin over the hip of anaesthetised rats. Pathological changes were detected in the dermis and subcutis.<sup>307</sup> These early animal studies are experimental and may be far from clinical situations. This can lead to difficulties in implementing results and conclusions in clinical situations. However, this can increase the understanding of development of PUs. Low microvascular response in sacral area and sensitivity to temperature may explain the development of PUs which has been reported by Ek (1984 and 1987) and Schubert (1989).<sup>134, 308,309</sup> At present research is focusing on the effect of biomechanical forces caused by a combination of pressure and shear stress on muscle.<sup>109, 118-121, 310-312</sup>

Even with significant efforts designed to reduce PUs, such as regular prevalence studies, guidelines and care-programmes, the prevalence of PUs continues to remain fairly stable over time. Modern preventive measures and materials, however have in many cases reduced the number of severe ulcers. It is likely that with more precise methods which detect the early signs of pressure and shear damage it will be possible to prevent even more PUs. Results of prevalence studies cannot always be compared, since different methodologies are used.<sup>19</sup> In some studies Category I pressure ulcers are excluded (Table 2), while in others, data are collected via interviews with the staff. Some studies draw conclusions from retrospective data. However, in recent years, a commonly used methodology to capture correct data has been developed by EPUAP.<sup>76, 313</sup> With the EPUAP-protocol, all patients in a unit are inspected and the number of patients with PUs is divided by the total number of patients investigated in the unit (= prevalence). Prevalence is however just one side of the coin. Severity and location of the PUs are as important. Prevalence at discharge from hospital may also be a very important information. Incidence is difficult to measure in patients with PUs, since we do not know the exact time frame between pressure damage and the appearance of the PU. In this thesis we have thus expressed the frequency as the number of patients with PUs upon admittance, hospital stay and at discharge as well as total of PUs documented at admission and during hospital stay (Study IV).

The previously reported trend towards fewer PUs in Southern Europe than in Northern Europe was confirmed in our first study.<sup>43</sup> The number of PUs developed between admittance and discharge was almost doubled both in south and north Europe. This might be explained by a number of risk factors such as reduced mobility and motility, but also by intrinsic factors such as morbidity, particularly pulmonary disease and diabetes. The patho-physiological explanation might be a reduced supply of oxygen to the tissues due to capillary occlusion or general lack of circulating oxygen.<sup>109, 110, 116, 158</sup> It is more difficult to explain the differences between north and south. It has been proposed that the texture of the skin might differ due to, for example, intake of different types of nutritional fat. This is however only speculation. Time factors did not offer an explanation either, since patients in the south were generally waiting longer for surgery and had surgical procedures which lasted longer.<sup>43</sup> Inspection of the skin was routine in the A&E, in 1% of the cases in the north and 8% in the south. In acute situations, other actions seem to have been given higher priority, even if the deterioration of a pressure lesion can be rapid.

### **Skin assessment**

One explanation of the difference in prevalence figures in the north and south might be that skin assessment is performed differently in different countries. Tissue tolerance is an individual response to external trauma and it decreases with age. It is important to understand the physiology of the skin in order to prevent PUs<sup>146, 314</sup>, thus a simple method to detect early pressure damage is required.

EPUAP has recommended that two people should perform skin assessment to detect potential PUs. This requires significant efforts and investments, mainly in staffing costs, because it is a staff-intensive assignment.<sup>315</sup>

A pan-European study offers many opportunities but also involves a number of difficulties. The healthcare system may be different in the countries, and it can lead to variations in care and adherence to protocols. Some centres that had agreed to participate were unable to do so, while others included more patients due to local conditions. In the present study a series of statistical analyses were originally planned. Several of these were rejected by the statistician who was later involved in the data analysis. This study, however, verified other studies that have reported a lower prevalence of pressure ulcers in Southern Europe. The reasons for this are poorly understood and require a special study.

There were also local discussions about the registration of mobility and activity in the Braden scale, since in some cases, the pre-fracture status of the patient was documented and not the present status. This is often discussed even when using other risk assessment scales and should be clarified in the instructions.

Study II aimed to investigate the inter-rater reliability of current methods of detecting Category I PUs, that is, visual assessment and the finger-press test<sup>37, 46, 53</sup> Visual assessment proved to be slightly more reliable, but observation can be impaired by a variety of factors. Perception of colours is subjective and based upon the varying sensitivity of different cells in the retina,

reacting to light of different wavelengths.<sup>227, 228</sup> External conditions also cause variations in the perception of the colors. Each person sees the color variations in different ways which means that light red for one person is not the same as for another. The perception of color is also dependent on contrasts in the surrounding environment. Colors for example, look different in bright sunlight compared to at dawn, or indoor lighting. The indoor lighting can be “warm” white or “cold” white depending on the type of bulbs used. This can influence skin assessment as well. Additionally, skin tone for example light or dark skin, can also lead to varying manifestation of color on the skin surface.<sup>96</sup>

Normally, after visual assessment, nurses determine if the finger-press test is required.<sup>46, 53, 146, 316</sup> The finger-press test which has hitherto been the golden standard of detecting Category I PUs, has in the present study proved to be unreliable and inter-rater reliability was poor between different assessors.

One factor that might bias the results of the finger-press test is the potential effect of time of off-loading prior to the skin assessment. Potential effects of off-loading, and the optimal time for this previous to assessment of erythema is hitherto unknown. Early animal studies have suggested that the time required for pressure relief is half as long as duration of applied pressure<sup>45</sup> e.g. 2 hours of exposure to pressure would require 1 hours of pressure relief. In the clinical setting, however, this is unrealistic. A patient with a hip fracture cannot be placed in the lateral position for a long period due to pain from the fracture. This position also predisposes the patient to develop new pressure ulcers.<sup>173</sup>

This study shows that it is difficult to assess the skin and tissue in this group of patients. The results from the subjective assessment lead to decisions about preventive actions. This can result in situations where patients do not receive the right preventive measures. The high PU incidence of 45% at discharge might be explained by more frequent assessments and careful documentation of PUs in the study context. It may also be explained by selection bias since patients admitted to orthopaedic wards were more likely to be older, to have comorbid conditions and to be more seriously ill, whereas patients initially regarded as having more positive prognosis were submitted to geriatric wards for post-surgery rehabilitation program. One strength, but also a limitation, in the present study was that it was not always the same assessors who assessed the patients, due to the clinical situation. All assessments were however performed independently. The number of patients involved in the study was lower than calculated for the power. We started with 97 but ended with 78 which could potentially reduce validity.

The poor agreement between the assessor’s could affect the preventive measures taken. The difficulty in current clinical practice is to determine if a patient’s skin is affected by reactive hyperaemia or by a Category I PUs. Our results also indicate a need to use great caution when interpreting point prevalence results overall, since reactive hyperaemia can in some instances be misinterpreted as a PU category I and vice versa.

At present, in some countries, assessment of pressure sites is performed using a transparent disc pressed carefully towards the skin, which has been reported to be a more reliable method than the finger-press test.<sup>236</sup> This method was not available at the start of our study. An

objective method to register non-blanching erythema with high precision is desirable. The Dermaspectrometer measured the E-Index, which was demonstrated to offer high precision in discriminating between blanching and non-blanching erythema (Category I PUs) in the sacrum of patients with hip fractures. This methodology needs to be further evaluated, and smaller and clinically more applicable devices for this purpose were developed during the study period.

Since early and precise classification of PUs is a prerequisite for prevention, this is an important part of the nurse's role. This patient safety issue demands an organisation supporting structured and optimal clinical methods to detect and classify PUs. The leadership role of the nurse cannot be overemphasized.<sup>6</sup>

Precision is also of utmost importance in prevalence studies where Category I PUs dominate. Failure in the classification might lead to a higher or lower total prevalence reported than is the actual case.

The Dermaspectrometer was easy to use and proved to deliver precision in the process of discriminating between blanching/non-blanching erythema. It was, however, tested only in the sacrum but since it worked well in this area, it seems probable that it will work in other locations as well. One limitation may be that the patient sample was modest. Several nurses were involved in the assessments, which comes close to clinical practice. All of the nurses were, however, carefully instructed before the start of the study and performed the assessments independently. Another potential problem was that the instrument had a small optical measuring head and if there was another red area near the measuring point, it is not clear whether this may have influenced the results. For this reason several measuring points were used. However, this may also have jeopardised reliability since the optical head can be held in different positions. The possibility to scan a larger area at the same time would have been desirable.

### **Nursing assessment and documentation**

Medical assessment is the evaluation of the patient for the purposes of forming a diagnosis and plan of treatment. This assessment also includes nursing parameters such as identification of the individual needs, preferences and coping abilities of a patient. The information is compiled and documented as an individual care plan.<sup>52</sup> The quality of the documentation can reflect the care delivered, but this is not always the case. The patient record is firstly a source of information aimed at providing continuity of good care of the patient, but it may also serve as a basis for improvement of quality of care by being scrutinized retrospectively. Since prevention of complications from sickness and care is one of the most important parts of the role of nurses, documentation of risk factors and manifest or potential complications, as well as early signs of such, must be documented. Prevention of pressure ulcers is one indicator of quality of care.

To be able to check for potential skin damage during the care episode, a primary status has to be documented. Documentation must also include risk factors and preventive strategies and actions and must be done continually. In the present retrospective study, documentation

was not optimal regarding development of manifest PUs during the care period. Neither was MNS, BMI and prevention documented in all patients. It was also surprising that the documentation of MNS and BMI was less frequent in the elderly patients. The high rate of missing data for elderly patients was indeed a noteworthy. It might also be that many elderly patients are confused upon arrival to hospital, or that they suffer from dementia, which can perhaps explain this sparse documentation.

It was also reported that younger and older patients had more missing data than middle-aged patients. This might be due to the fact that their risk of developing pressure ulcers was regarded as minimal for the younger patients and that it is difficult to assess acute older patients at admission. Differences in documentation of prevalence and BMI were most complete in patients undergoing elective hip replacement surgery. One reason for this may be that the care of acute patients can coincide with a heavy work load for the nurses whereas elective surgery is planned and documents can be prepared beforehand.

A weakness in this and other studies is the poor documentation of prevention. Whether this reflects actual negligence of preventive actions, or that prevention is conducted but not documented is unclear.<sup>317, 318</sup> The nurse has an important role in the documentation of preventive actions taken.

One weakness of this study is the fact that we only reported on data collected on certain days during the study period. In a retrospective study review there is also the risk of underreporting of data. Furthermore, the total number of patients registered as inpatients at the orthopedic wards was recognized but not the number of patients who were admitted on the day of registration. This made it difficult to determine if insufficient registration was due to a high workload because of an increased number of patients admitted. Furthermore, it was difficult to identify the cause of incomplete registration of BMI and MNS due to missing data in the patients' medical records. This study was performed in one single hospital which could reduce generalisability. However, National laws and guidelines regulate documentation standards. Noteworthy is that the present hospital earlier has reported the same or lower PU prevalence than the rest of the country.

### **Patient safety**

Patient safety can only be guaranteed by optimal individualised assessment, care and documentation. If nurses' documentation demonstrates gaps in important areas such as the prevention of pressure ulcers, then the quality of care cannot be guaranteed. It is also important to investigate if prevention taken can actually reduce the prevalence of PUs.<sup>14, 319</sup>



# CONCLUSIONS AND CLINICAL IMPLICATIONS

The prevalence of pressure ulcers in patients with hip fracture remains high. In Southern Europe, the prevalence of PUs was almost half the prevalence reported by Northern Europe. However, the number of PUs increased both in Northern and Southern Europe during the hospital stay. Risk assessment was sparse in A&E units. Both intrinsic and extrinsic risk factors of significant importance for PU development in patients with hip fractures were identified in centres throughout Europe.

Both visual assessment and the finger-press test were unreliable markers for the detection of Category I PUs. The Dermaspectrometer was proven to be a reliable method of classifying pressure ulcers and needs further investigation.

Documentation of risk factors and PUs, as well as continuity of documentation over the care period, was suboptimal for patients undergoing surgery for hip fractures and THR. Most missing data were noted in elderly patients.

## **Patient benefit and generalizability**

PUs cause great suffering and reduce quality of life.<sup>192, 196, 197</sup> They increase costs for the healthcare system in the form of prolonged hospital stay for the patient.<sup>3, 21</sup> PUs can also result in the death of the patient due to infection and sepsis.<sup>20, 191</sup> Pressure ulcers are still common in patients with hip fractures. This is due to immobility and co-morbidities as well as surgery.<sup>147</sup> Studies have shown that the optimal treatment of a displaced medial fracture of the collum is provided by replacing the fractured hip with a prosthetic joint.<sup>268-270</sup> Pressure ulcers are a potential danger because of the risk of transmitting pathogenic bacteria to the area of the prosthesis. This can lead to infection and re-operation (Lindgren 2007). For this and other reasons outlined in this thesis, it is important to pay attention to the risk factors for PUs, to implement strategies to prevent them and to introduce reliable methods for the early detection of Category I PUs. In this process, documentation plays a central role.



# FURTHER RESEARCH

---

Pressure as the primary factor in the development of PUs also requires more thorough studies. Several critical questions at the heart of pressure ulcer research still remain unanswered. These include:

Studies on the differences in the prevalence of pressure ulcers in patients with hip fractures and potential causative factors in Northern versus Southern Europe.

Development and validation of a specific risk assessment instrument for patients with hip fractures.

Studies on the reliability of the new, smaller Deraspectrometer to detect non-blanching erythema (Category I PUs) in alternative body locations.

Studies on factors influencing the adherence to guidelines and how to optimise the documentation in patient records.



# SUMMARY IN SWEDISH.

## POPULÄRVETENSKAPLIG SAMMANFATTNING

Trycksår är ett stort problem inom vården. Prevalensen av trycksår i Sverige ligger mellan 14-17% enligt SKL:s senaste punktprevalensmätning (2011). Det är lika vanligt både inom akutsjukvård som inom kommunal omsorg. Trycksår uppkommer när en person har svårighet att ändra kroppens läge tillräckligt ofta för att avlasta områden som är utsatta för tryck. Idag finns inga säkra metoder för att tidigt identifiera patienter som är i riskzonen för att få trycksår eller för att säkert skilja reaktiv hyperemi från kvarstående rodnad (kategori I, trycksår). Reaktiv hyperemi är kroppens normala svar på att cirkulationen har varit försämrad eller helt avstängd till ett område. Idag används subjektiva metoder för att skilja reaktiv (övergripande) rodnad från trycksår kategori I. Detta sker antingen med ”finger tryck test” där man med hjälp av lätt ett tryck med fingrarna eller med en transparent platta avgör om rodnaden bleknar eller kvarstår. Om hudområdet reagerar med att blekna (blanching) och sedan bli rodnat igen när trycket släpps, klassas det som att cirkulation föreligger. Detta är en subjektiv bedömning som inte ger svar på hur god cirkulationen är. Om området däremot inte bleknar föreligger ett patologiskt tillstånd i vävnaden, trycksår kategori I. Samstämmigheten mellan olika bedömare har i tidigare studier visat sig mindre god. Bristen på samstämmighet vid diagnostiseringen av tidiga trycksår kan medverka till att åtgärder inte vidtas i tid och trycksår av svårare grad utvecklas. Bestående tryckskada kan visa sig först efter flera dagar, vilket ytterligare försvårar säker identifiering. Andra tecken på begynnande trycksår är: temperaturskillnader mot omgivande vävnad, vävnad som är hård eller känns ”svampig” samt smärta, sveda eller stickningar.

Tryck och skjuv förekommer för det mesta samtidigt. En situation då skjuv skulle kunna undvikas är i viktlost tillstånd t.ex. under vatten. Skjuv är det tillstånd då vävnadslager rör sig i motsatt riktning, oftast över benutskott. Skjuv uppkommer när man glider ner i säng och stol eftersom en del av vävnaden blir kvar i ursprungsposition medan övrig vävnad strävar nedåt. Om hudområdet är påverkat av fukt, pga. inkontinens, behövs det mindre tryck och skjuv för att trycksår ska uppkomma. Det är viktigt att räkna med att mikroklimatet – den grad av fukt och värme som är mellan patient och underlag, kan bidra till att öka känsligheten för tryck och skjuv.

Andra riskfaktorer, förutom tryck, skjuv och fukt är olika sjukdomstillstånd med försämrad cirkulation. Även åldern spelar en stor roll i utvecklandet av trycksår. Små (särskilt prematura) barn har inte tillräckligt med subkutan vävnad och deras hudkostym är inte helt färdigutvecklad vilket leder till försämrad tolerans mot tryck och skjuv. Åldrande människor har en försämrad hudstruktur pga. sämre återuppbyggande av vävnadstrukturer, försämrad elasticitet och försämrad nutrition. En gemensam riskfaktor för både barn, ryggmärgsskadade och vid vissa neurologiska sjukdomstillstånd samt för äldre är nedsatt förmåga till lägesändring och minskad förmåga att känna och reagera på tryck.

Hur trycksår egentligen uppkommer är inte helt klarlagt. Men en teori är att trycket uppkommer på hudytan och sedan fortplantar sig ner i vävnaden sk ”top to bottom teorin”. En annan teori är att tryckskadan uppkommer först på djupet och därefter utvecklar sig mot ytan sk ”bottom-up-teorin”. Forskningen på cellnivå kan förhoppningsvis ge oss mer information om orsaker till varför vissa får trycksår och inte andra. Dagens forskning visar bla att celler, speciellt muskelceller, lättare deformeras vid tryck och skjuv vilket i sin tur leder att cellen tänjs ut, går i nekros och dör.

Mer än 80 procent av alla trycksår uppstår över korsben, sittbensknölar, höftbenskammar, hälar och fotknölar. Patienter med höftfraktur är speciellt utsatta för trycksår på grund av svårigheter att röra sig pga. frakturen samt för att patienter med höftfraktur oftast är äldre och har andra bakomliggande sjukdomar. Farmakologisk smärtlindring kan dölja kroppens signaler till behov av lägesändring och göra att patienten inte uppfattar att ett område är i behov av tryckavlastning.

För att öka patientsäkerheten och minska trycksårsfrekvensen är det viktigt att hitta objektiva mätmetoder för att tidigt kunna identifiera tryckutsatta områden. Snabbt insättande av prevention baserat på patientens behov kan minska risken för att trycksår utvecklas. Det medför minskat lidande och bibehållen livskvalitet. Korrekta bedömningar medför också att mobilisering kan tidigareläggas och sjukvårdens resurser kan användas optimalt.

Noll-vision mot uppkomst av trycksår är ett eftersträvarsvärt mål men kanske inte uppnåeligt under alla omständigheter. Patienter som är i ett terminalt sjukdomsskede riskerar att drabbas av trycksår oavsett förebyggande åtgärder och andra aktiviteter för att avlasta tryck. Det relateras till att även huden, som är kroppens största organ, sviktar när andra organ sviktar. Vid livets slut har patienten rätt till att få den omvårdnad som deras tillstånd kräver vilket betyder att personalen måste vara lyhörd för behov eller inte behov av lägesändring, smärstillande, värme, nutrition osv.

## Studie I

Denna studie omfattade patienter med höftfraktur i 6 europeiska länder.

Syftet var att undersöka andelen patienter med trycksår vid ankomst till sjukhuset och vid utskrivning, samt att identifiera vilka patientrelaterade och miljörelaterade riskfaktorer som förekommer vid utveckling av trycksår för patienter med höft fraktur.

Syftet var också att klargöra skillnader i omhändertagande, kirurgisk behandling, förekomst av trycksår (prevalens och incidens) mellan Nord- och Sydeuropa.

Resultatet visade att av 635 patienter med höftfraktur hade 10 % trycksår av alla patienter med höftfraktur hade trycksår när de skrevs in på sjukhuset och 22 % hade det vid utskrivningen. Det var skillnad mellan Nord (26 %) och Syd Europa (16 %). Majoriteten var grad 1 trycksår och ingen hade trycksår grad 4. Det var fler cervikala frakturer i norr medan trokantära frakturer dominerade i söder. Patienterna fick vänta längre på operation i södra Europa och hade oftare sträck behandling än patienterna i norra Europa. I Norra Europa var det vanligare med varma

infusionsvätskor under operationen. Signifikanta riskfaktorer relaterade till trycksår vid utskrivning var: ålder  $\geq 71$  ( $p = .020$ ), lågt vätskeintag ( $p = .005$ ), fuktig hud ( $p = .004$ ) och låg riskbedömningspoäng enligt Braden ( $p = .050$ ) och även låga poäng avseende undergrupperna friktion ( $p = .020$ ), nutrition ( $p = .020$ ), och nedsatt känsel ( $p = .040$ ). Bakgrundssjukdomar som var signifikant relaterade till trycksår vid utskrivning var diabetes ( $p = .005$ ) och lungsjukdom ( $p = .006$ ). Väntetid till operation, längden på operation, värme pre-operativt, typ av anestesi eller frakturtyp visade sig inte innebära ökad risk för trycksår.

En pan-europeisk studie bjuder många möjligheter men också en rad svårigheter. Kontexten för studien skiljer sig, följsamhet till protokollen kan variera. Centra som samtyckt till att delta fick förhinder. Trots detta kunde en del av de slutsatser som drogs i denna studie belysa generella problem kring trycksår för patienter med höftfraktur. Den i andra studier rapporterade lägre prevalensen av trycksår i Sydeuropa kunde verifieras i denna studie. Skälen till detta är svåra att förstå och kräver en speciell studie. I den aktuella studien planerades en rad statistiska analyser. Flera av dessa förkastades av den statistiker som sedermera kom att medverka i dataanalysen. Ytterligare en svårighet var att en av de huvudansvariga för studien professor Gerry Bennett blev svårt sjuk och avled under studiens gång.

## Studie II

Huvudsyftet med denna studie var att undersöka om två olika sjuksköterskor var för sig bedömde hudrodnad i sacrum (korsbenet) på samma sätt och ifall huden var rodnad eller inte. Studien undersökte även om man kunde identifiera några specifika riskfaktorer för trycksårsutveckling för patienter med höftfraktur, på en ortopedklinik.

Huvudfyndet var att finger-tryck test tillsammans med visuell observation inte var tillräckligt säkert vid bedömning om huden var rodnad eller inte. Bedömarna kom i stor utsträckning till olika slutsatser. Det var en signifikant skillnad ( $p = 0.013$ ), en sämre överensstämmelse mellan bedömarna gällande fingertryckstest i jämförelse med okulärbesiktning (dag 1 till dag 5). Analysen visade också att det var större risk för kvarstående hudrodnad från dag 2 till dag 5 ( $p < 0.01$ ).

Fyrtiosju procent av patienterna som deltog i studien hade en riskbedömningspoäng  $\leq 20$  vilket tyder på hög risk för trycksårsutveckling. Fyrtiofyra av de 78 patienter som deltog hade trycksår vid utskrivningen. Ingen av tidigare rapporterade riskfaktorerna som hög ålder, intagningsdiagnos och bakgrundssjukdomar, låg vikt, blodförlust/transfusions behov eller lågt värde på MNS var relaterat till trycksårsutveckling. Anledning till detta kan vara patientmixen. Under studiens gång var det endast de svårast sjuka patienterna som vårdades på ortopedisk vårdavdelning. Patienter som bedömdes kunna återgå till sitt vanliga boende efter vårdtiden hamnade på geriatrisk vårdavdelning och ingick inte i studien. Patientantalet var begränsat vilket kan påverka resultatet. Även om det inte var signifikant så var det fler patienter med inkontinens som hade trycksår. Ingen patient hade trycksår kategori III eller IV. Studiens resultat påvisar svårigheterna att göra samstämmiga bedömningar. Detta kan påverka vilka preventiva åtgärder som sätts in. Svårigheten att med nuvarande klinisk praxis, avgöra om en hudrodnad är en reaktiv (övergående) hyperemi eller ett trycksår kategori I gör också att resultaten av exempelvis prevalensstudier måste tolkas med stor försiktighet.

### Studie III

Syftet med denna studie var att testa om ett digitalt instrument (Dermaspektrometern) kunde användas för verifiering av graden av rodnad i sacrum hos patienter med höftfraktur. Samma patienter som undersöktes i studie II ingick. Det resultat som framkom i studie II kunde därför jämföras med resultaten av mätningarna med Dermaspektrometern. Mätningen genomfördes från dag ett efter operation och i upp till 5 dagar. Mätningen gjordes en gång per dag. Hudområdet över sacrum mättes på 7 förvalda, markerade punkter med det digitala instrumentet. En referenspunkt (kontroll) på den friska höften markerades och registrerades i samband med övriga mätningar för att få ett värde på ett hudområde som inte utsatts för tryck.

För att jämföra om det fanns någon samstämmighet mellan det digitala instrumentets resultat och resultatet från bedömningen av två bedömare som genomförde finger tryck test, som kan kallas ”golden standard”, delades sjuksköterskornas bedömningsresultat in i tre grupper. I den första gruppen bedömdes huden som icke rodnad av båda bedömarna, I den andra gruppen bedömde båda att huden var rodnad och i den tredje gruppen tyckte sjuksköterskorna olika. Resultatet visade en nästan perfekt överensstämmelse – i detta fall att instrumenten registrerade ett högre värde när bedömarna bedömde att huden var rodnad. Samstämmigheten mellan bedömningarna låg mellan 0.82 och 0.96 och visade även att förändringen över tid (från dag 1 till dag 5) var signifikant ( $p < 0.0001$ ). Under samma mätperiod visade referenspunkten ingen signifikant förändring ( $p = 0.32$ ). Detta tyder på att det går att objektivt mäta rodnad hud i sacrum och att instrumentet skulle kunna indikera ett tryckutsatt område även om inte ögat ser en rodnad. Större studier behövs eftersom materialet bestod av 78 patienter som inte kunde följas lika många dagar eftersom det inte går att kvarhålla patienten på avdelning om utskrivning sker tidigare.

### Studie IV

Syftet med denna studie var att genom journalgranskning undersöka sjuksköterskans dokumentation av ortopedpatienters riskbedömning, riskfaktorer och hudbedömning vid inskrivning och under vårdtiden, samt vilka förebyggande åtgärder som dokumenteras när riskbedömning enligt MNS visat risk för trycksår.

Totalt granskades 2,281 patienters journaler. Urvalet av patienter var taget från en dag per månad under fyra år och alla inneliggande just den dagen. Patientmaterialet delades in i fyra grupper – höftfraktur, höftplastik, övriga frakturer och övriga planerade patienter. Gruppen med höftfrakturer och planerade höftproteser granskades mer specifikt med fokus på dokumenterad förekomsten av trycksår vid inskrivning och under vårdtiden (inkl svårighetsgrad), genomförd riskbedömning (enl MNS) dokumenterat BMI värde samt dokumenterade förebyggande åtgärder vid inskrivning och under vårdtiden.

Resultatet visar att dokumentationen avseende riskbedömning enligt MNS och trycksåruppföljning visade stora brister. BMI värdet som kan ge information om undervikt vilket kan innebära risk för trycksår och behov av förebyggande åtgärder, var inte heller optimalt dokumenterat. Resultatet visade att vid akutintag (där patienter med höftfraktur

ingår) hade 46.5% (n=848) av patienterna ett BMI värde dokumenterat och 38.3% (n=697) av patienterna hade ett riskbedömningsvärde enligt MNS. För patienter som skulle genomgå en planerad behandling med höftprotes var det 63.3% (n=292) som hade ett BMI värde och 39.2% (n=180) som var riskbedömda enligt MNS. Detta kan förklaras av att patienter som kommer in för planerad behandling kan skrivas in under kontrollerade förhållanden.

Sjuttioåtta patienter hade trycksår vid inskrivning och de hade totalt 119 trycksår. Under vårdtiden noterades 226 patienter med trycksår och dessa hade 355 trycksår totalt. Alla kategorier finns representerade men förekomst av kategori I och II dominerade. Det som är förvånande är att trycksår av kategori 3 eller 4 inte hade en uppföljande dokumentation i journalen om trycksårsutvecklingen. Under vårdtiden dokumenterades heller inte vilka förebyggande åtgärder som vidtagits även om man kan anta att det skedde vid ett flertal tillfällen. Det framkom även att det fanns en tendens till att BMI och MNS dokumenterades bättre under sommarmånaderna, vilket var förvånande. Fyndet att både BMI och MNS dokumenterades i mindre grad för yngre och äldre är tankeväckande.

Att enbart basera sina resultat på det som är dokumenterat beskriver inte vården som genomförs eftersom man menar att ”det som inte är skrivet inte heller är genomfört”. Det känns också mindre bra att flera omvårdnadsbeslut baseras på det som inte egentligen kanske visar den faktiska vårdtyngden. Detta kan potentiellt påverka besluten om resursallokering.



# ACKNOWLEDGMENTS

---

I wish to express my sincere gratitude to all of you who helped and supported me to make this thesis possible. I am especially indebted to:

*Björn Fossum* – my supervisor, for constant support and for believing in me and my ability to do this work. You shared a wealth of knowledge within the field of science with me. To the last minute you helped me to reach all the way. I will never forget!

*Christina Lindholm* – my co-supervisor, without whose endless support I would not be here and who shared vast wisdom and knowledge in the field of wounds. You also gave me a lot of extra time and energy. You will always be in my mind.

*André Stark* – my co-supervisor, for your constant support, constructive criticism and stimulating discussions.

*Wilhelmina Ekström* – my mentor who was always available and who encouraged and inspired me to think one step further.

Many are those who in one way or another have influenced me to pursue a research career. I want to thank all my colleagues and the patients at the orthopaedic wards. I could not have managed without you. I would also like to thank all of my colleagues on the 7th floor for listening to all of the scientific jargon, complaints about lack of time, assistance with practical daily work and the laughter. I would especially like to thank the following people:

*Jan-Åke Lindgren* – for providing excellent research facilities at Sophiahemmet University College and for believing in my research.

*Lennart Adamsson* – current Chief of the Orthopaedic Department, Karolinska University Hospital, for understanding and believing in my capacity to juggle so many things at the same time.

*Susanne Stålenhag* – current head nurse, friend and colleague for many years. Thank you for listening and giving feedback and support when needed. You'll leave room for others to develop and grow.

*Gunnar Nemeth* – former Chief of the Orthopaedic Department who initially supported my first stumbling steps into science.

*Carl-Göran Eriksson* – former Chief at Danderyd Hospital, for a short period, for showing interest, belief and support in my field of research.

*Regina Wredling* – for your warm heart and support when you had responsibility for several other PhD students at the same time, but always had room for one more.

At the beginning of my research, I was helped by many people. In particular, I would like to thank *Lena Boman* and *Monika Ederström* who were working in the A&E at Karolinska. They helped me start the assessment study and provided a link to the A&E. Thank you for your support and problem solving along the way.

*Lena Svedberg* and *Ante Myrlund* who were Head Nurses in the ward and operating theatre. Nothing was impossible for them, and they managed to give me personal support in taking the measurements.

*Meta Uddnäs* – for months of help with practical data collection and for being so full of ideas, some of which I could not follow from the start.

*Maria Unbeck* – my co-author and friend, and also gifted fellow researcher. You supported me and lent me your ear when everything was tough and “up to my neck”.

*Elisabeth Berg* – for your fantastic support regarding statistics and your patience when you had to explain calculations several times.

Special thanks to my fellow doctoral students at Sophiahemmet University College: *Lena Axelsson*, *Helene Andersson*, *Caroline Löfvenmark*, *Bodil Samuelsson* and *Nina Asplin*. It feels good to know that you were there for me.

*Ann-Britt Wikström* – for keeping all my documents in order and *Lennart Helleday* who fixed computer problems. Nothing was impossible for you two.

*Maria Amnell* – for the interesting discussions about the different aspects of medical and veterinary science. And for being as crazy about cats as I am.

*Håkan Dahlqvist* – for valuable support with the technical issues that arose along the way regarding the methods of digital skin measurement and analysis.

*Kamilla Andersson* – for your wonderful illustrations and solutions to impossible problems.

*Ringvor Hägglöf* – for your brilliant work with the layout.

*Hans Petter Södergaard* - Language control was needed and when it seemed like the darkest night you suddenly was there, full of energy and willingness to help me with all the pages. Thank you for your helping hand. I cannot describe how I feel.

The staff at the Medical Library at Karolinska University Hospital under the leadership of *Marie Källberg*. – for being so friendly and helping me with articles and literature.

The members of the Board of Swedish Tissue Viability Nurses – SSiS. Thank you for understanding when financial reports were not forthcoming and for listening to my research concerns.

To my family: Thank you dear Mom, *Marjatta*, for everything you gave me during my childhood. It is what has made me what I am today. I always feel your comfort and support. To my brother and sister, *Leif* and *Anneli*, thank you for being there and believing in me.

To my lovely children *Anders* and *Annicka*, who now have their own families and who have given me two lovely grandchildren. Thank you for your support and understanding when everything was overwhelming and you didn't see much of me. You are all the joy of my life.

Last, but not least, I would like to thank my husband of so many years, *Bengt*, with whom I hope to spend much more time in future. Thank you for your calm and endless support and encouragement when computer issues were driving me insane. I love you so much.

### Funding

The PEPUS study was funded by EPUAP and the Order of St. John. Study III was funded by "Sven Noréns Gåvofond." Research resources were allocated by Sophiahemmet University College, Stockholm, and the Department of Orthopaedics at Karolinska University Hospital, Stockholm, Sweden.



# REFERENCES

---

1. European Pressure Ulcer Advisory Panel. EPUAP. 2012-02-27; Available from: <http://www.epuap.org>.
2. Reddy M, Gill SS, Rochon PA. Preventing pressure ulcers: a systematic review. *JAMA*. 2006 Aug 23;296(8):974-84.
3. Gethin G, Jordan-O'Brien J, Moore Z. Estimating costs of pressure area management based on a survey of ulcer care in one Irish hospital. *J Wound Care*. 2005 Apr;14(4):162-5.
4. Richardson GM, Gardner S, Frantz RA. Nursing assessment: impact on type and cost of interventions to prevent pressure ulcers. *J Wound Ostomy Continence Nurs*. 1998 Nov;25(6):273-80.
5. Van Den Bos J, Rustagi K, Gray T, Halford M, Ziemkiewicz E, Shreve J. The \$17.1 billion problem: the annual cost of measurable medical errors. *Health Aff (Millwood)*. 2011 Apr;30(4):596-603.
6. Riordan J, Voegeli D. Prevention and treatment of pressure ulcers. *British journal of nursing (Mark Allen Publishing)*. 2009 Nov 12-25;18(20):S20, S2, S4-7.
7. Hietanen H. Pressure ulcer patient's quality of life from a nurse's perspective. In: Romanelli M, editor. *Science and Practice of Pressure Ulcer Management*. London: Springer; 2006.
8. Dealey C. Pressure sores: the result of bad nursing? *British journal of nursing (Mark Allen Publishing)*. 1992 Dec 10-1993 Jan 13;1(15):748.
9. Bennett RG, O'Sullivan J, DeVito EM, Remsburg R. The increasing medical malpractice risk related to pressure ulcers in the United States. *J Am Geriatr Soc*. 2000 Jan;48(1):73-81.
10. Hampton S. Bedsore neglect is a result of reduced education and resources. *British journal of nursing (Mark Allen Publishing)*. 2011 Aug 11-Sep 8;20(15):S27.
11. Demarre L, Vanderwee K, Defloor T, Verhaeghe S, Schoonhoven L, Beeckman D. Pressure ulcers: knowledge and attitude of nurses and nursing assistants in Belgian nursing homes. *Journal of clinical nursing*. 2011 Nov 1.
12. Beeckman D, Schoonhoven L, Verhaeghe S, Vanderwee K. Pressure ulcer prevention, the state of the art: the contribution of Tom Defloor. *Int J Nurs Stud*. 2011 Jul;48(7):787-90.
13. Lyder CH, Ayello EA. *Patient Safety and Quality: An Evidence-Based Handbook for Nurses*. RG H, editor. Rockville: Agency for Healthcare Research and Quality; 2008.
14. Yap TL, Kennerly SM. A nurse-led approach to preventing pressure ulcers. *Rehabil Nurs*. 2011 May-Jun;36(3):106-10.
15. Tucker AL, Edmonson, A.C. Why hospitals don't learn from failure: Organizational and psychological dynamics that inhibit system change *California Management review*. 2003;45(2).
16. Rasmussen. The concept of human error: Is it useful for the design of safe system in health care? In: Vincent C. d, B., editor. *Risk and Safety in Medicine*. London: Elsevier; 1999.
17. Idvall E. *Kvalitetsindikatorer inom omvårdnad*. 5. uppl. ed. sjuksköterskeförening S, editor. Stockholm: Gothia: Svensk sjuksköterskeförening; 2009.
18. Gunningberg L, Lindholm C, Carlsson M, Sjoden PO. The development of pressure ulcers in patients with hip fractures: inadequate nursing documentation is still a problem. *Journal of advanced nursing*. 2000 May;31(5):1155-64.

19. Baharestani MM, Black JM, Carville K, Clark M, Cuddigan JE, Dealey C, et al. Dilemmas in measuring and using pressure ulcer prevalence and incidence: an international consensus. *International wound journal*. 2009 Apr;6(2):97-104.
20. Redelings MD, Lee NE, Sorvillo F. Pressure ulcers: more lethal than we thought? *Advances in skin & wound care*. 2005 Sep;18(7):367-72.
21. Primiano M, Friend M, McClure C, Nardi S, Fix L, Schafer M, et al. Pressure ulcer prevalence and risk factors during prolonged surgical procedures. *AORN J*. 2011 Dec;94(6):555-66.
22. Duncan KD. Preventing pressure ulcers: the goal is zero. *Jt Comm J Qual Patient Saf*. 2007 Oct;33(10):605-10.
23. Oriol MD. Crew resource management: applications in healthcare organizations. *J Nurs Adm*. 2006 Sep;36(9):402-6.
24. Halfens RJ, Bours GJ, Van Ast W. Relevance of the diagnosis 'stage 1 pressure ulcer': an empirical study of the clinical course of stage 1 ulcers in acute care and long-term care hospital populations. *Journal of clinical nursing*. 2001 Nov;10(6):748-57.
25. NPUAP. Pressure Ulcer Prevention & Treatment Clinical Practice Guideline. National Pressure Ulcer Advisory Panel & European Pressure Ulcer Advisory Panel; 2009; Available from: [www.npuap.org](http://www.npuap.org).
26. Lyder CH. Pressure ulcer prevention and management. *Annu Rev Nurs Res*. 2002;20:35-61.
27. Reddy M. Pressure ulcers. *Clin Evid (Online)*. 2011;2011.
28. Rich SE, Margolis D, Shardell M, Hawkes WG, Miller RR, Amr S, et al. Frequent manual repositioning and incidence of pressure ulcers among bed-bound elderly hip fracture patients. *Wound Repair Regen*. 2011 Jan;19(1):10-8.
29. Baumgarten M, Shardell M, Rich S. Methodological issues in studies of the effectiveness of pressure ulcer prevention interventions. *Advances in skin & wound care*. 2009 Apr;22(4):180-8; quiz 9-90.
30. Vanderwee K, Defloor T, Beeckman D, Demarre L, Verhaeghe S, Van Durme T, et al. Assessing the adequacy of pressure ulcer prevention in hospitals: a nationwide prevalence survey. *Qual Saf Health Care*. 2011 Jan 5.
31. Orsted HL, Ohura T, Harding K. Pressure ulcer prevention: pressure, shear, friction, and microclimate in context. London2010.
32. Romanelli M. Science and practice of pressure ulcer management. London: Springer; 2006.
33. Bliss M. Aetiology of pressure sores. *Rev Clin Gerontol*. 1993;3:379-97
34. Baumgarten M, Margolis DJ, Localio AR, Kagan SH, Lowe RA, Kinosian B, et al. Pressure ulcers among elderly patients early in the hospital stay. *J Gerontol A Biol Sci Med Sci*. 2006 Jul;61(7):749-54.
35. Thomas DR, Goode PS, Tarquine PH, Allman RM. Hospital-acquired pressure ulcers and risk of death. *J Am Geriatr Soc*. 1996 Dec;44(12):1435-40.
36. Kosiak M. Prevention and rehabilitation of pressure ulcers. *Decubitus*. 1991 May;4(2):60-2, 4, 6 passim.
37. Sharp CA, McLaws ML. Estimating the risk of pressure ulcer development: is it truly evidence based? *International wound journal*. 2006 Dec;3(4):344-53.
38. Soderqvist A, Miedel R, Ponzer S, Tidermark J. The influence of cognitive function on outcome after a hip fracture. *J Bone Joint Surg Am*. 2006 Oct;88(10):2115-23.
39. Nixon J, Brown J, McElvenny D, Mason S, Bond S. Prognostic factors associated with pressure sore development in the immediate post-operative period. *Int J Nurs Stud*. 2000 Aug;37(4):279-89.
40. Vanderwee K, Grypdonck M, De Bacquer D, Defloor T. The identification of older nursing home residents vulnerable for deterioration of grade 1 pressure ulcers. *Journal of clinical nursing*. 2009 Nov;18(21):3050-8.

41. Houwing R, Rozendaal M, Wouters-Wesseling W, Buskens E, Keller P, Haalboom J. Pressure ulcer risk in hip fracture patients. *Acta Orthop Scand*. 2004 Aug;75(4):390-3.
42. Gunningberg L. Risk, prevalence and prevention of pressure ulcers in three Swedish healthcare settings. *J Wound Care*. 2004 Jul;13(7):286-90.
43. Lindholm C, Sterner E, Romanelli M, Pina E, Torra y Bou J, Hietanen H, et al. Hip fracture and pressure ulcers - the Pan-European Pressure Ulcer Study - intrinsic and extrinsic risk factors. *International wound journal*. 2008 Jun;5(2):315-28.
44. Hommel A, Bjorkelund KB, Thorngren KG, Ulander K. Nutritional status among patients with hip fracture in relation to pressure ulcers. *Clin Nutr*. 2007 Oct;26(5):589-96.
45. Kosiak M. Etiology and pathology of ischemic ulcers. *Arch Phys Med Rehabil*. 1959 Feb;40(2):62-9.
46. Bliss MR. Hyperaemia. *Journal of tissue viability*. 1998 Oct;8(4):4-13.
47. Nixon. J, Cranny. G, Bond. S. Skin alterations of intact skin and risk factors associated with pressure ulcer development in surgical patients: a cohort study. *Int J Nurs Stud*. 2007;July(44(5)):655-63.
48. National Pressure Ulcer Advisory Panel. NPUAP. 2012-02-27; Available from: [www.npuap.org](http://www.npuap.org).
49. Lyder CH. Pressure ulcers: identification and care. *Kans Nurse*. 2007 May;82(5):10-1.
50. Kosiak M, Kubicek WG, Olson M, Danz JN, Kottke FJ. Evaluation of pressure as a factor in the production of ischial ulcers. *Arch Phys Med Rehabil*. 1958 Oct;39(10):623-9.
51. Takahashi PY, Kiemele LJ, Jones JP, Jr. Wound care for elderly patients: advances and clinical applications for practicing physicians. *Mayo Clin Proc*. 2004 Feb;79(2):260-7.
52. Medical Dictionary. 2012-02-27; Available from: <http://medical-dictionary.thefreedictionary.com/nursing+assessment>.
53. Collier M. Blanching and non-blanching hyperaemia. *J Wound Care*. 1999 Feb;8(2):63-4.
54. Klabunde RE. Reactive Hyperemia. 2012-02-27; Available from: <http://www.cvphysiology.com/Blood%20Flow/BF006.htm>.
55. Lewis T, Grant R. Observations upon reactive hyperaemia in man *Heart*. 1925;12(73):17.
56. Grey JE. Cellulitis associated with wounds. *J Wound Care*. 1998 Jul;7(7):338-9.
57. Lyder CH. Pressure ulcer prevention and management. *JAMA*. 2003 Jan 8;289(2):223-6.
58. Thorngren KG, Hommel A, Norrman PO, Thorngren J, Wingstrand H. Epidemiology of femoral neck fractures. *Injury*. 2002 Dec;33 Suppl 3:C1-7.
59. Russo CA, Elixhauser A. Hospitalizations Related to Pressure Sores, 2003: Statistical Brief #3. 2006 Feb.
60. Haleem S, Heinert G, Parker MJ. Pressure sores and hip fractures. *Injury*. 2008 Feb;39(2):219-23.
61. Whittington K, Patrick M, Roberts JL. A national study of pressure ulcer prevalence and incidence in acute care hospitals. *J Wound Ostomy Continence Nurs*. 2000 Jul;27(4):209-15.
62. Baumgarten M, Margolis D, Berlin JA, Strom BL, Garino J, Kagan SH, et al. Risk factors for pressure ulcers among elderly hip fracture patients. *Wound Repair Regen*. 2003 Mar-Apr;11(2):96-103.
63. Allman RM, Goode PS, Patrick MM, Burst N, Bartolucci AA. Pressure ulcer risk factors among hospitalized patients with activity limitation. *JAMA*. 1995 Mar 15;273(11):865-70.

64. Dolk T. Hip fractures--treatment and early complications. *Ups J Med Sci.* 1989;94(2):195-207.
65. Gunningberg L, Lindholm C, Carlsson M, Sjoden PO. Reduced incidence of pressure ulcers in patients with hip fractures: a 2-year follow-up of quality indicators. *Int J Qual Health Care.* 2001 Oct;13(5):399-407.
66. Jensen TT, Juncker Y. Pressure sores common after hip operations. *Acta Orthop Scand.* 1987 Jun;58(3):209-11.
67. Campbell AJ. Femoral neck fractures in elderly women: a prospective study. *Age Ageing.* 1976 May;5(2):102-9.
68. Versluisen M. Pressure sores in elderly patients. The epidemiology related to hip operations. *J Bone Joint Surg Br.* 1985 Jan;67(1):10-3.
69. Versluisen M. How elderly patients with femoral fracture develop pressure sores in hospital. *Br Med J (Clin Res Ed).* 1986 May 17;292(6531):1311-3.
70. Lindgren M, Unosson, M., Ek, A-C. Pressure sore prevalence within a public health services area. *International Journal of Nursing practice.* 2000;6:333-7.
71. Lahmann NA, Kottner J, Dassen T, Tannen A. Higher pressure ulcer risk on intensive care? - Comparison between general wards and intensive care units. *Journal of clinical nursing.* 2011 Mar 9.
72. Vanderwee K, Clark M, Dealey C, Gunningberg L, Defloor T. Pressure ulcer prevalence in Europe: a pilot study. *J Eval Clin Pract.* 2007 Apr;13(2):227-35.
73. Nixon J, Cranny G, Bond S. Skin alterations of intact skin and risk factors associated with pressure ulcer development in surgical patients: A cohort study. *Int J Nurs Stud.* 2006 Apr 24.
74. Schoonhoven L, Bousema MT, Buskens E. The prevalence and incidence of pressure ulcers in hospitalised patients in the Netherlands: a prospective inception cohort study. *Int J Nurs Stud.* 2007 Aug;44(6):927-35.
75. Schoonhoven L, Defloor T, Grypdonck MH. Incidence of pressure ulcers due to surgery. *Journal of clinical nursing.* 2002 Jul;11(4):479-87.
76. Gunningberg L. EPUAP pressure ulcer prevalence survey in Sweden: a two-year follow-up of quality indicators. *J Wound Ostomy Continence Nurs.* 2006 May-Jun;33(3):258-66.
77. Schuurman JP, Schoonhoven L, Keller BP, van Ramshorst B. Do pressure ulcers influence length of hospital stay in surgical cardiothoracic patients? A prospective evaluation. *Journal of clinical nursing.* 2009 Feb 5.
78. Lahmann NA, Tannen A, Dassen T, Kottner J. Friction and shear highly associated with pressure ulcers of residents in long-term care - Classification Tree Analysis (CHAID) of Braden items. *J Eval Clin Pract.* 2011 Feb;17(1):168-73.
79. Davis CM, Caseby NG. Prevalence and incidence studies of pressure ulcers in two long-term care facilities in Canada. *Ostomy Wound Manage.* 2001 Nov;47(11):28-34.
80. Landi F, Onder G, Russo A, Bernabei R. Pressure ulcer and mortality in frail elderly people living in community. *Arch Gerontol Geriatr.* 2007;44 Suppl 1:217-23.
81. Capon A, Pavoni N, Mastromattei A, Di Lallo D. Pressure ulcer risk in long-term units: prevalence and associated factors. *Journal of advanced nursing.* 2007 May;58(3):263-72.
82. Oot-Giromini BA. Pressure ulcer prevalence, incidence and associated risk factors in the community. *Decubitus.* 1993 Sep;6(5):24-32.
83. Sterner E, Lindholm C, Berg E, Stark A, Fossum B. Category I pressure ulcers: how reliable is clinical assessment? *Orthop Nurs.* 2011 May-Jun;30(3):194-205; quiz 6-7.
84. Salzberg CA, Byrne DW, Cayten CG, van Niewerburgh P, Murphy JG, Viehbeck M. A new pressure ulcer risk assessment scale for individuals with spinal cord injury. *Am J Phys Med Rehabil.* 1996 Mar-Apr;75(2):96-104.

85. Rodriguez GP, Garber SL. Prospective study of pressure ulcer risk in spinal cord injury patients. *Paraplegia*. 1994 Mar;32(3):150-8.
86. Krause JS. Skin sores after spinal cord injury: relationship to life adjustment. *Spinal Cord*. 1998 Jan;36(1):51-6.
87. Salzberg CA, Byrne DW, Cayten CG, Kabir R, van Niewerburgh P, Viehbeck M, et al. Predicting and preventing pressure ulcers in adults with paralysis. *Adv Wound Care*. 1998 Sep;11(5):237-46.
88. Byrne DW, Salzberg CA. Major risk factors for pressure ulcers in the spinal cord disabled: a literature review. *Spinal Cord*. 1996 May;34(5):255-63.
89. Holtz A, Levi R, Lyons L. Ryggmargsskador : behandling och rehabilitering. Lund: Studentlitteratur; 2006.
90. Curley MA, Razmus IS, Roberts KE, Wypij D. Predicting pressure ulcer risk in pediatric patients: the Braden Q Scale. *Nurs Res*. 2003 Jan-Feb;52(1):22-33.
91. Hoshowsky VM, Schramm CA. Intraoperative pressure sore prevention: an analysis of bedding materials. *Res Nurs Health*. 1994 Oct;17(5):333-9.
92. Walton-Geer PS. Prevention of pressure ulcers in the surgical patient. *AORN J*. 2009 Mar;89(3):538-48; quiz 49-51.
93. Schoonhoven L, Defloor T, van der Tweel I, Buskens E, Grypdonck MH. Risk indicators for pressure ulcers during surgery. *Appl Nurs Res*. 2002 Aug;15(3):163-73.
94. Nixon J, Cranny G, Bond S. Pathology, diagnosis, and classification of pressure ulcers: comparing clinical and imaging techniques. *Wound Repair Regen*. 2005 Jul-Aug;13(4):365-72.
95. Sprigle S, Linden M, Riordan B. Analysis of localized erythema using clinical indicators and spectroscopy. *Ostomy Wound Manage*. 2003 Mar;49(3):42-52.
96. Gefen A. Deep tissue injury from a bioengineering point of view. *Ostomy Wound Manage*. 2009 Apr;55(4):26-36.
97. Sveriges Kommuner och Landsting. 2012-02-27; Available from: [www.skl.se](http://www.skl.se).
98. Thomas DR. Are all pressure ulcers avoidable? *J Am Med Dir Assoc*. 2003 Mar-Apr;4(2 Suppl):S43-8.
99. Beldon P. Skin changes at life's end: SCALE ulcer or pressure ulcer? *Br J Community Nurs*. 2011 Oct;16(10):491-4.
100. Gaymar. Scale document. 2012-02-27; Available from: <http://www.gaymar.org>.
101. Baumgarten M, Margolis DJ, Localio AR, Kagan SH, Lowe RA, Kinoshian B, et al. Extrinsic risk factors for pressure ulcers early in the hospital stay: a nested case-control study. *J Gerontol A Biol Sci Med Sci*. 2008 Apr;63(4):408-13.
102. Collier M, Moore Z. Etiology and risk factors. In: Romanelli M, editor. *Science and Practice of Pressure Ulcer Management*. London: Springer; 2006. p. 27-35.
103. Linder-Ganz E, Shabshin N, Itzhak Y, Gefen A. Assessment of mechanical conditions in sub-dermal tissues during sitting: a combined experimental-MRI and finite element approach. *J Biomech*. 2007;40(7):1443-54.
104. Extton-Smith AN, Sherwin RW. The prevention of pressure sores. Significance of spontaneous bodily movements. *Lancet*. 1961 Nov 18;2(7212):1124-6.
105. Lindgren M, Unosson M, Fredrikson M, Ek AC. Immobility--a major risk factor for development of pressure ulcers among adult hospitalized patients: a prospective study. *Scand J Caring Sci*. 2004 Mar;18(1):57-64.
106. Gefen A. How much time does it take to get a pressure ulcer? Integrated evidence from human, animal, and in vitro studies. *Ostomy Wound Manage*. 2008 Oct;54(10):26-8, 30-5.
107. Bader DL, White SH. The viability of soft tissues in elderly subjects undergoing hip surgery. *Age Ageing*. 1998 Mar;27(2):217-21.
108. Linder-Ganz E, Gefen A. The effects of pressure and shear on capillary closure in the microstructure of skeletal muscles. *Ann Biomed Eng*. 2007 Dec;35(12):2095-107.

109. Bouten CV, Oomens CW, Baaijens FP, Bader DL. The etiology of pressure ulcers: skin deep or muscle bound? *Arch Phys Med Rehabil.* 2003 Apr;84(4):616-9.
110. Enoch S, Grey JE, Harding KG. ABC of wound healing. Non-surgical and drug treatments. *BMJ.* 2006 Apr 15;332(7546):900-3.
111. Sprigle S, Linden M, McKenna D, Davis K, Riordan B. Clinical skin temperature measurement to predict incipient pressure ulcers. *Advances in skin & wound care.* 2001 May-Jun;14(3):133-7.
112. Bader DL. The recovery characteristics of soft tissues following repeated loading. *J Rehabil Res Dev.* 1990 Spring;27(2):141-50.
113. Lindgren M, Malmqvist LA, Sjoberg F, Ek AC. Altered skin blood perfusion in areas with non blanchable erythema: an explorative study. *International wound journal.* 2006 Sep;3(3):215-23.
114. Shea JD. Pressure sores: classification and management. *Clin Orthop Relat Res.* 1975 Oct(112):89-100.
115. Kosiak M. Etiology of decubitus ulcers. *Arch Phys Med Rehabil.* 1961 Jan;42:19-29.
116. Witkowski JA, Parish LC. The decubitus ulcer: skin failure and destructive behavior. *Int J Dermatol.* 2000 Dec;39(12):894-6.
117. Landis E. Micro-injection studies of capillary blood pressure in human skin. *Heart.* 1930;15:209-28.
118. Bader D, Oomens C. Recent advances in pressure ulcer research. In: Romanelli M, editor. *Science and Practice of Pressure Ulcer Management.* London: Springer; 2006. p. 11-26.
119. Linder-Ganz E, Engelberg S, Scheinowitz M, Gefen A. Pressure-time cell death threshold for albino rat skeletal muscles as related to pressure sore biomechanics. *J Biomech.* 2006;39(14):2725-32.
120. Gefen A. Reswick and Rogers pressure-time curve for pressure ulcer risk. Part 1. *Nurs Stand.* 2009 Jul 15-21;23(45):64, 6, 8 passim.
121. Gefen A. Reswick and Rogers pressure-time curve for pressure ulcer risk. Part 2. *Nurs Stand.* 2009 Jul 22-28;23(46):40-4.
122. Reger S, Ranganathan, VK., Orsted, HL., Ohura, T., Gefen, A. Shear and friction in context. In: Orsted H, Ohura, T., Harding, K., editor. *Pressure ulcer prevention - pressure, shear, friction and microclimate in context.* London 2010. p. 11-8.
123. Gefen A. The biomechanics of sitting-acquired pressure ulcers in patients with spinal cord injury or lesions. *International wound journal.* 2007 Sep;4(3):222-31.
124. Kobara K, Eguchi A, Watanabe S, Shinkoda K. The influence of the distance between the backrest of a chair and the position of the pelvis on the maximum pressure on the ischium and estimated shear force. *Disabil Rehabil Assist Technol.* 2008 Sep;3(5):285-91.
125. Defloor T, De Schuijmer JD. Preventing pressure ulcers: an evaluation of four operating-table mattresses. *Appl Nurs Res.* 2000 Aug;13(3):134-41.
126. Dinsdale SM. Decubitus ulcers in swine: light and electron microscopy study of pathogenesis. *Arch Phys Med Rehabil.* 1973 Feb;54(2):51-6 passim.
127. Dinsdale SM. Decubitus ulcers: role of pressure and friction in causation. *Arch Phys Med Rehabil.* 1974 Apr;55(4):147-52.
128. Clark M, Romanelli, M., Reger, SI., Ranganathan, HL., Black, J., Dealey, C. Microclimate in context. In: Orsted HL, Ohura, T., Harding, K., editor. *Pressure ulcer prevention - pressure, shear, friction and microclimate in context.* London 2010. p. 19-25.
129. Bates-Jensen BM, McCreath HE, Kono A, Apeles NC, Alessi C. Subepidermal moisture predic130. Bale S, Cameron, J., Meaume, S. Skin Care. In: Romanelli M, editor. *Science and practise of pressure ulcer management: Springer; 2006. p. 75-84.*

131. Bale S, Tebble N, Jones V, Price P. The benefits of implementing a new skin care protocol in nursing homes. *Journal of tissue viability*. 2004 Apr;14(2):44-50.
132. Roaf R. The causation and prevention of bed sores. *Journal of tissue viability*. 2006 May;16(2):6-8.
133. Mayrovitz HN, Sims N. Biophysical effects of water and synthetic urine on skin. *Advances in skin & wound care*. 2001 Nov-Dec;14(6):302-8.
134. Ek AC, Gustavsson G, Lewis DH. Skin blood flow in relation to external pressure and temperature in the supine position on a standard hospital mattress. *Scand J Rehabil Med*. 1987;19(3):121-6.
135. Bergstrom N, Braden, B. A prospective study of pressure sore risk among institutionalized elderly. *J Am Geriatr Soc*. 1992 Aug;40(8):747-58.
136. Fisher SV, Szymke TE, Apte SY, Kosiak M. Wheelchair cushion effect on skin temperature. *Arch Phys Med Rehabil*. 1978 Feb;59(2):68-72.
137. Brienza DM, Geyer MJ. Using support surfaces to manage tissue integrity. *Advances in skin & wound care*. 2005 Apr;18(3):151-7.
138. Scott EM, Leaper DJ, Clark M, Kelly PJ. Effects of warming therapy on pressure ulcers--a randomized trial. *AORN J*. 2001 May;73(5):921-7, 9-33, 36-8.
139. Gefen A. How do microclimate factors affect the risk for superficial pressure ulcers: a mathematical modeling study. *Journal of tissue viability*. 2011 Aug;20(3):81-8.
140. Rorsman H, Björnberg A, Vahlquist A. *Dermatologi, Venerologi*. Stockholm: Studentlitteratur; 2007.
141. Hampton S, Collins F. *Tissue viability : the prevention, treatment, and management of wounds*. London ; Philadelphia: Whurr Publishers; 2004.
142. Holowatz LA, Thompson-Torgerson C, Kenney WL. Aging and the control of human skin blood flow. *Front Biosci*. 2010;15:718-39.
143. Fore J. A review of skin and the effects of aging on skin structure and function. *Ostomy Wound Manage*. 2006 Sep;52(9):24-35; quiz 6-7.
144. Taylor RP, Polliack AA, Bader DL. The analysis of metabolites in human sweat: analytical methods and potential application to investigation of pressure ischaemia of soft tissues. *Ann Clin Biochem*. 1994 Jan;31 ( Pt 1):18-24.
145. Knight SL, Taylor RP, Polliack AA, Bader DL. Establishing predictive indicators for the status of loaded soft tissues. *J Appl Physiol*. 2001 Jun;90(6):2231-7.
146. Sharp CA, McLaws ML. A discourse on pressure ulcer physiology: the implications of repositioning and staging 2005; Oct.
147. Landi F, Russo A, Danese P, Liperoti R, Barillaro C, Bernabei R, et al. Anemia status, hemoglobin concentration, and mortality in nursing home older residents. *J Am Med Dir Assoc*. 2007 Jun;8(5):322-7.
148. Bates-Jensen BM, Guihan M, Garber SL, Chin AS, Burns SP. Characteristics of recurrent pressure ulcers in veterans with spinal cord injury. *J Spinal Cord Med*. 2009;32(1):34-42.
149. Meaume S, Faucher N. Heel pressure ulcers on the increase? Epidemiological change or ineffective prevention strategies? *Journal of tissue viability*. 2008 Feb;17(1):30-3.
150. Bliss MR. Pressure injuries: causes and prevention. *Hosp Med*. 1998 Nov;59(11):841-4.
151. Anders J, Heinemann A, Leffmann C, Leutenegger M, Profener F, von Renteln-Kruse W. Decubitus ulcers: pathophysiology and primary prevention. *Dtsch Arztebl Int*. 2010 May;107(21):371-81; quiz 82.
152. Ek AC, Unosson M, Larsson J, Von Schenck H, Bjurulf P. The development and healing of pressure sores related to the nutritional state. *Clin Nutr*. 1991 Oct;10(5):245-50.
153. Gunningberg L, Persson, C., Åkerfeldt, T., Stridsberg, M., Leo Swenne, C. Pre- and postoperative nutritional status and predictors for surgical-wound infections in elective orthopaedic and thoracic patients. *The European e-journal of Clinical Nutrition and metabolism*. 2008;3:e93-e101.

154. Rypkema G, Adang E, Dicke H, Naber T, de Swart B, Disselhorst L, et al. Cost-effectiveness of an interdisciplinary intervention in geriatric inpatients to prevent malnutrition. *J Nutr Health Aging*. 2004;8(2):122-7.
155. Reddy M, Keast D, Fowler E, Sibbald RG. Pain in pressure ulcers. *Ostomy Wound Manage*. 2003 Apr;49(4 Suppl):30-5.
156. White JJ, Khan WS, Smitham PJ. Perioperative implications of surgery in elderly patients with hip fractures: an evidence-based review. *J Perioper Pract*. 2011 Jun;21(6):192-7.
157. Stockholms läns landsting. Regionalt vårdprogram, Trycksår prevention och behandling: In Swedish. 2012-02-27; Available from: <http://www.vardsamordning.sll.se/sv/TUFF-satsning/Material/Trycksar---prevention-och-behandling/>.
158. Coleridge Smith PD. Oxygen, oxygen-free radicals and reperfusion injury. In: editors. . Fourth edition. Malvern: HMP Communication, 2007. . In: Krasner DL, Rodehaver GT, Sibbald RG, editors. *Chronic Wound Care: A clinical source book for healthcare professionals*. 4th ed: Malvern:HMP Communication; 2007.
159. Jiang LP, Tu Q, Wang Y, Zhang E. Ischemia-reperfusion injury-induced histological changes affecting early stage pressure ulcer development in a rat model. *Ostomy Wound Manage*. 2011 Feb;57(2):55-60.
160. Sundin BM, Hussein MA, Glasofer S, El-Falaky MH, Abdel-Aleem SM, Sachse RE, et al. The role of allopurinol and deferoxamine in preventing pressure ulcers in pigs. *Plast Reconstr Surg*. 2000 Apr;105(4):1408-21.
161. Ritter L, Funk J, Schenkel L, Tipton A, Downey K, Wilson J, et al. Inflammatory and hemodynamic changes in the cerebral microcirculation of aged rats after global cerebral ischemia and reperfusion. *Microcirculation*. 2008 May;15(4):297-310.
162. Mustoe T. Understanding chronic wounds: a unifying hypothesis on their pathogenesis and implications for therapy. *Am J Surg*. 2004 May;187(5A):65S-70S.
163. Papanantonio CT, Wallop JM, Kolodner KB. Sacral ulcers following cardiac surgery: incidence and risks. *Adv Wound Care*. 1994 Mar;7(2):24-36.
164. Berlowitz DR, Wilking SV. Risk factors for pressure sores. A comparison of cross-sectional and cohort-derived data. *J Am Geriatr Soc*. 1989 Nov;37(11):1043-50.
165. Remaley DT, Jaebon T. Pressure ulcers in orthopaedics. *J Am Acad Orthop Surg*. 2010 Sep;18(9):568-75.
166. Soderqvist A, Ponzer S, Tidermark J. Cognitive function and pressure ulcers in hip fracture patients. *Scand J Caring Sci*. 2007 Mar;21(1):79-83.
167. Mecocci P, von Strauss E, Cherubini A, Ercolani S, Mariani E, Senin U, et al. Cognitive impairment is the major risk factor for development of geriatric syndromes during hospitalization: results from the GIFA study. *Dement Geriatr Cogn Disord*. 2005;20(4):262-9.
168. Johnell O, Kanis JA. An estimate of the worldwide prevalence, mortality and disability associated with hip fracture. *Osteoporos Int*. 2004 Nov;15(11):897-902.
169. Wong V. Skin blood flow response to 2-hour repositioning in long-term care residents: a pilot study. *J Wound Ostomy Continence Nurs*. 2011 Sep-Oct;38(5):529-37.
170. Defloor T, De Bacquer D, Grypdonck MH. The effect of various combinations of turning and pressure reducing devices on the incidence of pressure ulcers. *Int J Nurs Stud*. 2005 Jan;42(1):37-46.
171. Oertwich PA, Kindschuh AM, Bergstrom N. The effects of small shifts in body weight on blood flow and interface pressure. *Res Nurs Health*. 1995 Dec;18(6):481-8.
172. de Laat E, Schoonhoven L, Grypdonck M, Verbeek A, de Graaf R, Pickkers P, et al. Early postoperative 30 degrees lateral positioning after coronary artery surgery: influence on cardiac output. *Journal of clinical nursing*. 2007 Apr;16(4):654-61.

173. Moore Z, Cowman S, Conroy RM. A randomised controlled clinical trial of repositioning, using the 30 degrees tilt, for the prevention of pressure ulcers. *Journal of clinical nursing*. 2011 Sep;20(17-18):2633-44.
174. Takahashi M, Black, J., Dealey, C., Gefen, A. Pressure in context. In: Orsted HL, Ohura, T.,Harding, K., editor. *Pressure ulcer prevention - pressure, shear, friction and microclimate in context, a consensus document*. London 2010. p. 2-10.
175. Baumgarten M, Margolis D, Orwig D, Hawkes W, Rich S, Langenberg P, et al. Use of Pressure-Redistributing Support Surfaces Among Elderly Hip Fracture Patients Across the Continuum of Care: Adherence to Pressure Ulcer Prevention Guidelines. *Gerontologist*. 2009 Jul 8.
176. Nanjo Y, Nakagami G, Kaitani T, Naito A, Takehara K, Lijuan J, et al. Relationship Between Morphological Characteristics and Etiology of Pressure Ulcers in Intensive Care Unit Patients. *J Wound Ostomy Continence Nurs*. 2011 July/August;38(4):404-12.
177. Milne AC, Potter J, Vivanti A, Avenell A. Protein and energy supplementation in elderly people at risk from malnutrition. *Cochrane Database Syst Rev*. 2009(2):CD003288.
178. Beck AM, Balknas UN, Furst P, Hasunen K, Jones L, Keller U, et al. Food and nutritional care in hospitals: how to prevent undernutrition--report and guidelines from the Council of Europe. *Clin Nutr*. 2001 Oct;20(5):455-60.
179. Ödlund Olin A, Karlsson M, Lönnberg H. Regionalt vårdprogram Nutrition med inriktning undernäring. Medicinskt programarbete: Stockholms läns landsting; 2005.
180. Miyanishi K, Jingushi S, Torisu T. Mortality after hip fracture in Japan: the role of nutritional status. *J Orthop Surg (Hong Kong)*. 2010 Dec;18(3):265-70.
181. Hedstrom M, Ljungqvist O, Cederholm T. Metabolism and catabolism in hip fracture patients: nutritional and anabolic intervention--a review. *Acta Orthop*. 2006 Oct;77(5):741-7.
182. Beck AM, Ovesen L, Osler M. The 'Mini Nutritional Assessment' (MNA) and the 'Determine Your Nutritional Health' Checklist (NSI Checklist) as predictors of morbidity and mortality in an elderly Danish population. *Br J Nutr*. 1999 Jan;81(1):31-6.
183. Bauer JM, Vogl T, Wicklein S, Trogner J, Muhlberg W, Sieber CC. Comparison of the Mini Nutritional Assessment, Subjective Global Assessment, and Nutritional Risk Screening (NRS 2002) for nutritional screening and assessment in geriatric hospital patients. *Z Gerontol Geriatr*. 2005 Oct;38(5):322-7.
184. Kuhn BA, Coulter SJ. Balancing the pressure ulcer cost and quality equation. *Nurs Econ*. 1992 Sep-Oct;10(5):353-9.
185. Hommel A, Ulander K, Bjorkelund KB, Norrman PO, Wingstrand H, Thorngren KG. Influence of optimised treatment of people with hip fracture on time to operation, length of hospital stay, reoperations and mortality within 1 year. *Injury*. 2008 Oct;39(10):1164-74.
186. Ozturk A, Ozkan Y, Akgoz S, Yalcyn N, Ozdemir RM, Aykut S. The risk factors for mortality in elderly patients with hip fractures: postoperative one-year results. *Singapore Med J*. 2010 Feb;51(2):137-43.
187. Lindholm C, Bergsten A, Berglund E. Chronic wounds and nursing care. *J Wound Care*. 1999 Jan;8(1):5-10.
188. Allman RM. Pressure ulcers among the elderly. *N Engl J Med*. 1989 Mar 30;320(13):850-3.
189. Evans JM, Andrews KL, Chutka DS, Fleming KC, Garness SL. Pressure ulcers: prevention and management. *Mayo Clin Proc*. 1995 Aug;70(8):789-99.

190. Staas WE, Jr., Cioschi HM. Pressure sores--a multifaceted approach to prevention and treatment. *West J Med.* 1991 May;154(5):539-44.
191. Allman RM, Laprade CA, Noel LB, Walker JM, Moorer CA, Dear MR, et al. Pressure sores among hospitalized patients. *Ann Intern Med.* 1986 Sep;105(3):337-42.
192. Spilsbury K, Nelson A, Cullum N, Iglesias C, Nixon J, Mason S. Pressure ulcers and their treatment and effects on quality of life: hospital inpatient perspectives. *Journal of advanced nursing.* 2007 Mar;57(5):494-504.
193. Fox C. Living with a pressure ulcer: a descriptive study of patients' experiences. *Br J Community Nurs.* 2002 Jun;7(6 Suppl):10, 2, 4, 6, 20, 2.
194. Gorecki C, Brown JM, Nelson EA, Briggs M, Schoonhoven L, Dealey C, et al. Impact of pressure ulcers on quality of life in older patients: a systematic review. *J Am Geriatr Soc.* 2009 Jul;57(7):1175-83.
195. Jakobsson U, Hallberg IR, Westergren A. Exploring determinants for quality of life among older people in pain and in need of help for daily living. *Journal of clinical nursing.* 2007 Mar;16(3A):95-104.
196. Kayser-Jones JS, Beard RL, Sharpp TJ. Case study: dying with a stage IV pressure ulcer. *Am J Nurs.* 2009 Jan;109(1):40-8; quiz 9.
197. Hopkins A, Dealey C, Bale S, Defloor T, Worboys F. Patient stories of living with a pressure ulcer. *Journal of advanced nursing.* 2006 Nov;56(4):345-53.
198. Pieper B, Langemo D, Cuddigan J. Pressure ulcer pain: a systematic literature review and national pressure ulcer advisory panel white paper. *Ostomy Wound Manage.* 2009 Feb;55(2):16-31.
199. Langemo DK, Melland H, Hanson D, Olson B, Hunter S. The lived experience of having a pressure ulcer: a qualitative analysis. *Advances in skin & wound care.* 2000 Sep-Oct;13(5):225-35.
200. Bennett G, Dealey C, Posnett J. The cost of pressure ulcers in the UK. *Age Ageing.* 2004 May;33(3):230-5.
201. Schuurman JP, Schoonhoven L, Defloor T, van Engelshoven I, van Ramshorst B, Buskens E. Economic evaluation of pressure ulcer care: a cost minimization analysis of preventive strategies. *Nurs Econ.* 2009 Nov-Dec;27(6):390-400, 15.
202. Pham B, Teague L, Mahoney J, Goodman L, Paulden M, Poss J, et al. Early prevention of pressure ulcers among elderly patients admitted through emergency departments: a cost-effectiveness analysis. *Ann Emerg Med.* 2011 Nov;58(5):468-78 e3.
203. Severens JL, Habraken JM, Duivenvoorden S, Frederiks CM. The cost of illness of pressure ulcers in The Netherlands. *Advances in skin & wound care.* 2002 Mar-Apr;15(2):72-7.
204. Bertov K, Nordin, A. Synliggöra ekonomiska konsekvenser av förbättringsarbeten. Jönköping 2006.
205. Pham B, Stern A, Chen W, Sander B, John-Baptiste A, Thein HH, et al. Preventing pressure ulcers in long-term care: a cost-effectiveness analysis. *Arch Intern Med.* 2011 Nov 14;171(20):1839-47.
206. Moore ZE, Cowman S. Risk assessment tools for the prevention of pressure ulcers. *Cochrane Database Syst Rev.* 2008(3):CD006471.
207. Kottner J, Hauss A, Schluer AB, Dassen T. Validation and clinical impact of paediatric pressure ulcer risk assessment scales: A systematic review. *Int J Nurs Stud.* 2011 Jun 4.
208. Pancorbo-Hidalgo PL, Garcia-Fernandez FP, Lopez-Medina IM, Alvarez-Nieto C. Risk assessment scales for pressure ulcer prevention: a systematic review. *Journal of advanced nursing.* 2006 Apr;54(1):94-110.
209. Anthony D, Papanikolaou P, Parboteeah S, Saleh M. Do risk assessment scales for pressure ulcers work? *Journal of tissue viability.* 2010 Nov;19(4):132-6.

210. Kottner J, Balzer K. Do pressure ulcer risk assessment scales improve clinical practice? *J Multidiscip Healthc.* 2010;3:103-11.
211. Torra i Bou J-E, Garcia-Fernández FP, Pancorbo-Hidalgo PL, Furtado K. Risk assessment scales for predicting the risk of developing pressure ulcer. In: Romanelli M, editor. *Science and Practice of Pressure Ulcer Management.* London: Springer; 2006. p. 43-57.
212. Anthony D, Parboteeah S, Saleh M, Papanikolaou P. Norton, Waterlow and Braden scores: a review of the literature and a comparison between the scores and clinical judgement. *Journal of clinical nursing.* 2008 Mar;17(5):646-53.
213. Papanikolaou P, Lyne P, Anthony D. Risk assessment scales for pressure ulcers: a methodological review. *Int J Nurs Stud.* 2007 Feb;44(2):285-96.
214. Schoonhoven L, Haalboom JR, Bousema MT, Algra A, Grobbee DE, Grypdonck MH, et al. Prospective cohort study of routine use of risk assessment scales for prediction of pressure ulcers. *Bmj.* 2002 Oct 12;325(7368):797.
215. Defloor T, Grypdonck MF. Validation of pressure ulcer risk assessment scales: a critique. *Journal of advanced nursing.* 2004 Dec;48(6):613-21.
216. Defloor T. Pressure ulcers: Validation of two risk assessment scales. *Journal of clinical nursing.* 2004(14):373-82.
217. NICE guidelines. Pressure ulcers: The management of pressure ulcers in primary and secondary care 2012-02-27; Available from: <http://publications.nice.org.uk/pressure-ulcers-cg29>
218. Sveriges Kommuner och Landsting. Nationell satsning för ökad patientäkerhet, Trycksår, åtgärder för att förebygga. [In Swedish] 2012-02-27; Available from: <http://brs.skil.se/publikationer/index.jsp>.
219. Braden BJ, Bergstrom N. Predictive validity of the Braden Scale for pressure sore risk in a nursing home population. *Res Nurs Health.* 1994 Dec;17(6):459-70.
220. Baath C, Hall-Lord ML, Idvall E, Wiberg-Hedman K, Wilde Larsson B. Interrater reliability using Modified Norton Scale, Pressure Ulcer Card, Short Form-Mini Nutritional Assessment by registered and enrolled nurses in clinical practice. *Journal of clinical nursing.* 2008 Mar;17(5):618-26.
221. Balzer K, Pohl C, Dassen T, Halfens R. The Norton, Waterlow, Braden, and Care Dependency Scales: comparing their validity when identifying patients' pressure sore risk. *J Wound Ostomy Continence Nurs.* 2007 Jul-Aug;34(4):389-98.
222. Waterlow JA. Reliability of the Waterlow score. *J Wound Care.* 1995 Nov;4(10):474-5.
223. Norton D, McLawren, R., Exton-Smith, AN. An investigation of geriatric nursing problems in hospital. ed n, editor. New York: Churtchill Livingstone; 1975, reprinted 1979.
224. Ek AC, Bjurulf P. Interrater variability in a modified Norton Scale. *Scand J Caring Sci.* 1987;1(3-4):99-102.
225. Lindgren M, Unosson M, Krantz AM, Ek AC. A risk assessment scale for the prediction of pressure sore development: reliability and validity. *Journal of advanced nursing.* 2002 Apr;38(2):190-9.
226. Huffines B, Logsdon MC. The Neonatal Skin Risk Assessment Scale for predicting skin breakdown in neonates. *Issues Compr Pediatr Nurs.* 1997 Apr-Jun;20(2):103-14.
227. Wikipedia. 2012-02-27; Available from: <http://en.wikipedia.org/wiki/Color>
228. Fairchild M. *Chromatic Adaption: Color Appearance Models.* Wiley; 2005.
229. Lyder CH. Effective management of pressure ulcers. A review of proven strategies. *Adv Nurse Pract.* 2006 Jul;14(7):32-7; quiz 8.
230. Bergstrom N, Braden, B., Kemp, M., Champagne, M., Ruby, E. Multi-site study of incidence of pressure ulcers and the relationship between risk level, demographic characteristics, diagnoses, and prescription of preventive interventions. *J Am Geriatr Soc.* 1996 Jan;44(1):22-30.

231. Armstrong DG, Ayello, EA., Capitulo, KL., Fowler, E., Krasner, DL., Levine, JM., Sibbald, RG., Smith, A.P. New opportunities to improve pressure ulcer prevention and treatment: implications of the CMS inpatient hospital care Present on Admission (POA) indicators/hospital-acquired conditions (HAC) policy. A consensus paper from the International Expert Wound Care Advisory Panel. *J Wound Ostomy Continence Nurs.* 2008 Sep-Oct;35(5):485-92.
232. Beeckman D, Schoonhoven L, Fletcher J, Furtado K, Gunningberg L, Heyman H, et al. EPUAP classification system for pressure ulcers: European reliability study. *Journal of advanced nursing.* 2007 Dec;60(6):682-91.
233. Scanlon E, Stubbs N. Pressure ulcer risk assessment in patients with darkly pigmented skin. *Prof Nurse.* 2004 Feb;19(6):339-41.
234. Gunningberg L, Lindholm C, Carlsson M, Sjoden PO. Risk, prevention and treatment of pressure ulcers--nursing staff knowledge and documentation. *Scand J Caring Sci.* 2001;15(3):257-63.
235. Gronek JA, Standfill MH. Reporting The Severity of decubitus ulcers. *J AHIMA.* 2005;Apr(76(4)):68-9; Quiz 71-2.
236. Vanderwee K, Grypdonck MH, De Bacquer D, Defloor T. The reliability of two observation methods of nonblanchable erythema, Grade 1 pressure ulcer. *Appl Nurs Res.* 2006 Aug;19(3):156-62.
237. Keller BP, Schuurman JP, van der Werken C. Can near infrared spectroscopy measure the effect of pressure on oxygenation of sacral soft tissue? *J Wound Care.* 2006 May;15(5):213-7.
238. Clarys P, Alewaeters K, Lambrecht R, Barel AO. Skin color measurements: comparison between three instruments: the Chromameter(R), the DermaSpectrometer(R) and the Mexameter(R). *Skin Res Technol.* 2000 Nov;6(4):230-8.
239. Matas A, Sowa MG, Taylor V, Taylor G, Schattka BJ, Mantsch HH. Eliminating the issue of skin color in assessment of the blanch response. *Advances in skin & wound care.* 2001 Jul-Aug;14(4):180-8.
240. Kragelj R, Jarm T, Miklavcic D. Reproducibility of parameters of postocclusive reactive hyperemia measured by near infrared spectroscopy and transcutaneous oximetry. *Annals of Biomedical engineering.* 2000(28 ):pp 168-73.
241. Schubert V. The influence of local heating on skin microcirculation in pressure ulcers, monitored by 242. Schubert V, Perbeck L, Schubert PA. Skin microcirculatory and thermal changes in elderly subjects with early stage of pressure sores. *Clin Physiol.* 1994 Jan;14(1):1-13.
243. Lindberg LG, Tamura T, Oberg PA. Photoplethysmography. Part 1. Comparison with laser Doppler flowmetry. *Med Biol Eng Comput.* 1991 Jan;29(1):40-7.
244. Lyder C. The use of technology for improved pressure ulcer prevention. *Ostomy Wound Manage.* 2007 Apr;53(4):14-6.
245. Sprigle S, Linden M, Riordan B. Characterizing reactive hyperemia via tissue reflectance spectroscopy in response to an ischemic load across gender, age, skin pigmentation and diabetes. *Med Eng Phys.* 2002 Dec;24(10):651-61.
246. Herman EC, Knapp CF, Donofrido JC, Salcido R. Skin perfusion responses to surface pressure-induced ischemia: implication for the developing pressure ulcer. *Journal of Rehabilitation Research & Development.* 1999;April(36(2)).
247. Fujii M, Nakajima K, Sakamoto K, Kanai H. Orientation and deformation of erythrocytes in flowing blood. *Ann N Y Acad Sci.* 1999 Apr 20;873:245-61.
248. Anderson RR, Parrish JA. The optics of human skin. *J Invest Dermatol.* 1981 Jul;77(1):13-9.
249. Lindberg LG, Oberg PA. Photoplethysmography. Part 2. Influence of light source wavelength. *Med Biol Eng Comput.* 1991 Jan;29(1):48-54.

250. Riordan B, Sprigle S, Linden M. Testing the validity of erythema detection algorithms. *J Rehabil Res Dev.* 2001 Jan-Feb;38(1):13-22.
251. Dawson JB, Barker DJ, Ellis DJ, Grassam E, Cotterill JA, Fisher GW, et al. A theoretical and experimental study of light absorption and scattering by in vivo skin. *Phys Med Biol.* 1980 Jul;25(4):695-709.
252. Diffey BL, Oliver RJ, Farr PM. A portable instrument for quantifying erythema induced by ultraviolet radiation. *Br J Dermatol.* 1984 Dec;111(6):663-72.
253. Fullerton A, Fischer T, Lahti A, Wilhelm KP, Takiwaki H, Serup J. Guidelines for measurement of skin colour and erythema. A report from the Standardization Group of the European Society of Contact Dermatitis. *Contact Dermatitis.* 1996 Jul;35(1):1-10.
254. Lofman O, Berglund K, Larsson L, Toss G. Changes in hip fracture epidemiology: redistribution between ages, genders and fracture types. *Osteoporos Int.* 2002 Jan;13(1):18-25.
255. Karagas MR, Lu-Yao GL, Barrett JA, Beach ML, Baron JA. Heterogeneity of hip fracture: age, race, sex, and geographic patterns of femoral neck and trochanteric fractures among the US elderly. *Am J Epidemiol.* 1996 Apr 1;143(7):677-82.
256. Skog M. Regionalt vårdprogram Fallprevention. Medicinskt programarbete; In Swedish: Stockholms Läns Landsting; 2008.
257. Kanis JA, Johnell O, De Laet C, Jonsson B, Oden A, Ogelsby AK. International variations in hip fracture probabilities: implications for risk assessment. *J Bone Miner Res.* 2002 Jul;17(7):1237-44.
258. Thorngren KG. Full treatment spectrum for hip fractures: operation and rehabilitation. *Acta Orthop Scand.* 1997 Feb;68(1):1-2.
259. Gullberg B, Johnell O, Kanis JA. World-wide projections for hip fracture. *Osteoporos Int.* 1997;7(5):407-13.
260. Melton LJ, 3rd. Hip fractures: a worldwide problem today and tomorrow. *Bone.* 1993;14 Suppl 1:S1-8.
261. Michaelsson K, Weiderpass E, Farahmand BY, Baron JA, Persson P-G, Zidén L, et al. Differences in risk factor patterns between cervical and trochanteric hip fractures. *Osteoporos Int.* 1999(10):487-94.
262. Hommel A, Bjorkelund, K., B., Thorngren, K., G., Ulander, K., . A study of a pathway to reduce pressure ulcers for patients with a hip fracture. *Journal of Ortopaedic Nursing.* 2007;11:151-9.
263. Hamlet WP, Lieberman JR, Freedman EL, Dorey FJ, Fletcher A, Johnson EE. Influence of health status and the timing of surgery on mortality in hip fracture patients. *Am J Orthop.* 1997 Sep;26(9):621-7.
264. Al-Ani AN, Samuelsson B, Tidermark J, Norling A, Ekstrom W, Cederholm T, et al. Early operation on patients with a hip fracture improved the ability to return to independent living. A prospective study of 850 patients. *J Bone Joint Surg Am.* 2008 Jul;90(7):1436-42.
265. Lindgren U, Svensson O, Alfredson H, Johansson F. *Ortopedi. 3., [utök. och uppdaterade] uppl. / ed. Stockholm: Liber; 2007.*
266. Brem H, Tomic-Canic M, Tarnovskaya A, Ehrlich HP, Baskin-Bey E, Gill K, et al. Healing of elderly patients with diabetic foot ulcers, venous stasis ulcers, and pressure ulcers. *Surg Technol Int.* 2003;11:161-7.
267. Apelqvist J, Larsson J, Agardh CD. Long-term prognosis for diabetic patients with foot ulcers. *J Intern Med.* 1993 Jun;233(6):485-91.
268. Rogmark C, Johnell O. Ortopaedic treatment of displaced femoral neck fractures in elderly patients. *Disability and rehabilitation.* 2005 Sep 30-Oct 15;27(18-19):1143-9.

269. Tidermark J, Ponzer S, Svensson O, Soderqvist A, Tornkvist H. Internal fixation compared with total hip replacement for displaced femoral neck fractures in the elderly. A randomised, controlled trial. *J Bone Joint Surg Br.* 2003 Apr;85(3):380-8.
270. Ekström W. Proximal femoral fractures : functional outcome, quality of life and mortality. Stockholm: Karolinska Institutet; 2008.
271. Bornemark J, Svenaeus F. *Studies in practice knowledge*. . Huddinge: Södertörns högskola 2009.
272. Gustavsson B. *Utbildningens förändrade villkor, nya perspektiv på kunskap, bildning och demokrati*. Stockholm: Liber; 2011.
273. Beeckman D, Defloor T, Schoonhoven L, Vanderwee K. Knowledge and attitudes of nurses on pressure ulcer prevention: a cross-sectional multicenter study in Belgian hospitals. *Worldviews Evid Based Nurs.* 2011 Sep;8(3):166-76.
274. Yura H, Walsh M. *The nursing process: Assessing, planning, implementing, evaluating*. 2nd ed. New York: Appleton-Century-Crofts; 1973.
275. Björvell C. *Sjuksköterskans journalföring och informationshantering, en praktisk handbok*. Lund: Studentlitteratur; 2011.
276. Ehnfors M. *Quality of care from a nursing perspective*. Uppsala: Uppsala Universitet; 1993.
277. Thorell-Ekstrand I. *Clinical nursing education. The learning of individual care planning*. Stockholm 1994.
278. Ehrenberg A. *In pursuit of the common thread. Nursing content in patient records with special reference to nursing care*. Uppsala 2000.
279. Björvell C. *Nursing documentation in clinical practice. Instrument development and evaluation of a comprehensive intervention programme*. Stockholm 2002.
280. Ehrenberg A, Ehnfors M. The accuracy of patient records in Swedish nursing homes: congruence of record content and nurses' and patients' descriptions. *Scand J Caring Sci.* 2001;15(4):303-10.
281. O'Brien JA, C, S. An exploration of nursing documentation of pressure ulcer care in an acute setting in Ireland. *J Wound Care.* 2011 May;20(5):197-8, 200, 2-3 passim.
282. Gunningberg L, Ehrenberg A. Accuracy and quality in the nursing documentation of pressure ulcers: a comparison of record content and patient examination. *J Wound Ostomy Continence Nurs.* 2004 Nov-Dec;31(6):328-35.
283. Gunningberg L, Dahm MF, Ehrenberg A. Accuracy in the recording of pressure ulcers and prevention after implementing an electronic health record in hospital care. *Qual Saf Health Care.* 2008 Aug;17(4):281-5.
284. Stevenson JE, Nilsson G. Nurses' perceptions of an electronic patient record from a patient safety perspective: a qualitative study. *Journal of advanced nursing.* 2011 Jul 22.
285. Thoroddsen A, Ehnfors M, Ehrenberg A. Nursing specialty knowledge as expressed by standardized nursing languages. *Int J Nurs Terminol Classif.* 2010 Apr-Jun;21(2):69-79.
286. Wang N, Hailey D, Yu P. Quality of nursing documentation and approaches to its evaluation: a mixed-method systematic review. *Journal of advanced nursing.* 2011 Sep;67(9):1858-75.
287. Svensk Författnings Samling. *Patientdatalag (2008:355)*; In Swedish. Stockholm.
288. Benner P, Tanner CA, Chesla CA. *Expertise in clinical practice: Caring, clinical judgment, and ethics*. New York: Springer; 1996.
289. Herdman TH, editor. *Omvårdnadsdiagnoser enligt NANDA - definitioner och klassifikationer 2009-2011 (Nursing diagnoses in Swedish)*. Lund: Studentlitteratur; 2009.
290. Walsh K, Bennett, G. Pressure ulcers as indicators of neglect. In: Clark M, editor. *Pressure ulcer: Recent advances in tissue viability* 2004. p. 116-22.

291. Svensk Författningssamling. Patientsäkerhetslag (2010:659): In Swedish.
292. Socialstyrelsen. Ledningssystem för kvalitet och patientsäkerhet inom hälso- och sjukvården. SOSFS 2011:9 (M o S); In Swedish. Stockholm 2011.
293. Ayello EA, Lyder CH. Protecting patients from harm: preventing pressure ulcers in hospital patients. *Nursing*. 2007 Oct;37(10):36-40; quiz -1.
294. Healey F. Root cause analysis for tissue viability incidents. *Journal of tissue viability*. 2006 Feb;16(1):12-5.
295. Reason J. Beyond the organisational accident: the need for "error wisdom" on the frontline. *Qual Saf Health Care*. 2004 Dec;13 Suppl 2:ii28-33.
296. Reason J. *The Human Contribution, unsafe acts, accidents and heroic recoveries*: Ashgate Publishing Limited; 2008.
297. Reason J. Human error: models and management. *BMJ*. 2000 Mar 18;320(7237):768-70.
298. Rasmussen J. Risk management in a Dynamic Society: A Modelling Problem. *Safety Science*. 1997;27(2/3):183-213.
299. Reason J. Safety in the operating theatre - Part 2: human error and organisational failure. *Qual Saf Health Care*. 2005 Feb;14(1):56-60.
300. Flin R, O'Connor P, Crichton M. *Safety at the Sharp End. A Guide to Non-technical Skills* 2008.
301. Ödegård S. *Säker Vård - patientskador, rapportering och prevention*. Göteborg 2006.
302. Harvard Family Reseach Project. 2012-02-27; Available from: <http://www.hfrp.org/publications-resources/browse-our-publications/indicators-definition-and-use-in-a-results-based-accountability-system>.
303. Donabedian A. *Basic Approaches to assessment: structure, process and outcome*. . Ann Arbor, Michigan: Health Administration Press,; 1989.
304. Socialstyrelsen. *God vård - om ledningssystem för kvalitet och patientsäkerhet i hälso- och sjukvården*; In Swedish. Stockholm 2009.
305. Witkowski JA, Parish LC. Histopathology of the decubitus ulcer. *J Am Acad Dermatol*. 1982 Jun;6(6):1014-21.
306. Husain T. An experimental study of some pressure effects on tissues, with reference to the bed-sore problem. *J Pathol Bacteriol*. 1953 Oct;66(2):347-58.
307. Salcido R, Donofrio JC, Fisher SB, LeGrand EK, Dickey K, Carney JM, et al. Histopathology of pressure ulcers as a result of sequential computer-controlled pressure sessions in a fuzzy rat model. *Adv Wound Care*. 1994 Sep;7(5):23-4, 6, 8 passim.
308. Ek AC, Lewis DH, Zetterqvist H, Svensson PG. Skin blood flow in an area at risk for pressure sore. *Scand J Rehabil Med*. 1984;16(2):85-9.
309. Schubert V, Fagrell B. Local skin pressure and its effects on skin microcirculation as evaluated by laser-Doppler fluxmetry. *Clin Physiol*. 1989 Dec;9(6):535-45.
310. Stekelenburg A, Strijkers GJ, Parusel H, Bader DL, Nicolay K, Oomens CW. Role of ischemia and deformation in the onset of compression-induced deep tissue injury: MRI-based studies in a rat model. *J Appl Physiol*. 2007 May;102(5):2002-11.
311. Loerakker S, Manders E, Strijkers GJ, Nicolay K, Baaijens FP, Bader DL, et al. The effects of deformation, ischemia, and reperfusion on the development of muscle damage during prolonged loading. *J Appl Physiol*. 2011 Jul 14.
312. Oomens C, Baaijens F. An overview of theoretical studies of the mechanical effects on cellular behaviour. Preface. *Comput Methods Biomech Biomed Engin*. 2011 May;14(5):401.
313. Defloor T, Clark M, Witherow A, Colin D, Lindholm C, Schoonhoven L, et al. EPUAP statement on prevalence and incidence monitoring of pressure ulcer occurrence. *Journal of tissue viability*. 2005 Aug;15(3):20-7.

314. Barnett RI, Ablarde JA. Skin vascular reaction to short durations of normal seating. *Arch Phys Med Rehabil.* 1995 Jun;76(6):533-40.
315. Kottner J, Tannen A, Dassen T. Hospital pressure ulcer prevalence rates and number of raters. *Journal of clinical nursing.* 2009 Feb 5.
316. Bethell E. Controversies in classifying and assessing grade 1 pressure ulcers. *Nurs Times.* 2003;Apr 1-7(99(13)):73-5.
317. Thomas DR, Osterweil D. Is a pressure ulcer a marker for quality of care? *J Am Med Dir Assoc.* 2005 May-Jun;6(3):228-30.
318. Gunningberg L, Fogelberg-Dahm M, Ehrenberg A. Improved quality and comprehensiveness in nursing documentation of pressure ulcers after implementing an electronic health record in hospital care. *Journal of clinical nursing.* 2009 Feb 5.
319. Reddy M, Gill SS, Kalkar SR, Wu W, Anderson PJ, Rochon PA. Treatment of pressure ulcers: a systematic review. *Jama.* 2008 Dec 10;300(22):2647-62.
320. Pfeiffer E. A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. *J Am Geriatr Soc.* 1975 Oct;23(10):433-41.

## APPENDIX 1

The Braden Scale is composed of 6 broad clinical categories as sensory perception, moisture, activity, morbidity, nutrition and friction and shear, with a score of <18 indicate increased risk for PUs development <sup>219</sup>.

|   |   |   |   |   |
|---|---|---|---|---|
| <p><b>SENSORY PERCEPTION</b><br/>Ability to respond meaningfully to pressure-related discomfort</p>                                     | <p><b>1. COMPLETELY LIMITED</b> – Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation, <b>OR</b> limited ability to feel pain over most of body surface.</p>  | <p><b>2. VERY LIMITED</b> – Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness, <b>OR</b> has a sensory impairment which limits the ability to feel pain or discomfort over ½ of body.</p>   | <p><b>3. SLIGHTLY LIMITED</b> – Responds to verbal commands but cannot always communicate discomfort or need to be turned, <b>OR</b> has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.</p>   | <p><b>4. NO IMPAIRMENT</b> – Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.</p>   |
| <p><b>MOISTURE</b><br/>Degree to which skin is exposed to moisture</p>  | <p><b>1. CONSTANTLY MOIST</b>– Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.</p>  | <p><b>2. OFTEN MOIST</b> – Skin is often but not always moist. Linen must be changed at least once a shift.</p>   | <p><b>3. OCCASIONALLY MOIST</b> – Skin is occasionally moist, requiring an extra linen change approximately once a day.</p>   | <p><b>4. RARELY MOIST</b> – Skin is usually dry; linen only requires changing at routine intervals.</p>   |
| <p><b>ACTIVITY</b><br/>Degree of physical activity</p>  | <p><b>1. BEDFAST</b> – Confined to bed.</p>   | <p><b>2. CHAIRFAST</b> – Ability to walk severely limited or nonexistent. Cannot bear own weight and/or must be assisted into chair or wheelchair.</p>  | <p><b>3. WALKS OCCASIONALLY</b> – Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair.</p>   | <p><b>4. WALKS FREQUENTLY</b>– Walks outside the room at least twice a day and inside room at least once every 2 hours during waking hours.</p>   |
| <p><b>MOBILITY</b><br/>Ability to change and control body position</p>  | <p><b>1. COMPLETELY IMMOBILE</b> – Does not make even slight changes in body or extremity position without assistance.</p>  | <p><b>2. VERY LIMITED</b> – Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.</p>   | <p><b>3. SLIGHTLY LIMITED</b> – Makes frequent though slight changes in body or extremity position independently.</p>   | <p><b>4. NO LIMITATIONS</b> – Makes major and frequent changes in position without assistance.</p>  |
| <p><b>NUTRITION</b><br/>Usual food intake pattern<br/>1NPO: Nothing by mouth. 2IV: Intravenously. 3TPN: Total parenteral nutrition.</p> | <p><b>1. VERY POOR</b> – Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement.<br/><br/><b>OR</b> is NPO1 and/or maintained on clear liquids or IV2 for more than 5 days.</p> | <p><b>2. PROBABLY INADEQUATE</b> – Rarely eats a complete meal and generally eats only about ½ of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement<br/><br/><b>OR</b> receives less than optimum amount of liquid diet or tube feeding.</p> | <p><b>3. ADEQUATE</b> – Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) each day. Occasionally refuses a meal, but will usually take a supplement if offered,<br/><br/><b>OR</b> is on a tube feeding or TPN3 regimen, which probably meets most of nutritional needs.</p> | <p><b>4. EXCELLENT</b> – Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.</p> |
| <p><b>FRICTION AND SHEAR</b></p>  | <p><b>1. PROBLEM-</b><br/>Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction.</p>             | <p><b>2. POTENTIAL PROBLEM–</b><br/>Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.</p>                                      | <p><b>3. NO APPARENT PROBLEM</b> – Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.</p>  |   |

Source: Barbara Braden and Nancy Bergstrom. Copyright, 1988. Reprinted with permission. Permission should be sought to use this tool at [www.bradenscale.com](http://www.bradenscale.com)

## APPENDIX 2

The Norton Scale is composed of 5 broad clinical categories as physical condition, mental state, activity, mobility and incontinence. A score of <16 indicates increased risk for PU development<sup>223</sup>

| Physical state | Mental state | Activity                  | Mobility             | Incontinence            |
|----------------|--------------|---------------------------|----------------------|-------------------------|
| 4 – Good       | 4 – Alert    | 4 – Walks                 | 4 – Complete         | 4 - None                |
| 3 – Weak       | 3 – Apathic  | 3 – Walks with assistance | 3 – Slightly limited | 3 - Occasional          |
| 2 – Ill        | 2 - Confused | 2 – Wheelchair bound      | 2 – Very limited     | 2 – Mainly urinary      |
| 1 – Very ill   | 1 - Stupor   | 1 – Bed bound             | 1 - Immobile         | 1 – Double incontinence |

## APPENDIX 3

The Modified Norton Scale is composed of 7 broad clinical categories as physical condition, activity, mobility, *nutrition, fluid intake*, incontinence and *general condition*, with a score of <20 indicates increased risk for PUs development Modified Norton<sup>224</sup>.

| Score | Physical state | Activity              | Mobility         | Nutritional                | Fluid intake     | Incontinence        | General condition                              |
|-------|----------------|-----------------------|------------------|----------------------------|------------------|---------------------|--|
| 4     | Good           | Walks                 | Complete         | Normal Ration?             | Complete         | None                | Good   |
| 3     | Weak           | Walks with assistance | Slightly limited | ¾ of a normal potion ?     | Slightly limited | Occasional          | Pretty good (e.g. subfebrile, awake)           |
| 2     | Ill            | Wheelchair bound      | Very limited     | ½ of a normal portion      | Very limited     | Mainly urinary      | Poor (e.g. awake but apathetic)                |
| 1     | Very ill       | Bed bound             | Immobile         | less than ½ normal portion | Immobile         | Double incontinence | Very poor (e.g. sign circ. insuff., somnolent) |

## APPENDIX 4

SPMSQ – the short portable mental status questionnaire<sup>320</sup>

|       |   |                             |
|-------|---|-----------------------------|
| 1     | <i>What is the date today?</i>  |                             |
| 2     | <i>What day of the week is it?</i>  |                             |
| 3     | <i>What is the name of this palace?</i>   |                             |
| 4     | <i>What is your telephone number or (alt.) street address?</i>                          |                             |
| 4A    | <i>What is your street address?<br/>(Ask only if patient does not have a telephone)</i> |                             |
| 5     | <i>How old are you?</i>   |                             |
| 6     | <i>When were you born?</i>  |                             |
| 7     | <i>Who is the President of U.S. now?</i>  |                             |
| 8     | <i>Who was the President just before him?</i>   |                             |
| 9     | <i>What was your mother's maiden name?</i>  |                             |
| 10    | <i>Subtract 3 from 20 and keep subtracting 3 from each new number all the way down</i>  |                             |
| Score | <i>Intact cognitive function</i>  | <i>8-10 correct answers</i> |
|       | <i>Mild cognitive impairment?</i>   | <i>6-7 correct answers</i>  |
|       | <i>Moderate cognitive impairment?</i>   | <i>3-5 correct answers</i>  |
|       | <i>Severe cognitive dysfunction</i>   | <i>0-2 correct answers</i>  |

Swedish version. Question number 4, 7 and 8 has smaller changes.

|   |  |  |
|---|--|--|
| 4 | <i>What is your telephone number or (alt.) street address?</i> |  |
| 7 | <i>Who is the prime minister now?</i>                          |  |
| 8 | <i>Who was the prime minister before him?</i>                  |  |

