The value of pressure ulcer risk assessment and interface pressure measurements in patients

A nursing perspective

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De waarde van decubitus risico bepalingen en het meten van de interface druk bij patiënten
Een verpleegkundig perspectief

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Jan Tjitse Marinus Weststrate

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Promotiecommissie

Promotor: Prof.dr. H.A. Bruining

Overige leden: Prof.dr. S.E.R. Hovius
                    Prof.dr. ir. C.J. Snijders
                    Prof.dr. H.A.M. Neumann

Copromotor   Dr. F. Heule

Paranimfen: Renske Dam-Weststrate
                    Tjitze Weststrate

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If you can keep your head when all about you
Are losing theirs and blaming it on you,
If you can trust yourself when all men doubt you,
But make allowance for their doubting too;
If you can wait and not be tired by waiting,
Or being lied about, don't deal with lies,
Or being hated don't give way to hating,
And yet don't look too good, nor talk too wise:

If you can dream --- and not make dreams your master;
If you can think --- and not make thoughts your aim,
If you can meet with Triumph and Disaster
And treat those two impostors just the same;
If you can bear to hear the truth you've spoken
Twisted by knaves to make a trap of fools,
Or watch the things you gave your life to, broken,
And stoop and build 'em up with worn-out tools:

If you can make one heap of all your winnings
And risk it on one turn of pitch – and – toss,
And lose, and start again at your beginnings
And never breathe a word about your loss;
If you can force your heart and nerve and sinew
To serve your turn long after they are gone,
And so hold on when there is nothing in you
Except the Will which says to them: "Hold on!"

If you can talk with crowds and keep your virtue,
Or walk with Kings --- nor lose the common touch,
If neither foes nor loving friends can hurt you,
If all men count with you, but none too much;
If you can fill the unforgiving minute
With sixty seconds' worth of distance run,
Yours is the Earth and everything that's in it,
And --- which is more --- you'll be a Man, my son!

By Rudyard Kipling from *Rewards and Fairies*, 1910

Aan mijn ouders

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List of abbreviations

ANOVA	Analyses of Variance
APACHE	Acute Physiological and Chronic Health Evaluation
AR	Array
BMI	Body Mass Index = (weight (kg) /(length (m))^2)
BSA	Body Surface Area
CBO	The Dutch Institute for Healthcare Improvement
CBS	The Dutch Central Bureau for Statistics
CE	Conformité Européenne
EFCCNa	European Federation of Critical Care Nursing associations
EPUAP	European Pressure Ulcer Advisory panel
ESICM	European Society of Intensive Care Medicine
FSA	Forced Sensitive Array
ICC	Intra-class Correlation Coefficient
ICU	Intensive Care Unit
IP	Interface Pressure
Kg	Kilogram
kPa	1Kilo Pascal = ± 7.5 mmHg
M	Meter
Min	Minute
NAS	Nursing the patient on Alternate Sides
NT	Not Tested
OTM	Operation Table Mattress
PIP	Peak Interface Pressure
PU	Pressure Ulcer
RH	Reactive Hyperemia
SICU	Surgical Intensive Care Unit
SS	Single Sensor
TIP	Tissue Interface Pressure
TIPE	Texas Interface Pressure Evaluation
TPM III	Talley Pressure Monitor III
VC	Variation Coefficient
YRS	Years

Chapter 1

General introduction

1.1   Conceptual schemes in pressure ulcer development

The two widely used concepts for the development of pressure ulcer (PU) emphasise that the two critical factors are pressure[1]- and shear forces [2, 3] and the viability of the skin tissues. If no pressure is applied to the skin tissues, patients with the most severely compromised viability of these tissues do not develop a PU. However, if pressure is exerted to the skin tissues in this patient group, and particularly to skin over bony prominences, PU will rapidly develop. The role of the exerted pressure, the different measurement techniques and the viability of the skin tissues in the development of PUs are highlighted in this introduction.

Consequences of pressure in human body

A patient lying on a bed for a long time exerts forces on the mattress. In turn, the mattress exerts forces on the body of the patient. The actual stress exerted on different parts of the body depends on a number of factors such as:

- The actual sites of contact between the patient and the mattress
- The stress situation in the adjoining sites of the body
- The direction of the sites of contact compared with the gravitational force
- The material properties of the site (hard bone or soft tissue) of the body that is in contact with the mattress
- The material properties of the sites of the body surrounding this site
- The material properties of the mattress

Precise information on the actual stress on the body and its consequences may be the key to a fundamental explanation on "stress ulcer" development. Some investigators suspect that the deformation resulting from the stress on the body is an important parameter determining the actual damage caused by prolonged lying. The Technical University in Eindhoven has recently conducted several studies on methods to measure deformation [4, 5]. Complicating factors are the inability to measure the continuously changing stress on body sites directly and the translation of available measurements into an adequate description of stress on body sites.

Therefore, we have to rely on incomplete information in our efforts to describe and prevent development of "stress ulcers". In order to be able to combine the efforts of the scientific community in this field, it is necessary to accurately describe the parameters we measure. Although the term stress would be more appropriate as explained, we further adhere to the term **pressure** in order to conform with the medical tradition.

In this chapter a series of measurements on the stress exerted on a measuring

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[1] Physicists generally define pressure as the force exerted over a unit/area accordingly to Newton's first and second laws [1]. Stress as expressed in tractions and the collection of tractions, however, would be a more appropriate term. Here for conventional reasons, we use the phrase pressure.

device that was located between the individual and a mattress are described. This measurement is referred to as the "interface pressure" (IP).

Clinical characteristics of the skin after pressure application

The first visible sign after pressure has been applied is a local non-specific erythema of the skin. This is often mentioned in the literature as Reactive Hyperemia (RH). Reactive Hyperemia is defined as "a characteristic bright flush of the skin associated with an increased volume of the pulse on the release of an obstruction to the circulation" [6]. The maximum flow occurs immediately after the obstruction is removed. If the erythema disappears under the locally applied pressure (for example through pressure applied by a finger or by using a transparent disc) this is called "blanching'' hyperemia indicating an intact microcirculation [7]. If the redness does not disappear it is called "non-blanching" hyperemia, indicating a microcirculatory disruption.

The "blanching" and "non-blanching" hyperemia is the result of RH [8] and can be identified at two levels. The first is a vasodilatory response as a reaction to a complete standstill of arterial blood flow and is visualized by a "non-blanching hyperemia". This occurs mainly in the deeper tissue structures such as the muscles. There is also a more superficial response as a result of a reduction of blood flow prominently in the dermal tissues and noted as "blanching" hyperemia [7]. The occurrence of RH is a natural reflex vasodilatation of the dermal blood vessels for temporarily increasing the blood flow and therefore the oxygen supply and nutrition to specific ischemic parts of the dermal tissues [9]. This reaction is locally organized and independent of the central nervous control [6]. However, the intensity of the reaction may differ per patient [10].

At the stage of "blanching" hyperemia the blood vessels are already slightly damaged because of ischemia, which creates a local aseptic inflammatory reaction and causes redness that disappears under pressure (made visible through a transparent disc). If the exerted pressure on the dermal tissue continues, the "blanching" hyperemia will change into "non-blanching hyperemia" because the damaged blood vessels start leaking and thrombi are formed [11].

Lewis and Grant [6] reported that the duration of the RH response continued for half the time the blood vessels were occluded by the exerted pressure and that this fraction augmented with an increasing period of the occlusion. This was supported by Kosiak [12]. He also mentioned that the protective response of RH decreased before dermal tissue damage was noted indicating that the absence of redness did not guarantee that no PU will develop in the near future.

The exact role of the RH remains uncertain. Lewis and Grant [6] found evidence that the RH was initiated in order to pay off the metabolic debt accumulated during the occlusion. Other investigators reported that no "debt" was paid off upon gradual restoration of the blood flow but that the function of the hyperemia was to re-establish the resting metabolic state of the dermal tissues as quickly as possible [13].

If prevention is not initiated early enough, the exerted pressure will cause splitting of the cutis and the space is filled with fluid resulting in the formation of a blister. When dermal tissue remains under pressure the epidermis will break and extended damage is caused to the underlying tissues. Under the influence of bacteria and their waste products more subcutaneous tissue will be destroyed creating necrotic tissue which spreads to the deeper layers of fat and muscle tissue culminating in a PU. At this stage the borders of the PU are not yet undermined. The following stage is characterized by an aseptic inflammatory process in the deeper layers of the tissues and particularly by the necrosis of fat tissue. Bacteria invade and cause the formation of abscesses that break through to various sides of the ulcer creating cavities undermining the borders of the ulcer. Enzymatic breakdown of tissues by matrix metalloproteinases enhances this process [14].

Etiology of Pressure Ulcers

Clinical workers often view the development of a PU as a black box where the applied pressure to the dermal tissues results in a PU without exactly knowing what has happened at the cellular level in these tissues. As mentioned earlier, the primary cause of a PU is based on the exerted pressure and shear forces on the patient's dermal tissues whereby the supply of oxygen is reduced or cut-off causing necrosis of these tissues [15]. During the last forty years, a number of theories have been formulated which describe the possible pathophysiologic processes involved and ensuing histopathologic events. These theories are:
1. Theories based on localized ischemia [16-19]
2. Theories based on impaired interstitial fluid flow and lymphatic drainage [20-23]
3. Theories based on reperfusion injury [24, 25]
4. Theories based on sustained deformation of cells [26, 27].

Classification of Pressure ulcers

In the past various investigators have studied the process of PU development and searched for a visual description of the critical stages (the outside shows what is going on in the inside). This enables clinicians to place the actual observations in the perspective of the overall process in the development of a PU without the need for a histological description. Such a classification system indicates the severity, the necessary care and the prognosis of the actual observation [28]. Yarkony *et al.* [29] described three main purposes of such a system:
1. To improve uniform communication between clinicians on the severity of the PU
2. To evaluate various treatment regimes for PUs
3. To improve the accuracy of medical and nursing records for clinical care and research.

Haalboom [30] supported this concept and stated the following requirements for such a classification system.

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1. The classification system should be such that it can be used at the bedside
2. Its use should be uniform
3. It should have a high inter-observer reliability
4. There should be a logical correlation between the classification, the severity of the PU and the prognosis
5. Its use should be international
6. There should be a relationship between the classification and preventive or treatment policies.

Various PU classification systems have been developed over the years. In the United Kingdom alone 14 classification systems have been used [31]. One of the first classification systems based on histological / anatomical findings was created by Shea [32]. An overview of this classification is shown in Table I.

Table I.   Classification of pressure ulcers according to Shea [32]

Stage 1	Limited to epidermis, exposing dermis
Stage 2	Full-thickness of dermis to junction of subcutaneous fat
Stage 3	Fat obliterated, limited by deep fascia undermining the skin
Stage 4	Bone at the base of the ulceration
Stage 5	Closed large cavity through a small sinus.

The classification systems also changed, when the etiology of PU development became clearer. In the 1980s of the twentieth century, the Torrance system [33] was developed. The uniqueness of this classification system is the inclusion of blanching erythema as one of the stages in the development of a PU. Most other systems do not qualify blanching erythema as a PU stage as this is in principle reversible. Nevertheless the presence of blanching erythema may be seen as a pre-ulcer stage as this is the first visible sign that indicates that exerted pressure diminishes the blood flow in the dermal tissues. Including this stage in the classification system enhances the awareness of the clinical staff that in the particular patient there is a potential risk of developing a PU at the specific location [29]. Considering blanching erythema as stage 1 also suggests it is not normal as most other classification systems classify this stage as stage 0, the same as if there was no visible erythema at all.

Yarkony *et al.* [29] divided this stage further and related it to the duration of the blanching erythema (see Table II). In their classification system, stage 1 was divided into stages 1a and 1b where stage 1a is applicable when the erythema is present for between 30 minutes and 24 hours and stage 1b is applicable if erythema is present for longer than 24 hours. Their classification system thus included the influence of time in relation to the tissue damage. Since it is difficult for clinicians to keep track of the exact onset of the

erythema and whether it was present for longer than 24 hours, it is difficult to chart because various nurses and doctors take care of the patients.

Table II.   Classification of pressure ulcers according to Yarkony & Kirk [29[

Stage 1	a. Red areas lasting between 30 minutes and 24 hours
	b. Red areas lasting longer than 24 hours
Stage 2	Epidermis and / or dermis ulcerated with no subcutaneous fat observed
Stage 3	Subcutaneous fat observed, no muscle observed
Stage 4	Muscle / fascia observed, but no bone observed
Stage 5	Bone observed, but no involvement of joint space
Stage 6	Involvement of joint space.

This classification was criticized by Witkowski and Parish [34] who stated that blanching or non-blanching was more important than the duration. The argument that was put forward was that only reactive vasodilatation was involved if erythema persisted for less than 30 minutes. This was in contrast to erythema that persisted for longer than 30 minutes whereby edema in the upper portions of the dermis and vascular dilatation were present. They also stated that erythema that persisted for longer than 24 hours was always non-blanchable and showed all of the earlier mentioned histological changes in addition to inflammation of blood vessels with erythrocyte extravasation.

1.2   Measurement of pressure

Measuring the pressure between humans and their supporting material started within the realm of healthcare system [35]. Various pressure measurement techniques were developed, when it became apparent that the type of supporting material that was used for patients in a wheelchair mattered in relation to the risk of developing PUs.

If the type of mattress influenced the prevalence of PU in wheelchair patients it must also influence the prevalence in hospitalized patients and specifically in those patients who were immobile or unable to change their body position. Therefore, research in this area was undertaken in high risk patients with spinal cord injuries who were assigned to standard hospital mattresses and special pressure reducing mattresses.

As it is estimated that one quarter of all the work in industrial countries is sedentary [36], pressure measurements also caught the attention of industrial designers and the allied industry that designed and manufactured chairs. Seat pressure measurements were carried out in several types of industries in which seating comfort was an important issue [37] because comfortable seating influenced the productivity of the employee, whereas uncomfortable seats worsened the pre-existing medical conditions such as back aches.

During the last century investigators started to develop instruments that were able to indicate the pressure that was applied to areas of dermal tissues which were susceptible for the development PU. Kosiak *et al.* [38] measured the pressure exerted on the dermal tissues in the pelvic area of 11 adults who set on various surfaces. They used 12 rubber butterfly type valves of 2 cm long and 1 cm wide that were attached to a non-distensible closed system into which a steady flow of air was maintained from a pressure source of approximately 600 mmHg. The pressure at which equilibrium was reached between the inflow of air into the system and escape of air through the test valve was accepted as the pressure applied externally on the valve. This in turn gave an indication of the pressure that was applied to the skin of the volunteer. The instrument could only measure the pressure of up to 300 mmHg accurately. If the pressure exceeded 300 mmHg, the value was obtained by extrapolation as it was assumed that the error in this pressure range was similar to that from 0-300 mmHg.

The measurements by Kosiak and his team were carried out with the volunteer in an upright sitting position. Therefore, two valves were always positioned under the ischial tuberosities as it was expected that these were the two locations that exerted the highest pressure. Pressures were measured in volunteers sitting on 6 different surfaces (wooden office chair, flat board, 1 and 2 inch foam, and a padded and unpadded alternating mattress). The main conclusion was that the use of alternating mattresses provided the largest reduction in the exerted pressure, specifically in the buttock area, which was within the range of the capillary blood pressure (32 mmHg = 4.3 kPa) [38].

Since this was one of the first instruments with which one could actually measure and quantify (mmHg) the effect on the tissues when pressure was exerted by the body on the mattress, this method may be considered as the first method for evaluating commercially manufactured mattresses. The instrument consisted of various tubes, recorders and transducers and therefore, was complicated to use. It could only be effectively operated in a laboratory setting. This limited the use of such an instrument for measuring IP in a clinical setting.

Lindan, Greenway and Piazza [39] were the first who developed a measuring device which could create a total body pressure map of the pressures exerted on the human body supported on a mattress. They used the "bed of springs and nails" which consisted of a piece of plywood with holes 1.4 cm apart for sitting measurements and 2 cm apart for lying measurements. In each hole a 7.5 cm nail was placed whereby in a supine position 1000 nails created a "discontinuing surface" that supported the subject. Around each nail two types of steel springs with different resistance were used. The resistance of the "bed of springs and nails" could be changed by changing the spacing of the nails or changing the resistance of the springs. The force that was necessary to compress the spring 1 cm (range 1.9 kPa (14.9 mmHg) -8.2 kPa (61.8 mmHg)) was calculated for each nail in combination with the two types of springs and two spacing distances. The subjects were placed on the "bed of springs and nails". The number of nails that were displaced was measured by hand with an accuracy of within 0.5 mm. After the compression of each nail was known, the

points of identical compression were connected and isobars were drawn which created a "pressure map" of the body that graphically showed the high and the low pressure areas. In doing so they came to the following conclusions:

- Placing subjects in a prone position created more, but smaller high pressure areas, and also large low pressure areas compared with subjects in a supine position.
- Softer mattresses resulted in lowering the maximum pressures by increasing the area of lower pressures
- The effect of ischial cut-outs increased the areas of maximum pressures because the total pressure is distributed over a smaller area
- Obese subjects had larger areas with lower pressure compared with under-weight subjects who had small areas of high pressure.

This investigation may be considered as a breakthrough because it demonstrated that a mattress with different resistance clearly influenced the pressure that was exerted on the dermal tissues by the human body. It also emphasized that different body shapes created different "pressure maps" and therefore, underwrote the uniqueness of each human being. The consequence of this research was that human beings may react very differently in terms of "pressure mapping" to a standard mattress. This instrument can only be used in a laboratory setting considering its size and the technique for investigating the pressure distribution on the human body. The assessment of the pressure distribution in actual hospitalized patients is difficult to perform with such an instrument in view of the discomfort caused to the patients during the whole measurement.

Following these two investigations, a large number of instruments were developed over the last three decades which can be used to measure the pressure between cutaneous tissue and the mattress. Bush [40] as one of the first, measured the pressure between the dermal tissue and the seat with an electronic sensor at the site of the ischial tuberosities. Others followed quickly [41-44]. The main problems with these early types of electronic measuring devices were that they were unreliable in terms of validity and reproducibility [35]. This is probably the reason that other types of sensors based on other technologies were developed and refined so that measurements became more reproducible.

Electro-pneumatic sensors

The next generation of sensors that were developed were the so-called electro-pneumatic sensors [45]. Basically these consisted of an inflatable sac inside which electrical contacts strips are positioned cross diagonally. The working principle is as follows. The sensor is placed between the body and the mattress at the site of interest. Air is slowly pumped into the sensor to the level at which the electrical contact between both strips breaks. The pressure recorded at that moment is considered to be the pressure exerted on the sensor by the body on the mattress under study. Another example of an electro-pneumatic measuring device is the Pressure Evaluation Pad designed by Garber, Krouskop and Carter [46].

Pneumatic sensors

Bader [47] developed a pneumatic sensor which has been commercially available as the Talley Pressure Monitor (TPM). This sensor consists of air cells that are connected to a high pressure pump. The sensors are placed between the body and the mattress at the site of interest. The working of a pneumatic sensor is as follows. The pump will try to inflate the sensor by increasing the inflation pressure just above the pressure applied to the sensor by the body on the mattress. At the moment this is actual, a sudden drop in the pressure increase is recorded. The pressure at the moment the increase in pressure stops is the applied interface pressure [35].

Another much used pneumatic sensor, the Texas Interface Pressure Evaluator was developed by Krouskop [22]. The working of this system is as follows. The sensor is placed between the body and the mattress at the site of interest. The sensor is over-inflated, then a relief valve opens whereby the air pressure in the pad is slowly reduced. When the pressure exerted by the body on the sensor exceeds the pressure keeping the relief valve open, the valve closes and the corresponding pressure is displayed. This sensor was used in a number of investigations in which the pressure reducing capacity of mattresses was evaluated [48, 49]. Later, a mini-version was manufactured and used in various investigations [29, 50-52].

Another type of sensor that was used in studies on this subject was a 100 ml fluid filled intravenous bag connected to the bedside hemodynamic monitor [53]. The investigators stated that it was small enough (4½ x 3½ x ½ inch) so that it was unlikely that it would act as a secondary flotation device. They measured the pressure between the mattress and the sacrum and the heel of 57 intensive care patients. Using the device as an intravenous fluid bag connected to the hemodynamic monitoring system may be interesting as data are recorded continuously.

Electronic sensors

In the 1980s of the last century, there was renewed interest for using electronic sensors for measuring the pressure at the interface of the body and the mattress. This was probably the result of an improved developing technology that made it possible to overcome earlier problems in terms of validity and reproducibility. Two main types of electronic sensors with different working principles have been described in the literature. Both principles are discussed.

a. Capacitive sensors

Bethaves, [54] described the working principle of a capacitive sensor based on the fact that it consists of "*electrical elements that store energy in the form of an electrical field. This energy is supplied to the capacitor by a power supply and can be transformed into a useful reading with the appropriate circuitry*". Most capacitors consist of two metal plates that are oppositely charged. The amount of electrical charge that can be stored by the capacitor

depends on the size of the metal plates, and the distance between them (apart from the dielectric constant of the free space and the used material). If used for measuring pressure, in most cases, the change in the distance between the plates is used to calculate the exerted pressure [54]. The errors are extremely small because the capacitive sensor does not depend on the conductivity of its plates and the dimensions of the plates are not dependent on the temperature [55]. In relation to the measured pressure, a capacitive sensor measures the average pressure over the sensor area. This makes the size of the sensor an important characteristic. The larger the sensors the smaller are the areas of high pressure that are detectable. Examples of companies that manufacture measuring systems with capacitive sensors are Novel, with the Pliance® sensor and Crown Therapeutics with the Xsensor® sensor.

b. Resistive sensors

The working principle of this sensor is based on the change in the resistance of a special piezo-resistive layer when a force is applied [56]. This resistive layer contains strain gauges or force-sensing resistors that can map the applied force and translate this into a pressure reading. The pressure reading does not change as long as the same pressure is exerted on the sensor. It will only change when the applied force is changed or removed. Since the piezo-resistive sensor changes only its resistance when deformed, extremely small displacements can be measured [54]. Care must be taken because temperature and humidity affect the sensor reading.[54, 56]. It is important to know is that resistive sensors measure the peak pressure over the sensor area for measuring the pressure at the interface of the body and mattress. Examples of companies that manufacture resistive sensor technology are Vista Medical, with the Force Sensor Array® (FSA) and Clinseat with the Tekscan® sensors.

A comparison of capacitive and resistive sensors

Ashruf [56] stated that most suppliers (and probably users) favor the piezo-resistive sensor because it is fairly straightforward, fast, relatively simple and has a low sensitivity to electromagnetic fields. This is in contrast to capacitive sensors which are technologically more complex. Ashruf [56] further stated that some experts preferred the capacitive system as they found the disadvantages of the resistive sensors (non-linearity, influence of temperature, humidity and poor stability) more relevant than the more complicated electronics. The problem of non-linearity of resistive sensors was investigated by Ferguson-Pell and Cardi [57]. The resistive sensors FSA® and Tekscan® had a linear response (r > 0.99) to increasing pressure (0-160 mmHg in steps of 20 mmHg). Both types of sensors clearly have their pros and cons. Both types of sensors may be used for measuring the pressure at the interface of body and the mattress provided that the investigators are aware of the limitations of each type of the sensor.

Important sensor properties

Independent of the type of sensor that is used for measuring the pressure, there are a number of factors that need to be taken into account.

Hysteresis is the phenomenon that becomes manifest when the output of a sensor responds differently to an increasing load when compared with a decreasing load. Hysteresis is calculated as the maximum difference in measured average pressure as a percentage of the applied pressure at the point of the maximum difference [54]. *Creep or instability* is another source of error when the measured pressure does not remain at the same level over time when a constant load is applied to the sensor [54].

Ferguson-Pell and Cardi [57] investigated the hysteresis and the creep of the FSA®, Tekscan® and Talley Pressure Monitor III (TPM). During the investigation they observed high hysteresis for the FSA® (18.7%) and Tekscan® (21.7%) sensors. Creep was also reported to be high for both sensor systems. Tekscan® had an instability of 17.9% after 2 minutes and 26% after 10 minutes with a load of 50 mmHg. For a 100 mmHg load, this was 7.5% and 13.5% at an interval of 2 and 5 minutes, respectively. The FSA® sensor produced a creep of 3.3% and 4.6% instability at 50 mmHg. For the 100 mmHg this was reduced to 2.2 % and 7.6%, respectively.

Since then, the technology has improved and the software programming has advanced. A more recent evaluation of the three pressure mapping systems (Xsensor®, FSA® and Tekscan®) was carried out by Ferguson-Pell *et al.* [58, 59]. They observed a hysteresis of less than 5% for the FSA® and the Xsensor® with a reproducibility of less than 4% when tested at 100 and 150 mmHg. The Tekscan® showed a significantly higher hysteresis (17.3% at 100 mmHg). The stability (1.8%) of the FSA® was also better at 100 mmHg than that of the Tekscan®, which remained at 20%. The stability of the Xsensor® was 2.8%. The possible explanation for this, according the investigators, was the fact that calibration was carried out with the large air bladder of the FSA® calibration rig. When the whole procedure was carried out with a smaller air bladder, the Tekscan® sensor showed a much better performance. Unfortunately, they did not present the results of this investigation.

Another important sensor property when measuring the pressure at the interface of the body and the mattress is the *flexibility* of the sensor [60]. Inflexible sensors cause a "hammock" effect by preventing the body from sinking in the mattress, thus not following the contour of the body at a specific location. Under these circumstances, the sensor is "carrying" the body and not the mattress. The *thickness* of the sensor also influences the reliability of the pressure reading. Thick sensors can act as a "pebble in your shoe" creating an "unrealistic" high pressure compared with that in the absence of that sensor. In this respect, Ferguson- Pell [60] advised a maximum sensor thickness of 0.5 mm. The maximum diameter together with the thickness of the sensor is also important. Large inflated sensors may act as an additional mattress and change the interface between the body and the

mattress [61]. Ferguson-Pell [60] suggested a diameter of 14 mm for the sensors for measuring the peak pressure at specific sites.

Generally speaking, placing a sensor at the interface of the body and the mattress always affects the pressure measurement [62, 63]. The aim is to minimize this influence. Reddy and Cochran [20] investigated the influence of the actual sensor at the level of the interface pressure. They used 4 sensors (2 electro-pneumatic, 2 piezo-resistive) and placed them between two square blocks of soft material in which the top one (polyvinyl chloride) represented the body and the bottom one the cushion material. A known pressure was applied to the tip of the square blocks. The result was that all sensors produced significantly higher readings compared with the pressure that was applied to the blocks. They repeated the experiment and measured the interface pressure with the 4 sensors under the human thigh and compared this with the subcutaneous pressure measured with a wick catheter in the thigh at the same site. They observed that the interface measurements with the two pneumatic sensors correlated the best with the subcutaneously measured pressure.

The TPM III was used for measuring the exerted pressure in the studies described in this thesis. This decision was mainly motivated by the fact that Hobson [64] and Goossens *et al.* [65] used the device successfully for measuring the interface pressure in patients confined to a wheelchair. Last but not least, the equipment was readily available at the hospital. As the sensors were meant for use in a clinical environment, hygiene and easy positioning of the sensors in patients were also aspects that worked well in the TPM III. The fact that it ran on batteries instead off the main electrical circuit of 220 volt made the transfer to the operating rooms possible in order to carry out the measurements there.

Analysis of the interface pressure (IP) measurements

There are various ways to analyze the measured IP. Much depends on whether the collected measurements are needed to assess a current clinical case or for research purposes in which case statistical analysis was required. In this thesis, the IP generated on the dermal tissues of patients while lying on hospital mattresses was measured. Such measurements are used in many other situations for clinical and research purposes, but the methods of analysis are often not discussed.

The maximum IP as the primary outcome parameter in this area of research is used in many studies. This use is based on the assumption that the maximum IP is ultimately responsible for the development of a PU. Measurements are often carried out at predefined body sites such as the sacrum, the heels etc. as these sites are particularly vulnerable for developing PUs. In doing so, the maximum IP that is measured reflects the pressure at that particular body site and not the whole body.

The IP measurements at these sites are carried out using a single sensor or a sensor array. Using a single sensor may have the disadvantage of not being completely sure of measuring at the target site that generates the maximum pressure. Therefore, describing where and how the sensor is positioned becomes important for reproducible measurements.

Most investigators carry out a number of measurements varying from three to eight using single sensors. Repositioning the sensors between the measurements was sometimes carried out [53, 66-68]. In most cases an average of several measurements was used for statistical analysis [53, 66-73].

The disadvantage of a single sensor may be overcome by using an array of sensors that covers the target area. Two main types of IP measuring devices namely the Mini Texas Interface Pressure Evaluator (Mini TIPE) and the TPM (version MKII) are frequently mentioned in the literature. The Mini TIPE is a 5.5 x 5.5 inch bladder with 16 inflatable sections containing electro-pneumatic sensors. The TPM MKII has 12 sensors with a diameter of 20 mm distributed over an area of 105 x 140 mm and positioned in an array of 3x4 sensors. Positioning the array over the area of interest makes measuring the maximum pressure at the particular site more reliable. The method of analysing these values becomes important because during a single measurement 16 and 12 IP values are retrospectively recorded (frame). Averaging the values disguises the maximum IP at the site of interest [50, 51]. Using only the maximum value out of each frame seems to be the best way for selecting the point of maximum IP at the site of interest. A complicating factor in this is that often in one volunteer / patient a number of frames are generated within a specific time. This results in each sensor having more than one IP value recorded during the time of measurement. Analyzing the obtained IP values may be carried out according to the different strategies.

The simplest way is to take the maximum measured IP value of each frame and use the average values for analysis [74]. It is possible that the maximum IP is measured by different sensors of the sensor array over the different frames because of the movement of the volunteer or the patient. The maximum IP value may also be influenced by unreliable sensor measurements due to connection / measurement errors and thus in the end influence the average maximum IP values of the frames. Theoretically, the site of interest cannot change if the volunteer or the patient and sensor array do not move significantly. Therefore, changes in the location of the sensor that measures the maximum IP in an array over the different frames may be regarded as variation in the sensitivity of the sensor. In view of this, the most reliable way to locate the sensor with the maximum IP pressure is as follows:

1. Calculate the mean, maximum and standard deviation of the IP values measured by each separate sensor in the array over the number of measured frames
2. Delete the measurements of the sensors, which measured the maximum IP for that particular measuring device over each frame
3. Locate the sensor with the maximum mean IP (if there are two sensors with the same mean IP value, choose the one with the lowest standard deviation)

The maximum mean IP for each site of interest over which an array is positioned is thus established.

Another method for measuring the IP is with a pressure mapping array [75, 76]. This is a mat, like the FSA®, containing a large number of electronic pressure sensors. The IP measured with a pressure mapping array is not associated with a specific body site, but provides an overview of the measured IP over a large area of the body. The maximum IP

that is measured is used for the analysis irrespective of the site of interest.

Other ways to analyze the measured IP is by calculating the mean pressure at the site of interest. More complex methods that were studied involved the calculation of the tissue deformation index [77], various pressure indexes [62, 78], the pressure impulse [79] and the pressure relief index [80]. The pressure relief index is often used for comparing different mattresses that operate according an alternating working principle.

1.3   Risk assessment for developing a PU

The tolerance of dermal tissues to withstand the adverse effects of the exerted pressure ultimately determines the speed with which a PU develops. The "incubation period" (the period between the moment pressure is exerted on the tissues and the moment the PU grade I is visible) in patients in whom the dermal tissues are healthier is longer than that in patients whose dermal tissues are more susceptible under the same pressure. Risk assessment tools have been developed specifically in order to assess the tolerance of the dermal tissues for the development of a PU. These tools consist of a large number of factors that are subdivided into levels of severity, if present, increases the risk of developing a PU. The purpose of the risk assessment tools is to determine the level of risk a patient has for developing a PU at any time during hospitalization [81].

Gosnell [82] reported 126 items that were already identified as risk factors in different risk assessment scales. However, it is impractical to create a risk assessment tool consisting of all 126 items. The first PU risk assessment tool was developed by Norton, Mclaren and Exton-Smith [83]. This tool consisted of 5 items that were subdivided into 4 levels of severity. Many other risk assessment tools have been developed since then. Currently, more than 40 PU risk assessment tools are described in the literature [84]. Therefore, we can choose a tool in which the majority of the risk factors are included in relation to the population of interest.

In the literature, these tools are regarded as diagnostic tests and are evaluated accordingly. Basically, diagnostic tests provide the result of an investigation and the correct diagnoses based on necropsy, biopsy or surgical inspection [85]. A medical example in this area is the ability of a liver scan to predict liver pathology compared with a liver biopsy. The ability of a scan to detect pathological changes is evaluated by calculating the sensitivity (the proportion of true positives that are correctly identified by the tool) and the specificity (the proportion of true negatives that are correctly identified by the tool). Based on these data the predictive value of both the proportions can be calculated.

The difference between a PU risk assessment tool and a liver scan is that the latter visualises the abnormality that *is present* without any invasive intervention. Using a PU assessment tool one can assess the risk of developing a PU in the (near) future. Rightly so, Halfens [86] stated that measuring the risk of developing a PU is not the same as the actual development of a PU. Using the diagnostic test approach for evaluating the reliability of PU

risk assessment tools, every preventive measure taken during the time of hospitalization is bypassed. Physicians and nurses always wishfully thought to try and prevent PU development by starting preventive measures, especially in patients at risk (according to the risk assessment tool) for developing PUs. Thus, the quality of care during the "incubation period" may ultimately prevent the PU from developing, although the instrument "predicted" the development of a PU. In relation to this, De Laat [81] suggested that a low specificity in combination with a high sensitivity possibly indicates that adequate preventive measures have been taken.

As more awareness develops among researchers that risk assessment tools are recommended, and should be used, it is essential that these become a part of a more general PU prevention program and their findings should be combined with the clinical judgement of the nurse [86]. Otherwise, using a risk assessment tool for predicting the development of a PU may only be compared with flipping a coin [87].

Which other method may be used if using a diagnostic test approach is not the best method to evaluate the value of a PU risk assessment tool? Halfens [86] suggested to calculate the odds-ratios for each risk score. This would provide the nurses with the percentage-wise insight into the risk a patient has for developing a PU. Since the number of risk factors will change over time, calculating the odds-ratios does not provide the insight if the daily risk for developing a PU increases or decreases over time. As time is an important variable in the development of PUs, it would be essential to know the risk the patients have for developing a PU upon hospital admission. This may be achieved by using actuarial statistical methods like the Kaplan-Meier curves. In order to do so, daily risk assessment should be carried out in all patients during a prolonged period. Next to the risk assessment, information on the development of a PU and the type and the frequency of preventive interventions should also be collected. When the data of between 400 and 500 patients are collected (based on an expected prevalence of 25% and confidence interval of 4% or less) survival curves may inform the nurses and the physicians on the risk a patient faces for developing a PU at any given moment during hospitalization.

Type of PU risk assessment tools

The majority of the risk assessment tools were not designed with any particular patient group in mind. The three most used and tested tools are the Norton [83], the Braden [88] and the Waterlow [89]. All three tools have been tested in different patient groups with varying results.

The patients in the intensive care unit are at increased risk for developing PUs. In a Dutch prevalence study, a total of 850 intensive care patients (423 in 1998 and 427 in 1999) were assessed. Patients from surgical, medical, cardiac and pediatric intensive care units were included. The prevalence of PU in that study was 28.7 %. If the stage I PUs were excluded, the prevalence was 18.2 % indicating that one in every 5 patients had signs of damaged dermal tissue [90]. Other studies reported similar results. Carlson, Kemp and

Shott [91] measured a prevalence of 12.5% in a general intensive care unit with a mixed patient group. Marchette, Arnell and Redick [92] reported a prevalence of 40 % in a surgical intensive care unit and Fife *et al.* [93] reported a prevalence of 12.4 % in a neurological intensive care unit.

Specific risk factors in intensive care patients

A literature review by De Laat [81], highlighted several specific risk factors in intensive care patients. The healthcare status of the patient [94] and the APACHE score [91, 95, 96] are important parameters upon admission in the intensive care unit. Patients who have a compromised health in combination with a high APACHE score are more at risk for developing a PU.

The use of specific groups of medication increases the risk for developing a PU. In particular treatment with sympathomimetics [97, 98] and sedatives [91, 94] increases the risk for developing a PU. Nutritional status of the intensive care patients is another important factor. Since a number of intensive care patients are in a catabolic state, it is imperative that the patients' anabolic state is restored via an effective feeding regime [92, 95].

At the level of preventive interventions, it is not always possible to implement the PU preventive measures such as turning the patient because of respiratory and hemodynamic instability. Therefore, it is essential to use pressure-reducing mattresses. The time interval between ordering and installing a pressure-reducing mattress may be critical because most of the mattresses are rented. It must be also said that some patients are so critical that even changing the type of mattress is not an option. Other important risk factors that have been identified in this group of patients through other research are the presence of infection [90], the presence of moisture [90], the impaired mobility [90], old age [90, 99], prolonged hospitalization [90], emergency admission [99], number of days without nutrition [99] and a low Body Mass Index [93].

Risk assessment instruments for patients in the intensive care unit

Solars [100] reported that not all PU risk scales are suitable for patients in the intensive care unit. It is essential that such scales should include some of the most important risk factors that are mentioned in the paragraph. A number of risk assessment tools for intensive care patients are reported in the literature. Jackson and Cubin developed the original[98] and the revised version [101] of the pressure area risk calculator.

Barret [102] suggested that the Waterlow risk assessment tool could best be used for patients in the intensive care unit as it included a number of specific risk factors. Waterlow's idea was not to create a cut-off point with her tool, but to let it function as a clinical warning device [103]. The disadvantage of working with a cut-off point is that the difference between the high and the low risks is just one point [97, 103]. Therefore, she created risk groups based on the number of points calculated with the tool. Three groups were identified as low risk, moderate risk and high risk. Since the quality of the basic hospital

mattress and the standard prevention strategies for PUs affect the range of the Waterlow risk assessment tool namely low, moderate and high risk, the outcome in assessing the value of such a tool may vary between hospitals. This was also acknowledged by Braden *et al.* [88]. Therefore, the usefulness of the tool may vary between hospitals and even intensive care units. Personally, it acknowledges the opinion that using a risk assessment scale for predicting the development of a PU may only be seen as effective if used in combination with the clinical judgment of the nurse and appropriate PU preventive strategies are implemented [81, 86]. Taking this into account, we used the Waterlow risk assessment scale in the studies described here because it includes most of the factors that are relevant for intensive care patients (see Appendix 1).

Literature

1. Newton I. Principia Mathematica Philosophia Naturalis. 1687.
2. Braden BJ and Bergstrom N. A conceptual scheme for the study of the etiology of pressure sores. Rehabil Nurs 1987;12:8-12,6.
3. Defloor T. The risk of pressure sores: a conceptual scheme. J Clin Nurs 1999;8:206-16.
4. Bosboom EM, Bouten CV, Oomens CW, Baaijens FP and Nicolay K. Quantifying pressure sore-related muscle damage using high-resolution MRI. J Appl Physiol 2003;95:2235-40.
5. Bosboom EMH, Oomens CWJ, Bouten CVC, Kuipers H and Baaijens FPT, Deformation as a trigger for pressure sore related muscle damage Eindhoven, Technical University: Biomedical Technology, 2001.
6. Lewis T and Grant R. Observations upon reactive hyperemia in man. Heart 1925;12:73-120.
7. Collier M. Blanching and non-blanching hyperaemia. J Wound Care 1999;8:63-4.
8. van Marum RJ, Schut G, Ribbe MW and Meijer JH. Pathofysiologie en klinische kenmerken van decubitus. Ned Tijdsch Geneesk 1994;1994:66-71.
9. Xakellis GC, Frantz RA, Arteaga M and Meletiou S. Dermal blood flow response to constant pressure in healthy older and younger subjects. J Gerontol 1993;48:M6-9.
10. Thorfin JS, F. Lidman, D. Sitting pressure and perfusion of buttock skin in paraplegic and tetraplegic patients, and healthy subjects: a comparative study. Scand J Plast Reconstr Surg Hand Surg 2002;36:279-83.
11. van Marum RJ, The risk of developing decubitus ulcers (PhD thesis) Amsterdam, Free University: Faculty of Medicine, 1998.
12. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40:61-9.
13. Blair DA, Glover WE and Rodie IC. The abolition of reactive and post hyperaemia in the forearm by temporary restriction of arterial inflow. J. Physiol 1959;148:648.
14. Chakraborti S, Mandal M, Das S, Mandal A and Chakraborti T. Regulation of matrix metalloproteinases: an overview. Mol Cell Biochem 2003;253:269-85.
15. Crenshaw RP and Vistnes LM. A decade of pressure sore research: 1977-1987. J Rehabil Res Dev 1989;26:63-74.
16. Kosiak M. Etiology of decubitus ulcers. Arch Phys Med Rehabil 1961;42:19-29.
17. Dinsdale SM. Decubitus ulcers: role of pressure and friction in causation. Arch Phys Med Rehabil 1974;55:147-52.
18. Daniel RK, Priest DL and Wheatley DC. Etiologic factors in pressure sores: an experimental model. Arch Phys Med Rehabil 1982;62:492-8.
19. Bar CA, The response of tissues to applied pressure [dissertation] Cardiff (UK), University of Wales: College of Medicine, 1988.

20. Reddy NP and Cochran GV. Interstitial fluid flow as a factor in decubitus ulcer formation. J Biomech 1981;14:879-81.

21. Miller GE and Seale J. Lymphatic clearence during compressive loading. Lymphology 1981;14:161-6.

22. Krouskop TA. A synthesis of the factors that contribute to pressure sore formation. Med Hypothesis 1983;4:255-67.

23. Barbanel JC. Pressure management. Prosthet Orthot Int 1991;15:231.

24. Pierce SM, Skalak TC and Rodeheaver GT. Ischemia reperfusion injury in chronic pressure ulcer formation: a skin model in the rat. Wound Repair Regen 2000;8:68-76.

25. Herrman EC, Knapp CF, Donofrio JC and Salcido R. Skin perfusion responses to surface pressure-induced ischemia: implication for the developing pressure ulcer. J Rehabil Res Dev 1999;36:109-20.

26. Ryan TJ, Cellular responses to tissue distorsion, in Pressure sores: clinical practice and scientific approach, DL Bader, Editor. 1990, McMillan Press: London. p. 141-52.

27. Bouten CV, Oomens CW, Baaijens FP and Bader DL. The etiology of pressure ulcers: skin deep or muscle bound? Arch Phys Med Rehabil 2003;84:616-9.

28. Gunning-Scheper LJ, Wendte JF and Welleman E, Decubitus in Nederland: een onderzoek naar de mogelijkheden om het voorkomen van decubitus te meten. Amsterdam, University of Amsterdam: Department of Social Epidemiology, 1993.

29. Yarkony GM, Matthews Kirk P, Carlson C, et al. Classification of pressure ulcers. Arch Dermatol 1990;126:1218-9.

30. Haalboom JRE, Everdingen JJE and Cullum N, Incidence, prevalence and classification, in The decubitus ulcer in clinical practice, LCW Parish, J.A. and Crissey J.T., Editor. 1997, Springer-Verlag Berlin Heidelberg: New York. p. 12-23.

31. Harker J. Pressure ulcer classification. J Wound Care 2000;9:275-7.

32. Shea JD. Pressure sores classification and management. Clin Orthop 1975;112:89-100.

33. Torrance C, Pressure sores: aetiolgie, treatment and prevention. 1983, London: Croom Helm.

34. Witkowski JA and Parisch LC. Classification of pressure ulcers. Arch Dermatol 1991;127:907-8.

35. Gyi DE, Porter JM and Robertson NKB. Seat pressure measurement technologies: considerations for their evaluation. Appl Ergon 1997;27:85-91.

36. Treaster D. Measurement of seat distributions. Human Factors 1987;29:563-75.

37. Porter JM, Gyi DE and Tait HA. Interface pressure data and the prediction of driver discomfort in road trials. Appl Ergon 2003;34:207-14.

38. Kosiak M, Kubicek W, Olson M, Danz JN and Kottke W. Evaluation of pressure as a factor in the production of Ischial Ulcers. Arch Phys Med Rehabil

1958;39:623.

39.   Lindan O, Greenway RM and Piazza JM. Pressure distribution on the surface of the human body: I. Evaluation in lying and sitting positions using A "bed of springs and nails". Arch Phys Med Rehabil 1965;5:378-85.

40.   Bush CA. Study of pressures on skin under ischial tuberosities, and thights during sitting. Arch Phys Med Rehabil 1969;4:207-13.

41.   Hertzberg HTE, The human buttocks in sitting: Pressures, patterns and palliatives. 1972, Wright-Patterson Air Force Base, OH: Aerospace Medical research laboratory.

42.   Holley LK, Long J, Stewart J and Jones RF. A new pressure measuring system for cushions and beds-with a review of the literature. Paraplegia 1979;17:461-74.

43.   Drummond DS, Narechania RG, Rosenthal AN, *et al.* Pressure distributions measured during balanced and unbalanced sitting. J Bone Joint Surg 1982;64A:1034-9.

44.   Fisher SV and Patterson P. Long-term pressure recordings under the ischial tuberosities of tetraplegics. Paraplegia 1983;23:99-106.

45.   Robertson JC, Shah J, Amos H, Druett JE and Gisby J. An interface pressure sensor for routine clinical use. Eng Med 1980;9:151-6.

46.   Garber SL, Krouskop TA and Carter RE. A system for clinically evaluating wheelchair pressure relief cushions. Am J Occup Ther 1978;32:565-70.

47.   Bader DL. Interface pressure measurement. Care Sci Prac 1982;1:22-4.

48.   Krouskop TA, Noble PS, Brown J and Marburger R. Factors affecting the pressure-distributing properties of foam mattress overlays. J Rehabil Res Dev 1986;23:33-9.

49.   Krouskop TA, Randall C, Davis J, *et al.* Evaluating the long-term performance of a foam-core hospital replacement mattress. J Wound Ostomy Continence Nurs 1994;21:241-6.

50.   Suarez CH and Reynolds A. Pressure reduction with a hospitalized population using a mattress overlay. Ostomy Wound Manage 1995;41:58-60, 2-3.

51.   Rojas MG and Reynolds A. Pressure-reducing capability of the Therarest hospital replacement mattress. J Wound Ostomy Continence Nurs 1996;23:100-4.

52.   Oertwich PA, Kindschuh AM and Bergstrom N. The effects of small shifts in body weight on blood flow and interface pressure. Res Nurs Health 1995;18:481-8.

53.   Sideranko S, Quinn A, Burns K and Froman RD. Effects of position and mattress overlay on sacral and heel pressures in a clinical population. Res Nurs Health 1992;15:245-51.

54.   Bethaves T. Interface pressure measurement: testing and selecting sensors. J Wound Care 2003;11:325-9.

55.   Geddes LA and Baker LE, Principles of Applied Biomedical Instrumentation. 1975, New York: John Wiley & Sons.

56.   Ashruf CMA. Thin flexible pressure sensors. Sensor Rev 2002;22:322-7.

57. Ferguson-Pell MW and Cardi MD. Prototype development and comparative evaluation of wheelchair pressure mapping system. Assist Technol 1993;5:78-91.

58. Ferguson-Pell MW, Nicholson G, Lennon P and Bain D, Comparative evaluation of pressure mapping systems (1): bench testing results, in RESNA handbook. 2001, RESNA.

59. Ferguson-Pell MW, Nicholson G, Lennon P and Bain D, Comparative evaluation of pressure mapping systems (2): cushing testing, in RESNA handbook. 2001, RESNA.

60. Ferguson-Pell MW. Design criteria for the measurement of pressure at the body/support interfaces. Eng Med 1980;9:209-14.

61. Swain IDB, D.L. The measurement of interface pressure and its role in soft tissue breakdown. J Tissue Viability 2002;12:132-46.

62. Shelton F, Barnett R and Meyer E. Full-body interface pressure testing as a method for performance evaluation of clinical support surfaces. Appl Ergon 1998;29:491-7.

63. Allen V, Ryan DW, Lomax N and Murray A. Accuracy of interface pressure measurement systems. J Biomed Eng 1993;15:344-8.

64. Hobson DA. Comparative effects of posture on pressure and shear at the body-seat interface. J Rehabil Res Dev 1992;29:21-31.

65. Goossens RHM, Groeneveld WH and Snijders CJ. Assessment of decubitus risk in a test circuit using different wheelchair cushions. J Rehabil Sci 1993;6:8-12.

66. Ryan DW, Allen V and Murray A. An investigation of interface pressures in low air loss beds. Int J Clin Pract 1997;51:296-8.

67. Flemister B. A pilot study of interface pressure with heel proctectors used for pressure reduction. J ET Nurs 1991;158-61.

68. Allen V, Ryan DW and Murray A. Measurements of interface pressure between body sites and the surfaces of four specialised air mattresses. Br J Clin Pract 1994;48:125-9.

69. Weaver V and Jester J. A clinical tool: updated readings on tissue interface pressures. Ostomy Wound Manage 1994;40:34-6, 8, 40 passim.

70. Thompson-Bishop JY and Mottola CM. Tissue interface pressure and estimated subcutaneous pressures of 11 different pressure-reducing support surfaces. Decubitus 1992;5:42-6, 8.

71. Wells JA and Karr D. Interface pressure, wound healing, and satisfaction in the evaluation of a non-powered fluid mattress. Ostomy Wound Manage 1998;44:38-42, 4-6, 8 passim.

72. Fontaine R, Risley S and Castellino R. A quantitative analysis of pressure and shear in the effectiveness of support surfaces. J Wound Ostomy Continence Nurs 1998;25:233-9.

73. Counsell C, Seymour S, Guin P and Hudson A. Interface skin pressures on four pressure-relieving devices. J Enterostomal Ther 1990;17:150-3.

74.   Holscher TG, Goossens RHM, Snijders CJ, Nieuwenhuis FJM and Joziasse F. A new low-cost anti-decubitus mattress for home care; a comparison with other matresses. J Rehabil Sciences 1995;8:72-5.

75.   Scott EM, Baker EA, Kelly PJ, Stoddard EJ and Leaper DJ. Measurement of interface pressures in the evaluation of operating theatre mattresses. J Wound Care 1999;8:437-41.

76.   Defloor T and Grypdonck MH. Sitting posture and prevention of pressure ulcers. Appl Nurs Res 1999;12:136-42.

77.   Neumark OW. Deformation, not pressure, is the prime cause of pressure sores. Care Sci Prac 1981;1:41-6.

78.   Patel UH, Jones JT, Babbs CF, Bourland JD and Graber GP. The evaluation of five specialized support surfaces by use of a pressure-sensitive mat. Decubitus 1993;6:28-31, 4, 6-7.

79.   Clark M. Continuous interface pressure measurement using an electropneumatic sensor: the scp monitor. Care Sci Prac 1987;june:5-8.

80.   Rithalia SV and Gonsalkorale M. Assessment of alternating air mattresses using a time-based interface pressure threshold technique. J Rehabil Res Dev 1998;35:225-30.

81.   De Laat E. [Pressure sores in ICU patients--a literature review] Drukletsel bij IC-patienten--een literatuuronderzoek. Verpleegkunde 1997;12:4-14.

82.   Gosnell DJ. Pressure ulcer risk assessment: tools, techniques and targets. in 10th Annual Clinical Symposium on Wound Management. Minneapolis, Minn.

83.   Norton RM, McLaren R and Exton-Smith AN, An investigation of geriatric nursing problems in hospitals. Vol. 2nd Ed. 1975, Edinburgh: Churchill Livingstone. 193-238.

84.   Nixon J and McGough A, Principles of patient assessment: screening for pressure ulcers and potentional risk., in The prevention and treatment of pressure ulcers, M Morison, Editor. 2001, Mosby. p. 55-74.

85.   Altman DG and Bland JM. Diagnostic tests. 1: Sensitivity and specificity. Br Med J 1994;308:1552.

86.   Halfens RJ. Risk assessment scales for pressure ulcers: a theoretical, methodological, and clinical perspective. Ostomy Wound Manage 2000;46:36-40, 2-4.

87.   Schoonhoven L, Haalboom JRE, Bousema MT, et al. Prospective cohort study of routine use of risk assessment scales for the prediction of pressure ulcers. Br Med J 2002;325:797-800.

88.   Bergstrom N, Braden BJ, Laguzza A and Holman V. The Braden Scale for Predicting Pressure Sore Risk. Nurs Res 1987;36:205-10.

89.   Waterlow JA. Pressure sores: a risk assessment card. Nurs Times 1985;81:49-55.

90.   Bours GJ, De Laat E, Halfens RJ and Lubbers M. Prevalence, risk factors and prevention of pressure ulcers in Dutch intensive care units. Results of a cross-

sectional survey. J Intensive Care Med 2001;27:1599-605.

91.   Carlson EV, Kemp MG and Shott S. Predicting the risk of pressure ulcers in critically ill patients. Am J Crit Care 1999;8:262-9.

92.   Marchette L, Arnell I and Redick E. Skin ulcers of elderly surgical patients in critical care units. Dimens Crit Care Nurs 1991;10:321-9.

93.   Fife C, Otto G, Capsuto EG, *et al.* Incidence of pressure ulcers in a neurologic intensive care unit. Crit Care Med 2001;29:283-90.

94.   CBO. Centraal Begeleidingsorgaan voor de Intercollegiale Toetsing: Consensus bijeenkomst decubitus preventie. in Consensus -bijeenkomst decubitus preventie. 1985. Utrecht.

95.   Inman KJ, Sibbald WJ, Rutledge FS and Clark BJ. Clinical utility and cost-effectiveness of an air suspension bed in the prevention of pressure ulcers [see comments]. JAMA 1993;269:1139-43.

96.   Clough N. The cost of pressure area management in an intensive care unit. J Wound Care 1994;33-5.

97.   Hunt J. Application of a pressure area risk calculator in an intensive care unit. Intensive Crit Care Nurs 1993;9:226-31.

98.   Cubbin B and Jackson C. Trial of a pressure area risk calculator for intensive therapy patients. Intensive Care Nurs 1991;7:40-4.

99.   Eachempati SR, Hydo LJ and Barie PS. Factors influencing the development of decubitus ulcers in critically ill surgical patients. Crit Care Med 2001;29:1678-82.

100.   Sollars A. Pressure area risk assessment in intensive care. Nurs Crit Care 1998;3:267-73.

101.   Jackson C. The revised Jackson/Cubbin Pressure Area Risk Calculator. Intensive Crit Care Nurs 1999;15:169-75.

102.   Barratt E. Pressure sores in intensive care. Intensive Ther Clin Monitoring 1990;158-67.

103.   Waterlow JA. Reliability of the Waterlow score [letter; comment]. J Wound Care 1995;4:474-5.

Chapter 2

Pressure sores in an intensive care unit and related
variables: a descriptive study

Jan T.M. Weststrate [1]

Hajo A. Bruining [1]

1.   Surgical intensive care unit, Erasmus MC, University Medical Center, Rotterdam

Abstract

The development of pressure sores is associated with negative patient outcome. Patients in an intensive care unit (ICU) are particularly prone to develop pressure sores caused by severe illness and being immobile for long periods. A method to prevent the development of pressure sores is to use the Waterlow pressure sore risk calculator as a warning device, followed by appropriate action.

A prevalence study on pressure sores was conducted in the ICU, where the following variables were noted for each patient admitted: pressure sore stage on the sacrum or the buttocks, the Waterlow pressure sore risk score, and the number and the kind of preventive measures taken by the nursing staff.

The results show that the prevalence of pressure sore varies greatly over the study period, that a relationship exists between the pressure sore stage and the risk of developing a pressure sore and that nurses are more motivated to take pressure sore precautions based on the visible damage due to pressure than by the warning provided by the presence of specific risk factors.

It is concluded that point prevalence measurement does not give reliable information about the pressure sore problem in general in an ICU and that daily measurement of the risk of developing pressure sores with the Waterlow pressure sore risk calculator helps nurses to indicate specific risk factors and assists them with decisions on the frequency and method of pressure sore precautions to be taken.

Introduction

Haalboom and Bakker [1] estimated that in The Netherlands on average 6-10% of all hospitalized patients suffer from pressure ulcer (PU) problems and that the calculated yearly costs of prevention and treating pressure ulcers (PUs) amounted to D.Fl 700,000,000. The development of PUs is associated with negative patient effects such as pain, depression, loss of function and independence, increased incidence of infection, sepsis, additional surgical procedures, all resulting in a prolonged hospital stay [2]. The most vulnerable categories are: 1) patients with spinal cord injuries; 2) geriatric patients; 3) hospitalized patients, especially those who have undergone orthopedic surgery; and 4) patients who need to be admitted to the intensive care unit (ICU) [3]. The last patient group especially has a high risk of developing PUs, as ICU patients are often immobile for long periods of time and have an elevated Acute Physiology And Chronic Health Evaluation (APACHE) score indicating an increased severity of illness [3]. Bergstrom *et al.* [4] documented that 40% of ICU patients develop PUs. A point prevalence study performed in the University hospital of Amsterdam, The Netherlands showed that 43% of adult intensive care patients had a PU problem [5]. The workload of the nursing staff is increased by 50% once a patient has developed a PU[6], so prevention of PUs is of primary importance to the patient and has a secondary economic benefit for the health system. Prevention is the domain of nursing staff [7]. This involves promoting patient care that prevents physical, psychological and social deterioration of the patient's current health status. Therefore, planning care that prevents the development of PUs falls directly under the responsibility of the nursing staff.

The development of a PU is a product of *time* and *pressure* in combination with a number of predisposing *intrinsic and extrinsic factors* [6]. Kosiak [8] found that PUs develop between 1-5 days after pressure is applied to the skin with a severity depending on the force and duration of the applied pressure. In order to prevent PUs it is important that at least one of these components is reduced or eliminated. The factors of *time* and *pressure* are effectively influenced by turning the patient regularly on one side for a short time (< 5 minutes) or nursing the patient for longer time on alternate sides or by using special pressure relieving mattresses.

The influence of the intrinsic and extrinsic factors on the development of PUs is more difficult to assess. Over the last 30 years several instruments have been developed in order to measure the influence of intrinsic and extrinsic factors on the development of PUs [9], of which the Norton Pressure Sore Risk Scale is most well known [10]. For intensive care patients this scale is not applicable because it over-predicts the risk of developing an ulcer [11]. Barrat [6] suggested that the Waterlow Pressure Sore Risk Scale [12] (see Appendix 1) is suitable for patients in an ICU because it incorporates special risk factors related to this patient group. So far no studies have been published in which the Waterlow scale has been evaluated for intensive care patients.

Therefore, a study was designed to investigate the general size of the PU problem in an ICU, the relationship between the Waterlow scale score and the PU stage in ICU patients,

and at what different PU stages various PU precautions are taken by the nursing staff.

Materials and Methods

An indication of the average situation concerning the development of PUs in an ICU can best be performed by a period prevalence study. One investigator screened every possible working day all patients between 08:00 and 10:00 o'clock each morning in the surgical ICU during a period of 5 months. Each screened patient had been admitted before midnight the previous day.

The ICU is divided into a short-stay unit and a long-stay unit. The short-stay unit has 5 beds (mostly postoperative and trauma patients). The average length of stay per patient in this unit is 4.5 days. The long-stay unit has 4 beds occupied by patients with severe infections needing ventilatory support for more than 5 days. The average length of stay per patient in this unit is 12.8 days.

The following variables were noted:

a) The risk of developing a PU, scored using the Waterlow scale [12] (see Appendix 1);
b) The total number of preventive nursing measures used, such as the number of times the patient was turned (every 2 to 3 hours for less than 5 minutes), nursed on alternate sides (nursed for at least 30 minutes on alternate sides), or mobilized out of bed into a chair for at least 30 minutes;
c) The type of mattress used;
d) The stage of the PU on the sacral area or the buttocks, classified according to a scale used by the Dutch National Pressure Ulcer Advisory Panel [1] (see Appendix 2 for the definition).

In this study, a patient was classified as having a PU when stage II was diagnosed. The calculation of the patient's risk of developing a PU was made on the basis of the risk factors that were present at some time during the 24 hours before screening. The number of points scored was unknown to the intensive care nurses. We specified five risk categories of the Waterlow scale in order to make sure that throughout the whole study the same criteria were used. In the category *Incontinence*, fecal incontinence was defined as having two or more defecations in bed within 24 hours. *Mobility* always scored 5 points because intensive care patients spend the majority of time in bed. In the category *Tissue Malnutrition*, cardiac failure was scored when the patient was receiving a catecholamine medication. In the category *Neurological Deficit*, paraplegia was scored when paralyzing drugs such as Pancuroniumbromide or Vecuroniumbromide were administered to the patient. This item was also scored when patients had leg fractures and were therefore immobilized. *Major Surgery* was only scored when it took place within the previous 24 hours.

The total number of nursing precautions taken for PUs was calculated on the basis of 24 hours periods. When a patient remained in the ICU for less than 24 hours the frequency of

the PU precautions was extrapolated for 24 hours. The type of mattress that was used was noted as a low-air loss type mattress/bed or no special mattress.

Statistical analysis was performed with the Pearson Product-moment correlation to investigate a relationship between the measured variables and the PU stage. The statistical difference between the measured variables was analyzed with the student t-test for two independent groups. The level of significance was set at $p < 0.05$.

Results

On 80 days during a period of 18 weeks, all variables were measured for all intensive care patients in both the short-stay and the long-stay unit. All measurements were performed (583 times in total) in 130 patients. On average all measurements were performed 4.5 times in each admitted patient in the ICU. Both units on average were screened every two days, so that patients who stayed for longer than two days were often screened more than once. Table I lists the descriptive data for both units.

Table I. Descriptive data for the short-stay and long-stay units during the study period (± SD).

Descriptive data of sample	Short-stay unit	Long-stay Unit
Number of measurements	317	266
Number of patients	109	21
Age	61.6 (± 16.1)	59.9 (± 17.7)
Average measurements per patient	2.9 (± 3.1)	12.7 (± 8.9)
Average Waterlow scale score	17.5 (± 5.9)	16.2 (± 4.7)
Average number of PU precaution interventions	5.5 (± 1.6)	5.4 (± 1.6)

Figure 1 gives an overview of the PU prevalence in each week during the five months study period in which measurements were done in both units.
Table II gives the prevalence of the number of days the various PU stages were seen on each of the units and the mean Waterlow scale score for the patient with these sores. In the short-stay unit 13.6% of the measurements (43 patient days) showed a PU stage ≥ II. In the long-stay unit this was 42.1% (112 patient days). There was a significant correlation between the Waterlow scale score and the PU stage in both units (short-stay: $r = 0.15$; long-stay: $r = 0.43$). In the short stay-unit the mean risk scores of the various PU stages were not always statistically related. The difference of the Waterlow scale score between stages 0 and III was significant ($p = < 0.001$). It was noteworthy that stages II and IV were occasionally seen during the study. In the long-stay unit a significant difference was found between all Waterlow scale scores of the different PU stages except pressure stage II (2 measurements).
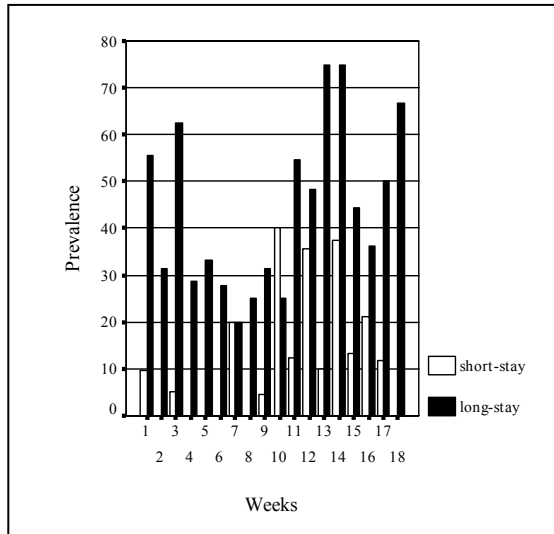
Figure 1. Prevalence of PUs in the short- and long-stay surgical intensive care units

Table II   An overview of the number of measurements for each PU stage with the mean PU risk score (± SD) on the short- and long-stay units.

PU stage	Number of measurements (short-stay)	Waterlow scale score (short-stay)	Number of measurements (long-stay)	Waterlow scale score (long-stay)
Grade 0	233	16.5 (± 5.8) (+)	139	14.3 (± 3.8) (*)(**)(***)
Grade 1	41	18.4 (± 6.1)	15	16.1 (± 4.0) (****)
Grade 2	8	13.9 (± 4.8) (++)	2	21.5 (± 5.5) (*)
Grade 2	33	20.2 (± 6.1) (+) (++)	68	18.1 (± 5.1) (**)
Grade 4	2	14.5 (± 2.5)	42	19.1 (± 3.9) (***)(****)

(+) p = 0.001; (++) p = 0.012(*); p = 0.009; (**) p = 0.000; (***) p = 0.000; (****) p = 0.015

The frequency and the sort of PU precautions is shown in Figure 2. No distinction was made between the data collected in the short- or the long-stay units as these PU precautions were carried out by the same nursing staff who worked in both units. The statistically significant differences between the PU stages and the frequency of the PU precautions taken are shown in Table III. There was a significant negative correlation (r = -0.30) between the PU stage and turning the patient every 2 - 3 hours and a positive correlation between the PU stage and turning on alternate sides (r = 0.28). A significant positive correlation (r = 0.53) was also found between the total number of precautions and the number of times of nursing on alternate sides. This indicates that the increase in the total number of precautions taken was mostly caused by an increase in the number of times a patient was nursed on alternate sides.

In both units special beds were only used when a patient was diagnosed as having a PU of stage III or IV and it was impossible to nurse the patient on alternate sides. The correlation between the PU stage and the use of a special bed was statistically significant (r = 0.60).
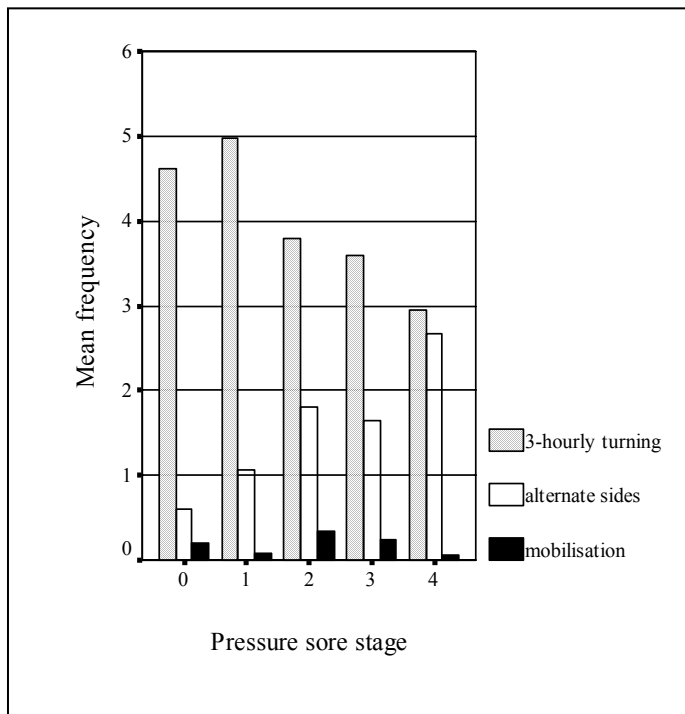

Figure 2. Mean frequency of precautions taken at various PU stages

Table III. An overview of the statistically significant differences (p < 0.05) between the frequencies of the PU precautions taken by the nursing staff at the various PU stages. (Turning = Turning the patient every three hours; NAS = Nursing the patient on Alternate Sides; MOB = Mobilizing the patient Out of Bed)

	Stage 0	Stage 1	Stage 2	Stage 3	Stage 4
Stage 0			NAS	Turning NAS	Turning NAS
Stage 1			MOB	Turning	Turning NAS
Stage 2	NAS	MOB			MOB
Stage 3	Turning NAS	Turning			MOB
Stage 4	Turning NAS	Turning NAS	MOB	MOB	

Discussion

A method of measuring the occurrence of PUs in an ICU is to perform a prevalence study [13]. Prevalence can be defined as: "The number of cases in a population at a particular point in time (point prevalence) or during a specific period (period prevalence)" [14]. There is limited knowledge on the prevalence of PUs in intensive care patients. The reason why prevalence studies have not been carried out in this category of patients may have to do with the opinion that PUs are an indication of "bad nursing" [15]. A point prevalence study may not be the optimum method of PU research in an ICU unit because of the limited number of beds (8-15 beds) and it is a transition unit. The percentage of patients who have a PU problem at one moment in time does not therefore reflect the general extent of the problem. With people moving in and out every day, the percentage of patients with a PU varies from day to day. Our results show (Figure 1) that the percentage of PUs varies significantly over the weeks. Variation in prevalence was also found by Dealey [15] during three different point prevalence studies. In order to achieve an accurate indication of the occurrence of PUs, it would be better to perform frequent point prevalence surveys (for example one day a week) or a period prevalence study over a longer period in these units.

The prevalence of PUs > grade I in the short-stay unit was lower than that in the long-stay unit. The prevalence (13.6%) in the short-stay unit, in terms of percentage of measurements which revealed a PU ≥ stage II, may be influenced by the fact that patients on the average stayed for only 4.5 days before returning to the ward. The long-stay unit had a

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prevalence of (42.1%). Patients in this unit on average stayed for 12.8 days. These results indicated that the risk of developing a PU increases when patients have to stay longer in the ICU.

Whether the Waterlow scale can be effectively used to measure the risk that the patients have of developing a PU cannot be stated definitely. Waterlow [12] suggested that the total Waterlow scale score may consist of all Waterlow scale criteria that are present in the patient. This can cause a large variation in the number of scoring points in each patient between the measurement days. To overcome this in this study the procedure was changed so that out of each category only one criterion was selected and if two or more criteria were applicable the one with the highest score was chosen. To date there have been a limited number of studies in which the Waterlow scale was compared with the PU risk scales such as the Norton Pressure Sore Risk Scale. Hamilton [16] cited three studies, but none of these were performed in an ICU. The studies showed that generally the Waterlow scale was highly sensitive in predicting the risk of PUs compared with the Norton Pressure Sore Risk Scale ,[16] but often at the expense of gross over-prediction [17]. At present no results are available for the sensitivity and the specificity of the Waterlow scale used in an intensive care setting.

To our knowledge there have been no studies that mentioned the number and the type of PU precautions that are carried out in the evaluation of a PU risk scale. This is important because PU precautions influence the development of PUs and influence the accuracy of the used PU risk scale [15]. In this study PU prevention precautions were carried out every 4 - 5 hours. When PUs developed to a more visible stage (> stage I), the nursing staff increased the frequency of nursing the patients on alternate sides without increasing the total number of precautions. This suggested that the nurses were motivated to carry out a more effective PU precaution (nursing on alternate sides) on the basis of visible PU than on the basis of the number of PU risk factors that are present in the patient when PU stage is less visible. This feature can be explained by the nature of work carried out by the intensive care nurses. In general, they act on what they see or monitor. No intensive care nurse will administer a dopamine infusion as a preventive intervention. The beginning of a PU is often an invisible process in the tissues of the risk areas. Daily monitoring of intrinsic and extrinsic factors by intensive care nurses with the Waterlow scale may help them to identify patients who are at risk of developing PUs. Further study is needed to determine if combining specific Waterlow scale scores with a special kind and frequency of preventive measures may prevent PUs.

In general, the results of this study provide enough reason for daily registration of the PU stage, measuring the risk of PUs with the Waterlow scale and recording daily the number and the type of PU precautions taken by the nursing staff. The Waterlow scale score may be used to plan the frequency of PU preventive measures to be taken by the nursing staff. A further validation study of the Waterlow scale is merited, on the basis of the results of this study.

Literature

1.   Haalboom JR and Bakker H. Herziening consensus preventie en behandeling decubitus [Revised consensus prevention and treatment decubitus]. Ned Tijdschr Geneesk 1992;136:1306-8.

2.   Jiricka MK, Ryan P, Carvalho MA and Bukvich J. Pressure ulcer risk factors in an ICU population. Am J Crit Care 1995;4:361-7.

3.   Herman LE and Rothman KF. Prevention, care and treatment of pressure (decubitus) ulcers in intensive care units. J Intensive Care Med 1989;4:117-23.

4.   Bergstrom N, Demuth PJ and Braden BJ. A clinical trial of the Braden scale for predicting pressure sore risk. Nurs Clin North Am 1987;22:417-28.

5.   Gunning-Scheper LJ, Wendte JF and Welleman E, Decubitus in Nederland: een onderzoek naar de mogelijkheden om het voorkomen van decubitus te meten. 1993, Amsterdam, Universiteit van Amsterdam: Instituut voor Sociale Geneeskunde.

6.   Barratt E. Pressure sores in intensive care. Intensive Ther Clin Monitoring 1990;158-67.

7.   Neuman B, The Neuman systems model. Vol. 2nd edn. 1989, Norwalk: Appleton & Lange.

8.   Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40:61-9.

9.   Gould D. Pressure sore prevention and treatment: an example of nurses' failure to implement research findings. J Adv Nurs 1986;11:389-94.

10.   Norton O, Exton-Smith AN and McLaren R, An investigation of geriatric nursing problems in hospital. 1962, National Cooperation for Care of Old people: London.

11.   Glavis C and Barbour S. Pressure ulcer prevention in critical care: state of the art. AACN Clin Issues Crit Care Nurs 1990;1:602-13.

12.   Waterlow JA. Pressure sores: a risk assessment card. Nurs Times 1985;81:49-55.

13.   Dealey C. Measuring the prevalence and incidence of pressure sores. Br J Nurs 1993;2:998-1000, 1002.

14.   Farmer R and Miller D, Lecture notes on epidemiology and public health medicine. 1991, Oxford: Blackwell Scientific Publications.

15.   Dealey C. The size of the pressure-sore problem in a teaching hospital. J Adv Nurs 1991;16:663-70.

16.   Hamilton F. An analysis of the literature pertaining pressure sore risk- assessment scales. J Clin Nurs 1992;1:185-94.

17.   Edwards M. The rationale for the use of risk calculators in pressure sore prevention, and the evidence of the reliability and validity of published scales. J Adv Nurs 1994;20:288-96.

Chapter 3

Prevalence of PU, risk factors and use of pressure-relieving mattresses in ICU patients

Jan T.M. Weststrate [1]

Freerk Heule [2]

1.   Surgical intensive care unit, Erasmus MC, University Medical Center, Rotterdam
2.   Department of Dermatology, Erasmus MC, University Medical Center, Rotterdam

Abstract

Objective: To investigate the prevalence of PU in intensive care units (ICU) in 4 European countries. Secondly, to investigate which organizational and clinical strategies are used in those countries to prevent the development of PU in intensive care patients.

Method: A questionnaire was distributed among the ICUs in Denmark, Italy, Germany and The Netherlands. Part 1 of the questionnaire was devoted to questions on the organization in relation to the development of PU. Part 2 of the questionnaire was devoted to questions on the risk, the presence and the prevention strategies followed for each patient in the ICU.

Results: A total of 299 patients in 44 ICUs were investigated. A special protocol in relation to preventing the development of PU was used in 71% of the ICUs and 41% had a specialist nurse, who was appointed for this task, working within the unit. Forty-three percent of the units used the Norton scale to assess the risk of PU development. Twenty-seven percent of the units used no risk assessment scale, but used their own clinical judgment in assessing the risk of PU development in patients. The commonly observed risk factors were a decreased mobility and activity, an increased sensitivity and the use of vasoactive medication.

Twenty-seven percent of the 299 patients had a stage II or higher PU. This varied between the countries from 4%-49%. Eighty percent of the patients who ran a high risk of developing a PU used a special support mattress. In one country, 36% of the patients with a PU used no special support mattress.

Discussion: The development of a PU is a complication that occurred frequently in the ICUs that participated in this study. The prevalence of PUs varied considerably between the countries and the various ICUs. The PU prevention strategies also varied between the countries and the participating ICUs. Standardization in using risk assessment scales, preventive interventions and the use of a special mattress is strongly recommended. The European PU Advisory Panel (EPUAP) in collaboration with the European Federation of Critical Care Nursing associations (EFCCNa) and the European Society of Intensive Care Medicine (ESICM) may all play in important role in developing and implementing such a protocol in the ICUs in Europe.

Introduction

Preventing the development of a pressure ulcer (PU) is an important aspect of the care provided by nurses and physicians. Besides being a very painful and uncomfortable complication, pressure ulcers (PUs) also affect nursing by increasing the workload per patient by 50% [1]. Furthermore, it is a very costly healthcare problem. In the Netherlands alone, it is estimated that approximately 350 million Euros are spent each year on the prevention and the treatment of PUs [2]. In another study, the cost of treating a PU per patient was calculated to be between 5,500 and 45,000 Euros [3]. Although prevention is 'treating' something that is not yet there, it is still the best and the cheapest option compared with the costs of treating a PU [4].

In general, there are three groups of patients who run a particularly high risk of developing a PU. Besides patients with spinal cord injuries or geriatric patients, patients in an intensive care unit (ICU) are particularly prone to developing PUs. Jiricka *et al.* [5] reported that 25% of patients developed a PU of higher than stage II during their stay in the ICU. If PUs of stage I were included, the percentage of patients who developed a PU increased to 56% [5]. In a more recent study, Fifi *et al.* [6] reported that the incidence of PUs (stage II or higher) was 12.4% in patients in the ICU. In another study, the incidence of PUs (stage II or higher) was noted to be 7.9% among 594 surgical ICU patients [7]. These results demonstrate the large differences in the incidence between different ICUs. This may be caused not only by the fact that different groups of patients were included in those studies, but also due to the differences in the care of the patients. This last issue is difficult to discuss among nurses because in the past, the development of PUs was often associated with poor basic patient care [8]. Nowadays, however, the problem of PUs in most institutions is regarded as a multifactorial, multidisciplinary problem that requires a multidisciplinary approach for solution. The influence of the European PU Advisory Panel (EPUAP) may have contributed to this change in perception as this organization includes physicians, nurses, technicians and scientists.

PU risk assessment scales
There is a variety of PU risk assessment scales, which are used in ICU patients to quantify the level of risk (low, medium, high and extremely high). These may help ICU nurses in an early identification of patients needing preventive intervention to stop the development or worsening of a PU. Different risk assessment scales have been used by different investigators. For example, the Braden risk assessment scale was used by Fifi *et al.* [6], Carlson *et al.* [9] and Jiricka *et al.* [5], where as Weststrate *et al.* [7] used the Waterlow risk assessment scale, and Jackson [10] used the Jackson/Cubbin pressure area risk calculator. It is important to realize that these assessment scales are not predictive instruments, as they are unable to predict the development of a PU, irrespective of what the intensive care nurse does to prevent it. Instead, these scales provide a clinical indication of the risk a patient has of developing a PU if no preventive interventions are implemented [11].

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Risk factors

Several studies have identified risk factors for PUs, which are particularly associated with ICU patients. Batson *et al.* [12] identified four highly significant, critical care-related factors: noradrenaline infusions, adrenaline infusions, restrictive movement (due to traction, post-operative pain, intra-aortic balloon pump and hemofiltration), patients who were too unstable to turn. In another study, extracorporeal circulation and time on the operating table were reported to be significant factors [13], whereas sensory perception of the patient was a critical factor in one study [9]. From three national prevalence studies done in the Netherlands, Bours *et al.*[14] observed that the most significant risk factors were: age, day since admission, malnutrition, and the three Braden subscales, moisture, sensory perception and mobility. Thus, specific risk factors may be identified, though different studies emphasized different factors.

Pressure-relieving mattresses

In the area of critical care, the use of pressure-relieving mattresses is an essential component for the prevention of PUs. Patients in ICU can develop a PU within hours because of the above mentioned risk factors. The availability and the readiness of special support surfaces that relieve pressure and therefore stimulate skin perfusion are essential. Having to wait for another 12–24 hours for a special mattress may be detrimental to the skin of the intensive care patient [9].

Prevalence of PUs

From a European perspective, O'Dea [15] investigated the prevalence of PUs in four countries (The Netherlands, Italy, Germany and the U.K.); prevalence ranged from 7–18%. At present, there are no studies in which the difference in the prevalence of PU in ICUs in different countries has been investigated. As European nations become more integrated and work together more closely, knowledge of PU prevention strategies used by the various ICUs in Europe may be exchanged to identify the most successful strategies. The prevalence of PUs in intensive care patients in ICUs in four European countries and factors that could possibly influence the results were investigated in this study.

Materials and Methods

A questionnaire, specially developed for this study, was distributed to ICUs in Denmark, Germany, Italy and The Netherlands. Part 1 of the questionnaire consisted of questions on the organization of the ICU in relation to PU prevention (Table I). Part 2 consisted of questions on the risk, the presence and the prevention strategies for each patient in the ICU (Table I).

Table I.   Study questionnaire provided to ICUs

Part 1: Questions answered by the nurse manager on organizational issues in relation to prevention of PUs in the ICU

1. Presence of a specialized nurse in the unit or in the hospital for advise regarding assessment of the risk of PU development and its prevention
2. Name of PU risk assessment scale
3. Frequency of PU risk assessment
4. Presence of a hospital PU prevention and treatment protocol in the unit

Part 2: Patient observations answered by the ICU nurse in relation to PU prevention

1. Demographic data
2. Risk of PU development according to the PU risk scale used (high, medium or low)
3. Surgery in the previous 24 hours or more than 24 hours ago
4. Use of vasoactive medication in the previous 24 hours
5. Mobility (immobile, changes position sometimes, changes position frequently)
6. Activity (inactive, on chair, active)
7. Sensitivity (no reaction to stimuli, limited reaction, normal reaction)
8. Presence of incontinence and edema
9. Type of nursing interventions taken to prevent PU development
10. Type of surface upon which the patient is lying.

The nurse managers of the different ICUs were approached by national representatives of KCI Medical (Houten, The Netherlands), and asked to participate in the study. If participation was agreed, the KCI representative explained the questionnaire to the nurse manager and the nursing team. The management of the ICU was asked to choose a day in the subsequent week to complete a questionnaire for each patient present on that day in the unit at 8.00 a.m. Part 1 of the questionnaire was filled in by the nurse manager and part 2 was filled in by the ICU nurse looking after the patient at the time of assessment. After completion, of the questionnaire was checked and signed by the nurse manager for authenticity and sent to the Dutch office of KCI Medical, where the information was entered into a Microsoft Access database. Once all the data had been entered, the data entry was checked for accuracy and the data file with the original data was sent to the investigators who analyzed the data using the statistical software program SPSS 9.0.

Results

Participating ICUs

A total of 299 patients in 44 ICUs were evaluated between March and June 2000. The distribution of the ICUs in the participating countries is shown in Table II. The majority of them were general ICUs (n = 29) and coronary care units (n = 8). The remaining ICUs were medical (n = 1), neurological (n = 2), surgical (n = 3) and cardiothoracic (n = 1). The average number of beds in each ICU was 8.7 (range between 3 and 18). Of all the ICUs, 71% used a hospital PU prevention and treatment protocol and 61% had a nurse who was specialized in the prevention and the treatment of PUs at the hospital. Forty-one percent of the units actually had a specialist PU nurse working in the unit. In relation to the use of a PU risk assessment scale, the Norton risk assessment scale was used in 43.2%, the Waterlow scale in 6.8%, the Braden in 4.5% and the Dutch CBO scale in 4.5% of the ICUs. In 27.3% of the ICUs the nurses used their own clinical judgement instead of an existing scale to evaluate the patients' risk of developing a PU. In 9.1% of the ICUs, the nurses used their local scale, while 4.5% of the ICUs used another nameless scale. In 25% of the ICUs own risk assessment scale was used during every shift and 36% of the ICUs did this daily. A risk assessment scale was used by 34% of the ICUs once a week, whereas in 5% of the ICU no risk assessment was carried out.

Table II. Number (No) of participating Intensive care units (ICUs,) total number of patients and pressure ulcer (PU) prevalence data per country

Country	No. of participating ICUs	No. of patients	No. of patients with a PU
Denmark	3	24	1
Italy	25	150	21
Germany	11	99	49
The Netherlands	5	26	10
Total	44	299	81

Patients

A total of 299 patients participated in the study. An overview of the number of patients with a PU is shown in Table II, and the demographic data for all patients in the study are shown in Table III.

Of the total number of patients, 27% had a PU of stage II or higher. The prevalence of PUs varied between countries; it was 4% in Denmark, 14% in Italy, 49% in Germany, and 38% in The Netherlands. At the moment of observation, intensive care nurses were asked to classify their patient as having a high, medium or low risk for

developing a PU, according to the risk assessment scale used in their ICU. Overall, 150

Table III.   Demographic data of participating patients (± SD)

	Female	Male
Age (years)	62 (± 20)	59 (± 18)
Height (m)	1.63 (± 0.13)	1.74 (± 0.13)
Weight (kg)	67 (± 22)	76 (± 18)
Days in ICU	11 (± 23)	17 (± 30)

patients were identified as being at high risk of developing a PU, of whom 31% already had a PU. Of the patients (n = 88), with a medium risk of developing a PU 27% already had a PU. Of the patients (n = 29) at low risk of developing a PU, 3% had a PU. In Germany, among high-risk patients with no PUs, 63% were being given preventive treatment with a special mattress; in the other countries in the study, this figure was 85%. The risk level for PUs per country is shown in Table IV.

Table IV.   A breakdown of the number of patients (n), their levels of risk (high, medium, low) and the percentage of patients in each risk level who had a pressure ulcer (PU) (n-PU (%) per country

Level of risk	Denmark		Germany		Italy		The Netherlands	
	n	n-PU %)	n	n-PU (%)	n	n-PU (%)	n	n-PU (%)
High	16	1(6)	37	21(57)	79	15(19)	18	10(56)
Medium	6	0	43	21(49)	34	3(9)	5	0
Low	1	0	6	1(17)	19	0	3	0
Missing data	1	0	13	6(46)	18	3(17)	0	0
Total of patients	24		99		150		26	

Risk factors

The following risk factors for developing a PU were observed in this study: mobility, activity, sensitivity, vasoactive medication, incontinence, edema, surgery less than 24 hours ago and surgery more than 24 hours ago. The frequency with which these factors were present in all patients and specifically in those patients who had developed a PU are shown in Table V.

Table V.   Number of patients without a pressure ulcer (n-PU), but with a specific risk factor at the time of assessment (n-total) compared to the number of patients with a PU in whom a specific risk factor is present.

Risk factor	Denmark		Germany		Italy		The Netherlands	
	n-total	n-PU	n-total	n-PU	n-total	n-PU	n-total	n-PU
Immobility	13	1	63	39	77	13	20	7
Inactive	16	1	67	39	113	16	24	8
Limited (or no reaction)	14	1	83	43	90	14	19	6
Vasoactive medication	10	1	53	29	76	10	10	4
Incontinence	12	1	73	42	94	15	6	2
Edema	8	1	1	1	27	7	12	7
Surgery < 24 hours	7	1	20	9	32	0	5	2
Surgery > 24 hours	17	1	55	28	73	16	14	5

Support mattress

According to the risk level of developing a PU, the results indicated that a special support mattress was used by 80% of the high risk patients, 64% of the medium risk patients and 54% of the low risk patients. The frequency of choice for each type of commonly used support mattresses for patients with and without a PU is shown in Table VI.

Table VI.   Number of patients (with or without a pressure ulcer (PU)) using each mattress type at the time of assessment, and the number of patients with a PU ( ) using each type of mattress

Type of mattress	Denmark	Germany	Italy	The Netherlands
Hospital mattress	6	39 (14)	31 (1)	3
Foam mattress	3	22 (13)	6 (1)	-
Air mattress	-	-	5 (2)	7 (4)
Water mattress	8	-	23 (1)	-
Alternating mattress	-	10 (5)	14 (2)	4
Low-air-loss mattress	-	24 (16)	44 (9)	10 (5)
Rotation bed	-	-	11 (5)	2 (1)
Other	7 (1)	4 (1)	16	-
Total no. of patients	24 (1)	99 (49)	150 (21)	26 (10)

Discussion

The number of ICUs that participated in the study in each country varied significantly. An explanation for this variance may be that some ICUs that were approached refused to participate because of the additional work involved in collecting the data or because they did not want to make their prevalence data public. Whatever the reason, the only way to improve the standard care in this area is to share the information on the prevalence and the incidence for the PU prevention and treatment protocols. The importance of this was seen in a number of articles that discussed different methods for improving the care of patients at risk of PU development [16-19].

Need for standardization
The variety of risk assessment scales implies that there is no general consensus among the intensive care nursing organizations on which risk assessment scale to use for intensive care patients. Barrett suggested using the Waterlow scale for the assessment of risk of PU development in intensive care patients [1]. At present, only Weststrate *et al.* [7] have evaluated the Waterlow scale in this patient group. Other investigators found various cut-off levels when they validated the Braden PU risk assessment scale for intensive care patients [5, 6]. Braden and Bergstrom [20] suggested that often these differences may be related to the influence of external factors such as staffing ratios. However, regardless of which risk assessment scale is chosen, the scale needs to be validated based on the patients and working conditions in the ICU. Besides validation, standardization in using a particular PU risk assessment scale for ICU patients would make comparison and analysis possible between the different PU prevention programs used in different ICUs.

Frequency of risk assessment
The frequency of assessment depends on the type of patients. The more rapid the changes are likely to occur in a patient's condition, the more frequently the risk assessment should be carried out. For ICUs, it was recommended that risk assessment should be carried out once every 24 hours [20]. This is endorsed by the fact that the majority of patients, regardless of whether they already had a PU, could be classified as being at high risk of developing a PU (Table IV). Despite this finding, 34% of the ICUs only assessed the patients' risk once a week. This period is far too long as nursing awareness lags behind the actual status of most patients, and in some cases, PUs can develop in only a few hours. It is striking that there was a large amount of missing data concerning the calculated risk levels for Germany and Italy. It is possible that the risk assessment scale used for these patients did not discriminate between different risk levels.

Risk factors
Immobility, inactivity, impaired sensitivity, vasoactive medication and incontinence were observed to be the most important risk factors for the development of a PU in this study.

Impaired sensitivity was also found by Carlson *et al.* [9]. In other studies, these risk factors including inactivity and the use of vasoactive medication[12], immobility [21], and incontinence[22] were noted to be important for a PU development.

Prevalence rates

The PU prevalence rates (stage II or higher) for the various countries varied significantly. The highest figure of prevalence was 49% in ICUs in Germany. In an earlier study, O'Dea [15] reported that Germany had a PU prevalence of 4% among hospital patients, but this study was conducted in general patients and not only in intensive care patients. The prevalence of PU in ICUs in Europe has not been compared thus far. Prevalence studies are difficult to compare with incidence studies because they include every patient with a new PU within a specific time frame [23]. Prevalence studies take a 'snapshot' at one moment in time. Another reason why comparison is difficult is that some studies include stage I (non-blanching erythema) as being a PU, although this stage is reversible. In our study, we excluded this stage because it was shown that nurses do not always know the difference between blanching and non-blanching erythema. At PU stages II, III and IV, the skin is broken and so they are easier to classify accurately [24] than stage I.

Type of support mattress

The use of support mattresses is interesting. The largest number of different types of mattresses was used in Italy, whereas it was the smallest in Germany. Besides the low-air-loss and alternating mattress, the water mattress was frequently used. Compared with a standard mattress, Cullum *et al.* [25] reported that the water mattress prevented the development of a PU more effectively, but there was no significant difference between a water mattress and an alternating mattress. Furthermore, there was no evidence that low-air-loss mattresses are more effective than alternating mattresses in preventing PUs, although these was only limited evidence for a reduced incidence of PUs in ICU patients with low-air-loss mattresses. In order to decide which kind of mattress (low air-loss or alternating) is more effective in preventing PUs in intensive care patients, ICUs should standardize the type of mattresses used to one of these two alternatives. Italy and The Netherlands were the only countries where rotation beds are used. Primarily, rotation beds have been used in (ventilated) patients to prevent further pulmonary complications [26]. However, rotation beds have a secondary effect in preventing the development of a PU. The original, primary reason for using such expensive beds was not given in that study.

It was found that Germany had the highest proportion of at-risk patients (high and medium). The finding that care providers waited too long before installing a special mattress for high and medium risk patients may be the reason why Germany had the highest prevalence of PUs in ICUs in this study. As the average 'incubation period' of a PU is 4 days [27]. It is critical that some sort of pressure relief is installed as soon as a patient has an increased risk (medium or high) of developing PUs. Compared to other countries, Germany had the lowest proportion of high-risk patients using a special mattress.

Conclusions

It is evident that the prevalence of PUs in intensive care patients in different ICUs varies between the different European countries, and in relation to risk assessment scales and type of mattresses used. Protocols provide professional guidance regarding procedures to follow when a PU is likely to occur in a patient. Apparently some protocols are more effective than others in preventing PUs. In order to prevent the development of PUs in intensive care patients in ICUs in Europe, it is essential that protocols for the prevention are standardized for ICU patients. The EPUAP can play an important role in developing such a protocol because they have an extensive network of clinical healthcare professionals. For successful implementation of such a protocol, collaboration with other European professional societies such as the European Federation of Critical Care Nursing associations (EFCCNa) and the European Society of Intensive Care Medicine (ESICM) is critical. Together with monthly prevalence studies at each ICU, successful strategies can be developed and further implemented in all ICUs in Europe.

Literature

1.   Barratt E. Pressure sores in intensive care. Intensive Ther Clin Monitoring 1990;158-67.
2.   Haalboom JR. [The costs of decubitus] De kosten van decubitus. Ned Tijdschr Geneeskd 1991;135:606-10.
3.   Inman KJ, Sibbald WJ, Rutledge FS and Clark BJ. Clinical utility and cost-effectiveness of an air suspension bed in the prevention of PUs [see comments]. JAMA 1993;269:1139-43.
4.   Xakellis GC and Frantz RA. The cost-effectiveness of interventions for preventing PUs. J Am Board Fam Pract 1996;9:79-85.
5.   Jiricka MK, Ryan P, Carvalho MA and Bukvich J. PU risk factors in an ICU population. Am J Crit Care 1995;4:361-7.
6.   Fife C, Otto G, Capsuto EG, *et al.* Incidence of PUs in a neurologic intensive care unit. Crit Care Med 2001;29:283-90.
7.   Weststrate JT, Hop WC, Aalbers AG, Vreeling AW and Bruining HA. The clinical relevance of the Waterlow pressure sore risk scale in the ICU. Intensive Care Med 1998;24:815-20.
8.   Dealey C. The size of the pressure-sore problem in a teaching hospital. J Adv Nurs 1991;16:663-70.
9.   Carlson EV, Kemp MG and Shott S. Predicting the risk of PUs in critically ill patients. Am J Crit Care 1999;8:262-9.
10.   Jackson C. The revised Jackson/Cubbin Pressure Area Risk Calculator. Intensive Crit Care Nurs 1999;15:169-75.
11.   Waterlow JA. Reliability of the Waterlow score [letter; comment]. J Wound Care 1995;4:474-5.
12.   Batson S, Adam S, Hall G and Quirke S. The development of a pressure area scoring system for critically ill patients: a pilot study. Intensive Crit Care Nurs 1993;9:146-51.
13.   Kemp MG, Keithley JK, Smith DW and Morreale B. Factors that contribute to pressure sores in surgical patients. Res Nurs Health 1990;13:293-301.
14.   Bours GJ, De Laat E, Halfens RJ and Lubbers M. Prevalence, risk factors and prevention of PUs in Dutch intensive care units. Results of a cross-sectional survey. Intensive Care Med 2001;27:1599-605.
15.   O'Dea K. The prevalence of pressure sores in four European countries. J Wound Care 1995;4:192-5.
16.   Sacharok C and Drew J. Use of a total quality management model to reduce PU prevalence in the acute care setting. J Wound Ostomy Continence Nurs 1998;25:88-92.
17.   Cockbill-Black S, Bond J, Bersee-Mills A, *et al.* Audit of pressure area care and documentation. Prof Nurse 1999;15:173-6.

18. Logan J, Harrison MB, Graham ID, Dunn K and Bissonnette J. Evidence-based pressure-ulcer practice: the Ottawa model of research use. Can J Nurs Res 1999;31:37-52.
19. Weststrate JT and Bruining HA. Pressure sores in an intensive care unit and related variables: a descriptive study. Intensive Crit Care Nurs 1996;12:280-4.
20. Braden BJ and Bergstrom N. Risk assessment and risk-based programs of prevention in various settings. Ostomy Wound Management 1996;42:6S-12S.
21. Jesurum J, Joseph K, Davis JM and Suki R. Balloons, beds, and breakdown. Effects of low-air loss therapy on the development of PUs in cardiovascular surgical patients with intra-aortic balloon pump support. Crit Care Nurs Clin North Am 1996;8:423-40.
22. Allman RM, Goode PS, Patrick MM, Burst N and Bartolucci AA. PU risk factors among hospitalized patients with activity limitation [see comments]. JAMA 1995;273:865-70.
23. Lake NO. Measuring incidence and prevalence of PUs for intergroup comparison. Adv Wound Care 1999;12:31-4.
24. Collier M. Blanching and non-blanching hyperaemia. J Wound Care 1999;8:63-4.
25. Cullum N, Deeks J, Sheldon TA and Fletcher AW. Beds, matresses and cushions for pressure sore preventions and treatment. Cochrane Database Syst Re 2000;4.
26. Nelson LD and Choi SC. Kinetic therapy in critically ill trauma patients. Clin Intensive Care 1992;3:248-52.
27. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40:61-9.

Chapter 4

The clinical relevance of the Waterlow Pressure Sore Risk Scale in the ICU

Jan T.M. Weststrate [1]

Wim C. J. Hop [2]

Arend G. J. Aalbers [1]

Albert W. J .Vreeling [1]

Hajo A. Bruining [1]

1. Surgical intensive care unit, Erasmus MC, University Medical Center, Rotterdam
2. Department of Epidemiology & Biostatistics, Erasmus MC, University Medical Center, Rotterdam

Abstract

Objective: Evaluation of whether the Waterlow pressure ulcer risk (PUR) scale (see Appendix 1) has prognostic significance for intensive care patients.

Design: A prospective study.

Setting: The surgical intensive care unit (SICU) of the Erasmus MC, University Medical Center, Rotterdam.

Patients: A total of 594 patients who had been admitted to the SICU during the year 1994 were investigated.

Methods and Results: Each patient was assessed daily with respect to their Waterlow PUR score and the development of pressure ulcers (PUs) in the sacral region. Actuarial statistical methods were used to analyze the predictive value of the risk score. When a patient had a Waterlow scale score > 25 on admission, the risk of developing a PU was significantly increased compared with patients with a Waterlow PUR score < 25. After admission, the daily Waterlow PUR scores obtained were significantly associated with the risk of developing a PU. For each additional point this risk increased by 23% (95% confidence interval 17% - 28%).

Conclusions: The Waterlow scale provides the medical and nursing staff with reliable information on the risk patients run to develop a PU at an early stage.

Introduction

Both time and pressure [1, 2] in combination with several predisposing intrinsic and extrinsic factors are responsible for patients developing pressure ulcers (PUs) [3]. Generally, PUs are related to a negative patient outcome associated with pain, depression, loss of function and independence, increased incidence of infection, sepsis and additional surgical interventions which all result in a prolonged hospital stay [4]. Those particularly prone to develop PUs are: 1) patients with spinal cord injuries; 2) geriatric patients; 3) patients who have undergone major orthopedic surgery; and 4) patients who are to be admitted to the intensive care unit (ICU) [5].

Pressure ulcers not only affect the patients but also increase the nursing workload by 50% once a patient has developed a PU [3]. The yearly costs of prevention and treatment of PUs in The Netherlands is estimated to be 350 million Euros [6]. Therefore, prevention of PUs is not only beneficial to the patient, but also has a secondary economical benefit for the health system.

On average 6-10% of all hospitalized patients in The Netherlands suffer from a PU [6]. In order to determine which patient has an increased risk of developing a PU, PU risk scales have been developed to measure the influence of intrinsic factors on the development of PUs [7]. Several of those scales have been used over the last 30 years. The Norton Scale is probably the most known [8].

The working of these scales is based on the summation of a specific number of points for each intrinsic factor that is present in the patient indicating the level of the risk a patient has of developing a PU. Most scales make use of a threshold score. When a patient reaches this threshold then the development of a PU is likely in the near future. Waterlow stressed that scales are not designed to predict the inevitable development of PUs, but should mainly serve as a clinical warning device [9]. The primary purpose of such a scale is to provide the physicians and the nurses with information which indicates the risk a patient has of developing a PU [10]. This information may be helpful in deciding which appropriate preventive measures need to be taken.

A scale widely used in the United Kingdom is the Waterlow Pressure Sore Risk Scale (Waterlow scale) (Appendix I) [9]. This scale contains a number of intrinsic factors, which makes it suitable for a variety of clinical settings [11]. In the Waterlow scale, the risk of developing PUs varies across a gradient of "no risk" to "very high risk" and uses categories to identify these risks. Hunt [12] suggested that for the clinical use of a scale this may be a more suitable approach than using a single threshold score to identify patients at risk.

On the basis of an earlier study [13] and the indication that the Waterlow scale is suitable in a variety of clinical settings, we designed a study to evaluate whether the Waterlow scale was of clinical value when used in the special group of intensive care patients.

Patients and Methods

A prospective study was performed in all patients who were admitted to the surgical intensive care unit (SICU) of the University Hospital Rotterdam in 1994. They were assessed with respect to their PU risk score and the development of PUs in the sacral region. Patients were excluded from the study if their stay in the ICU was less than 24 hours or had a stage II PU on admission (n = 31) or used a special mattress on admission other than the standard hospital mattress (n = 34). The PU risk scale developed by Waterlow was used to measure the risk of PUs [11]. This scale uses risk categories in order to determine the risk patients have of developing a PU. It has been suggested that the Waterlow scale is one of the most appropriate instruments for use in intensive care patients [3].

The various elements in the sections of the Waterlow scale score (Appendix 1) were assessed daily by the nurses night staff for the previous 24 hours and registered in the hospital information system. The nurses were unaware of the actual score or any previous score. The highest score was recorded from each section of the Waterlow scale. Some of the elements of the Waterlow scale sections were redefined to prevent dual explanation. In the section Mobility, all patients scored 5 points. In the section Tissue Malnutrition, cardiac failure was scored when the patient used catecholamines. In the section Neurological deficit, paraplegia was scored when the patient was on muscle relaxants. This item was also scored when patients had leg fracture and were thus immobilized. Lesions of the skin were staged according to the staging of PUs developed by the European PU Advisory Panel. Stage 0: normal skin; Stage I: non-blanchable erythema of skin; Stage II: formation of blisters; Stage III: superficial (sub)cutaneous necrosis; Stage IV: deep subcutaneous necrosis. (see Appendix II). From stage II onwards skin lesions were considered to be PUs.

If the condition of the patient allowed, the following precautions to prevent the development of PUs were carried out by the nurses: Turning the patient every three hours for a short time (< 5 minutes) onto one side nursing the patient for at least 1 hour continuously on alternate sides and mobilizing the patient out of bed in order to stand for a few minutes next to the bed or sit for 15 minutes in a chair.

These measures were performed according to the hospital protocol. Barcodes were used for registration. Each element of the Waterlow scale was linked with a barcode. Each computer that was connected to the hospital information system had a light pencil which could read the barcodes connected to it. On the average, it took a nurse between 30 and 60 seconds to record a scale score for one patient. The advantage of this registration method was that all information was stored in a central computer by the nurse who carried out the scoring. In order to analyze the information, it was transferred to a personal computer.

For statistical analysis continuous outcomes between groups were compared using the Mann-Whitney test and percentages were compared using the Chi-square test. The risk of developing PUs increased with length of stay in the ICU. To adjust the number of days the patients had been admitted to the ICU, actuarial methods (Kaplan -Meier curves,

logrank-tests) were used to evaluate and compare the risk of developing PUs. To asses the current value of the Waterlow scale score and the rate of developing a PU, Cox-regression, with the daily Waterlow scale score as time dependent covariate was used [14]. The limit of significance was considered to be p = .05 or less (two sided).

Results

After the exclusion criteria were applied, 594 of the in total 686 admitted patients were included in this study. The studied group consisted of 389 men and 205 women with a mean age of 58.8 years (range 9 to 96 years). The mean stay in the ICU was 6.3 days (range 2 to 183 days). Of all patients, 47 (7.9 %) of the 594 patients developed a PU. The characteristics of patients who did and did not develop PUs are given in Table I. All characteristics, except age differed significantly between the groups. The number of patients with various Waterlow scale score characteristics present on admission is given in Table II.

Table I.   Baseline characteristics of all included patients. Data are given as median (interquartile ranges) or percentages.

	PU: no (N = 547)	PU: yes (N = 47)	Level of significance
Age (years)	62.7 (48.8 – 70.9)	68.9 (54.6 – 75.9)	Not significant
Male/Female	67% / 33%	49% / 51%	p = 0.001
Duration of stay in the ICU (days)	3 (2 – 5)	19 (8 – 33)	p < 0.001
Waterlow scale score at baseline	17 (14 – 21)	20 (16 – 26)	p = 0.001
Turning patient (< 5 min)/24 hrs	2.5 (1.5 – 3.5)	4.2 (3.7 - 5.0)	p < 0.001
Nursing patient on alternate sides/24 hrs	0.3 (0.0 – 0.3)	0.6 (0.0 – 0.7)	p = 0.003
Mobilizing patient out of bed/24 hrs	0.4 (0.0 – 0.7)	0.1 (0.0 – 0.1)	p < 0.001

Table II.   The Waterlow scale. The * column gives the number of patients having the characteristics on the day of ICU admission

Build/weight for height	*	Sex /Age	*
Average	471	Male	389
Above average	71	Female	205
Obese	27	0 - 14	5
Below average	25	14-49	148
Continence		50-64	164
Completely catheterised	550	65-74	175
Occasional incontinence	9	75-80	68
Catheter and fecal incontinence	31	81 +	5
Incontinent for feces and urine	4	Appetite	
Skin type visual risk areas		Average	91
Healthy	521	Poor	27
Tissue paper, dry, clammy, edematous	59	NG[a] tubes and fluids only	51
Discolored	12	NBM[b]/anorexia	425
Broken spot	2	Tissue Malnutrition	
Mobility		Terminal cachexia	0
Fully mobile	0	Cardiac failure or peripheral vascular disease	145
Restless or fidgety	0	Anemia	54
Apathic	0	Smoking	ND
Restricted	0	Neurological deficit	
Inert/traction	0	Diabetes, MS[c], CVA[d]; Motor/sensory paraplegia	72
Chairbound/complete bedrest	594	Major surgery/trauma	
Medication		Orthopedic: below the waist;or spinal cord; operation time > 2 hours	364
Cytotoxics, high doses steroids, anti-inflammatory drugs	114		

a= nasogastric tube; b= nil by mouth; c= multiple sclerosis; d= central vascular accident

66

The risk of patients developing a PU according to the number of days they were in the ICU is shown in Figure 1 (upper panel). If ICU stay lasted 30 days, this risk would have increased to 60%. Patients were grouped according to baseline Waterlow scale score: < 15 points (n = 165); 15 – 19 points (n = 213); 20 – 24 points (n = 140); ≥ 25 points (n = 76). In these groups the numbers of patients developing a PU during their stay in the ICU were 9, 13, 10 and 15 respectively. The lower panel in Figure 1 shows that when patients have a score ≥ 25 on admission, the risk of developing a PU during their stay is significantly increased compared with those patients with a score < 25.
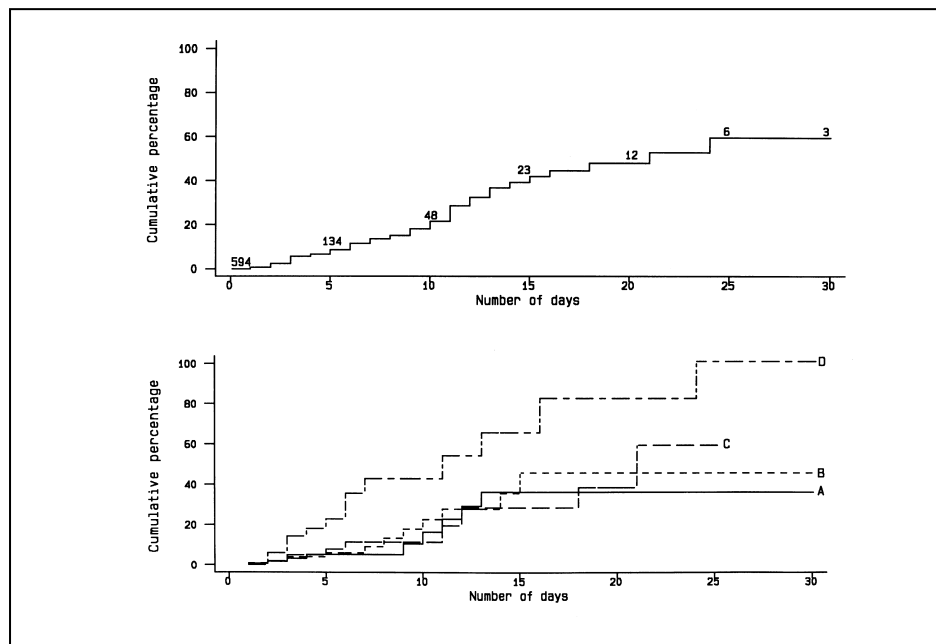

Figure 1. Upper panel: Cumulative percentage (actuarial) of patients developing a PU according to days of ICU stay. Numbers along the curve denote the numbers of patients at risk of developing a PU on the indicated days.
Lower panel: Cumulative percentage (actuarial) of patients developing PUs along time. Patients are grouped according to Waterlow scale at admission to the ICU. Curve A: < 15 points (n = 165); curve B: 15-19 points (n = 213); curve C: 20-24 points (n = 140); curve D: □ 25 points (n=76). Difference between curve D and curves A, B and C: all p < 0.001.

The longitudinally obtained Waterlow scale scores are shown in Figure 2. At each day of ICU stay, the range which covers 95% of the Waterlow scale scores for those patients who did not develop a PU at or before that day, is shown. It can be seen that patients who developed a PU on a particular day tended to have higher Waterlow scale scores compared with those who did not develop a PU on the same day. Cox-regression revealed that the rate of development of PUs increases linearly with an increasing Waterlow

scale score. For each additional point of the Waterlow scale score this rate increased by 23% (p < 0.001, 95% confidence limits: 17% - 28%). Relating both the current score and the score on the previous day to the rate of development of PUs, it appeared that the score on the previous day did not significantly improve the predictive value of the current score. Further analyses showed that as long as the Waterlow scale score continuously remained below 15 in the ICU (total: 166 patient days) none of the patients developed a PU.
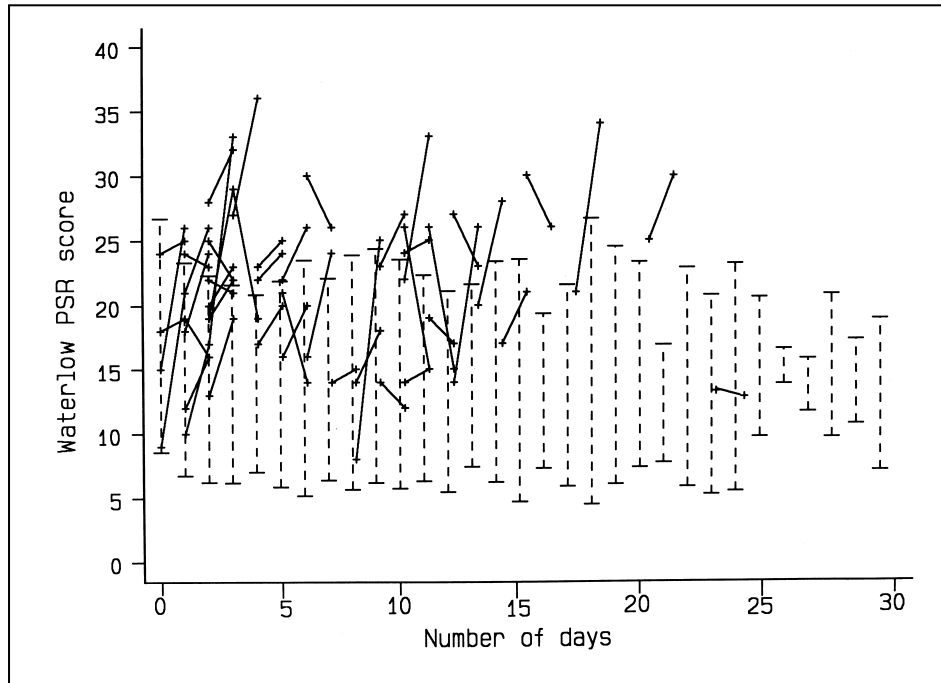

Figure 2. Graph of the Waterlow scale score versus the number of days after entry into study. Solid lines (n = 47) represent data of patients who developed a PU. The right end of each solid line represents the score value on the day of the occurrence of the PU. The left end shows the score value on the preceding day. The vertical dotted lines represent the 5-95 percentile range of the Waterlow scale score for those patients who are still at risk for developing a PU at each day, except those who already developed a PU on that day.

Figure 3 shows the actual risk of developing a PU according to whether or not patients have a Waterlow scale score, which exceeded a specific threshold. Curve I demonstrates the risk for patients if the Waterlow scale score remained below 20. Curve II shows the risk for those had a score between 20 and 25, and curve III shows the risk for patients with a Waterlow scale score exceeding 25. The risk increases considerably with increasing cut-off level. When patients have exceeded the score of 25, the risk of developing a PU within a period of 10 days is 50%.
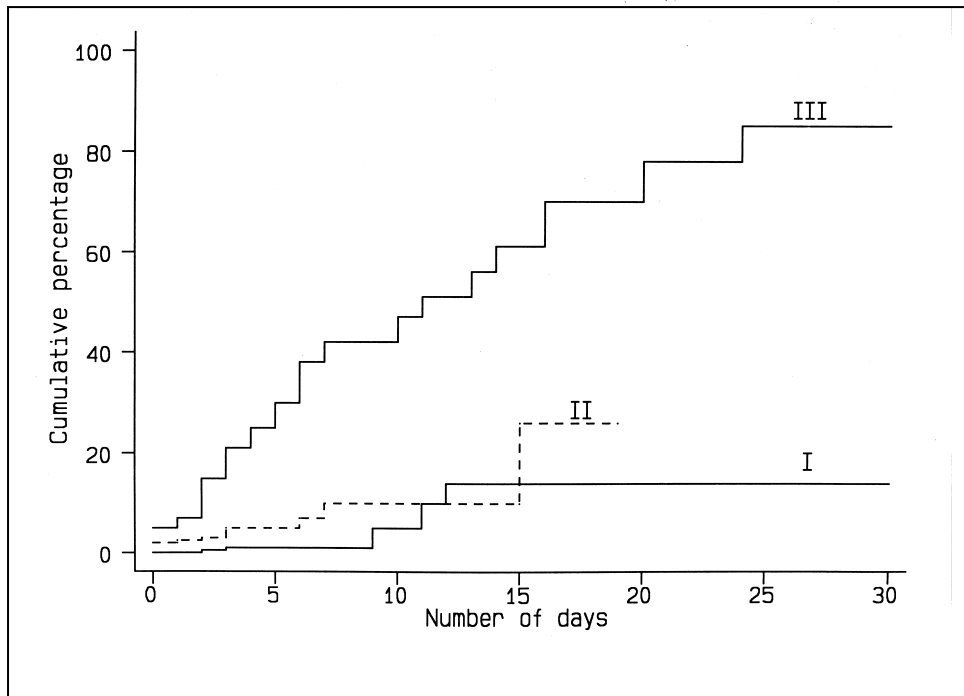
Figure 3. The risk (actuarial) of developing a pressure sore (PU)

Curve I: As long as patients continuously show a Waterlow scale score below 20 points after admission.

Curve II: After the first occurrence of at least 20 points but less than 25 points. The time axis represents the number of days patients continuously remained within this range.

Curve III: After the first occurrence of at least 25 points. The number of patients at day 0 for curves I, II and III are 378, 206 and 116 respectively.

Discussion

The most important result of our study is that it produced a PU risk assessment model, which can be used as a reliable warning device for intensive care patients in daily practice. When the Waterlow scale was developed four risk categories were suggested: No risk (<10 pnt), at risk ($\geq$10 - $\leq$14 pnt), high risk ($\geq$ 15 - $\leq$19 pnt) and extra high risk ($\geq$ 20 pnt) [11]. These risk categories were global and never quantified through research. When we quantified the risk categories in our study, on the one hand, we found that as long as the patients had a score below 15, they developed no PUs during their stay in the ICU. This finding is in contrast to the suggested risk of that category. On the other hand, when patients developed a score $\geq$ 25, the risk of developing a PU during their ICU stay was extremely high. This finding has prompted us to place patients having a Waterlow scale

score $\geq$ 25 points for more than 2-3 days on a special bed or mattress that reduces the pressure on the skin.

Without considering the length of stay in the ICU we found a PU incidence of 7.9%. Other studies reported incidences varying between 13% and 56% [4, 15-17]. On the average between 6% and 10% of the patients admitted in Dutch hospitals develop a PU [6]. The moderate risk for the total group of ICU patients in our study is mainly due to the fact that most patients had only a short stay in the ICU. At 30 days the risk of developing a PU in our study increased to about 60% (Figure 1, top panel). As a result of this study, far more attention to PU prevention is given to patients with a prolonged stay in ICU.

Our results indicated that the scale score over the last 24 hours is the best indicator for the development of a PU in the next 24 hours. As the physical condition of ICU patients can change dramatically in a very short period, the risk of developing a PU is influenced likewise [17]. This study showed that it is important to assess this risk every day, that both the nurses and the physicians must have access to this information and that the day staff compares the score in the morning with the scores of the previous days in relation to the patient's present condition. Following this, the kind and frequency of preventive measures should be adjusted if necessary. Due to respiratory and hemodynamic instability, patients in the ICU often do not tolerate being nursed on alternate sides or even being turned. In these cases the only other way to reduce pressure on the affected areas is to use a special mattress.

Most categories that are incorporated in the Waterlow scale have a confirmed influence on the development of PUs. The combination of these categories and the weighing of the various category items were based on a study of relevant literature and a discussion with other healthcare professionals. This process was confirmed in a clinical study in which 650 patients from different types of wards participated [9]. The scale was designed as an instrument to warn care providers about the risk level in patients and not to predict whether or not the patient will inevitably develop a PU [18]. The use of this evaluation method is therefore different from other scales that calculate the sensitivity and the specificity of a threshold score above which a patient will develop a PU. No quantitative risk evaluations of the Waterlow scale score regarding the development of PUs are available and so far only global evaluations have been made: two in a geriatric patient population [19, 20] and one in a community setting [21]. In both the studies, it was reported that the Waterlow scale was highly sensitive but the specificity was very low (10% -14%) indicating that it over-predicted those at risk of developing a PU [22]. By using actuarial statistical methods, as in our study, the results not only indicate a new score range in the various risk categories, but also provides insight on the course of the risk in due time. This is a valuable contribution because care providers can then determine at which risk level financially more expensive PU precautions have to be taken [11, 16].

In our study, patients who developed a PU, had a longer ICU stay compared with those who did not develop a PU. This was also reported by Jiricka, *et al.* [4]. Patients who developed a PU in our study also had a significantly higher Waterlow scale score on

admission. Again other investigators did find the same results, although they used the scale developed by Braden [23, 24]. This indicated that the longer a patient stays in an ICU, with an increased risk score on admission, the higher the risk he or she has of developing a PU despite the fact whether or not the risk score is decreasing. This supports the view that when a patient a stays in the ICU, more attention should be given to PU prevention.

Patients who developed a PU in our study received significantly more frequent short turning episodes (< 5 minutes) and were nursed significantly more on alternate sides. These precautions did not prevent the development of PUs. Since nurses were blinded to the total PU risk score of each patient they relied on the clinical assessment of the patients' skin as an indicator whether or not to initiate preventive precautions as was also found in an earlier study [13]. The results of this study showed that initiating PU preventive precautions on the basis of the patients' skin assessment does not prevent the development of PUs, but often only confines the area of damage.

The following limitations and drawbacks need to be considered. A possible reason why not all patients with an increased risk developed a PU is because the nurses increased the number of preventive measures at a very early stage. The influence of such interventions in combination with the use of a risk scale is often overlooked in other studies evaluating the effectiveness of these scales. Furthermore, different kinds and timing of interventions influence the time span between measuring an increased risk of developing a PU and the appearance of PU. It is therefore difficult to generalize the results of this study to other patient settings due to probable different levels of nurse alertness in taking measures against PUs.

The results of this study now serve as a model for the management of PU prevention in our ICU. The method of evaluation can be used for quantitative risk evaluations of any pressure risk model. This study demonstrates that evaluating pressure risk models by means of actuarial statistical methods provides the user with much valuable information on the development of PUs.

Literature

1. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40:61-9.

2. Husain T. An experimental study of some pressure effects on tissues, with reference to the bed-sore problem. J Bacteriol Pathol 1953;66:347-58.

3. Barratt E. Pressure sores in intensive care. Intensive Ther Clin Monitoring 1990;158-67.

4. Jiricka MK, Ryan P, Carvalho MA and Bukvich J. Pressure ulcer risk factors in an ICU population. Am J Crit Care 1995;4:361-7.

5. Herman LE and Rothman KF. Prevention, Care and Treatment of pressure (decubitus) ulcers in intensive care units. J Intensive Care Med 1989;4:117-23.

6. Haalboom JR and Bakker H. [Revision consensus prevention and treatment decubitus (see comments)] Herziening consensus preventie en behandeling decubitus. Ned Tijdschr Geneeskd 1992;136:1306-8.

7. Gould D. Pressure sore prevention and treatment: an example of nurses' failure to implement research findings. J Adv Nurs 1986;11:389-94.

8. Norton RM, McLaren R and Exton-Smith AN, An investigation of geriatric nursing problems in hospitals. Vol. 2nd Ed. 1975, Edinburgh: Churchill Livingstone. 193-238.

9. Waterlow J. Pressure sores: a risk assessment card. Nurs Times 1985;81:49-55.

10. Wolfswinkel-de Jong CJA and Halfens RJG. Prevention of decubitus: the effect of pressure sore risk assessement scoring system (in Dutch with English summary). Verpleegkunde 1995;17:29-36.

11. Smith LN, Booth N, Douglas D, *et al*. A critique of 'at risk' pressure sore assessment tools. J Clin Nurs 1995;4:153-9.

12. Hunt J. Application of a pressure area risk calculator in an intensive care unit. Intensive Crit Care Nurs 1993;9:226-31.

13. Weststrate JT and Bruining HA. Pressure sores in an intensive care unit and related variables: a descriptive study. Intensive Crit Care Nurs 1996;12:280-4.

14. Cox DR. Regression models and life tables. J R Stat Soc 1972;B 34:187-220.

15. Bergstrom N, Demuth PJ and Braden BJ. A clinical trial of the Braden Scale for Predicting Pressure Sore Risk. Nurs Clin North Am 1987;22:417-28.

16. Edwards M. The rationale for the use of risk calculators in pressure sore prevention, and the evidence of the reliability and validity of published scales. J of Adv Nurs 1994;20:288-96.

17. Langemo DK, Olson B, Hunter S, *et al*. Incidence and prediction of pressure ulcers in five patient care settings. Decubitus 1991;4:25-6, 8, 30.

18. Waterlow JA. Reliability of the Waterlow score [letter; comment]. J Wound Care 1995;4:474-5.

19. Clough N. The cost of pressure area management in an intensive care unit. J Woundcare 1994;33-5.

20. Dealey C. Risk assessment of pressure sores: a comparative study of Norton and Waterlow scores. Nurs Stand 1989;Spec Suppl 3:11-2.

21. Wardman C. Norton v. Waterlow [see comments]. Nurs Times 1991;87:74, 6, 8.

22. Edwards M. The levels of reliability and validity of the Waterlow pressure sore risk calculator. J Wound Care 1995;4:373-8.

23. Dealey C. Matresses and beds. A guide to systems available for relieving and reducing pressure. J Wound Care 1995;4:409-12.

24. Bergstrom N, Braden BJ, Laguzza A and Holman V. The Braden Scale for Predicting Pressure Sore Risk. Nurs Res 1987;36:205-10.

Chapter 5

The reproducibility of interface pressure measurements
in patients at risk of developing a pressure ulcer

Jan T.M. Weststrate [1]

Hajo A. Bruining [1]

Richard H.M. Goossens [2,3]

Freerk Heule [4]

Wim C.J. Hop [5]

1. Department of Surgery, Erasmus MC, University Medical Center, Rotterdam
2. Faculty of Industrial Design, Delft University of Technology, Delft
3. Department of Biomedical Engineering and Technology, Erasmus MC, University Medical Center, Rotterdam
4. Department of Dermatology, Erasmus MC, University Medical Center, Rotterdam
5. Department of Epidemiology and Bio-statistics, Erasmus MC, University Medical Center, Rotterdam

Abstract

Background: Although pressure is a critical factor in the development of a pressure ulcer (PU), clinical studies addressing the value of this parameter in a patient population are unavailable.

Objectives: To examine the "between day" reproducibility of the Tissue Interface Pressure (TIP) measurements at the sacrum and the buttocks, measured at an interval of at least 24 hours in bed rest patients.

Methods: A descriptive, longitudinal study in 76 surgical patients. The TIP was measured using the Talley Pressure Monitor III (TPM III).

Results: The main outcome was the Intra-Class Correlation coefficient (ICC) between two Peak Interface Pressures (PIP) measured with a time interval of at least 24 hours. The ICC Coefficients for assessing the "between day" reproducibility were low (the sacrum: 0.23, the right buttock: 0.13 and the left buttock: 0.15).

Conclusion: The TIP measurements at the sacrum and the buttocks vary significantly over days in a clinical population. Measurement of TIP under more standardized conditions may shed further light on this issue.

Introduction

After 45 years of intensive research, the development of pressure ulcers (PUs) is still poorly understood and imposes a significant burden on the health care budget. Prevalence studies in the Netherlands showed that about 22% of the patients in hospitals and nursing homes demonstrated evidence of the negative effects of pressure on the skin. Ten percent of these patients progress to develop a wound necessitating treatment by the medical and the nursing staff [1]. If these figures are translated into financial burden, approximately 350 million Euros are spent annually in the Netherlands in order to prevent or treat PUs. Although prevention is not "free", it is still the best option for the patient [2].

The phrase "pressure ulcer" suggests that pressure is an essential component in its development. This was first quantified by Kosiak [3] who observed that the magnitude and the duration of pressure on the skin are the primary components starting a cascade of patho-physiological and biochemical changes, which ultimately lead to the development of an ulcer. It is generally accepted that the exerted pressure on the skin inhibits satisfactory tissue perfusion, jeopardizing adequate nutrition and oxygenation at cellular level [4]. A PU will develop rapidly or slowly depending on which other risk factors are present [5]. If we assume that the exerted pressure on the skin by the support surface (Tissue Interface Pressure, TIP) is the primary cause of PU development, then this parameter may be used to predict PU development. Currently PU risk assessment scales are used for this purpose. However, research has shown that these are not optimum [6]

The reliability of the TIP is assessed by establishing the reproducibility. The European Pressure Ulcer Advisory Panel (EPUAP) defined the reproducibility as follows: "The variation in analysis outcomes when a calculation is performed on pressure data derived from a test subject who has repositioned several times". Thus, a low reproducibility implies a large variation in TIP measurements making the parameter less reliable for predicting PU development [7]. Generally three methods of establishing the reproducibility of the TIP are cited in the literature.

The first method is to repeat the measurement without removing the pressure sensor or repositioning the patient. In his study Hobson [8] investigated the pressure distribution differences affected by deformity and/or alterations in 8 different body positions within and between non-disabled and patients with a spinal cord injury. The sensors to measure the pressure were fixed to the seat surface so that the distance between them was the same in all patients. Successive pressure measurements were taken in one position without reseating the subjects in order to verify the repeatability and the variability [8]. The practical value of such a procedure for the reproducibility is minimal because the measurement was made under the same conditions so that it can be regarded as more or less continuous measurement that was interrupted for a short period.

The second method is to remove and replace the sensor between two or more consecutive measurements on the same day in the same subject [9-13]. It is important that not only the sensor is repositioned, but also that the patient or the volunteer is removed from

the support surface so that it can regain its initial thickness [13]. In a number of studies only the sensor was repositioned [10, 11].

The third method is to increase the time interval and repeat the measurement on the following or another day with the same subject on the same support surface. This procedure was not undertaken frequently because it is time-consuming and often impractical for the volunteers because of the long time period [14]. An example of such a study is that by Allen *et al.* who repeated IP measurements with an interval of at least 24 hours [10, 11].

TIP measurements may be relevant in the guidelines for the clinical practice. Therefore, the TIP measurement must be reproducible, especially taking into account the frequently changing circumstances that may affect such measurements. The majority of studies to date, in which the TIP was measured on various support surfaces and in different body positions were carried out in healthy volunteers. However, it has not yet been established that a study on measuring the TIP in the healthy volunteers mimics the situation in the patients [15]. It is more likely that the patients choose the most comfortable position during a period of bed rest, especially after surgery. Moreover, this position is not always the same for the whole day because prolonged exerted pressure on the skin forces the patient to change position. We cannot assume that such changes do not influence the reproducibility of the TIP measurement.

To our knowledge, no study has yet been undertaken in which TIP measurements were repeated on different days in hospitalized patients in order to determine the reproducibility of the TIP measurement and to assess the influence of extraneous variables such as body mass, position of backrest, use of incontinence pads and the wearing of pyjamas and underwear on this reproducibility. Therefore, we undertook a clinical study in which we addressed the "between day" reproducibility of the TIP measurements at the sacrum and the buttocks in patients who were confined to bed for at least 4 days.

Materials and Methods

Patients
Patients were selected from the general surgical and medical units of our hospital from September 2000 to July 2001. In total 76 patients participated in the study. The inclusion and the exclusion criteria for patients who participated in the investigation are shown in Table I. Data were collected after the patient had signed the informed consent form. In elective surgical patients, the first day of bed rest was defined as the day they underwent surgery. The study was approved by the local Medical Ethics Committee of the Erasmus MC, University Medical Center.

Table I.   **Inclusion and exclusion criteria for patients participating in the study.**

Inclusion criteria	Exclusion criteria
Bed rest for at least 4 days after inclusion	An existing pressure ulcer >grade 1[31]
Using a standard hospital mattress	Using a special bed frame
Caucasian	> 48 hours of bed rest before inclusion
Age => 18 years	Body Mass > 125 kg
Signed informed consent	

Pressure measurement technique
The TIP was measured with the Talley Pressure Monitor 3 (TPM III). It is a device developed at the Oxford Orthopedic Engineering Centre, Nuffield Orthopedic Centre, Oxford, England [16]. Currently Talley Group Ltd[a] markets this device. Different versions of the TPM were used extensively by several researchers for measuring the Interface Pressure (IP) in patients [8, 17, 18]. The TPM III consists of three basic components: a sensor array, a pneumatic monitor and a calibrating jig. It is able to connect to 8 arrays, each array containing 12 sensors (sensor 20 mm in diameter). The general working of the TPM was described in detail by Hobson (1992) [8]. Calibration data published by the developers showed a maximum deviation from linearity of 3 percent over the optimum range of 0-33.3 kPa [16]. We used the TPM III with 3 arrays, each containing 12 sensors (3 x 4) (see Appendix 3) and calibrated it weekly during the study.

Positioning the arrays
The arrays were positioned under the sacrum and the right and the left buttocks of the patient. The three arrays were positioned as follows: the sensor array at the sacrum was positioned first. The patient was asked to lift the buttocks and the sensor array was manually positioned by the investigator over the sacral area by positioning the sacrum as close as possible to the center of the sensor array. The second array was placed under the left buttock. To do so the patient was asked to pull up the left knee as much as possible towards the chin and to turn approximately 30^0 to the right side, without moving the right buttock over the mattress. The investigator positioned the sensor array manually over the buttock such that the ischial tuberosity was felt as close as possible to the center of the matrix. The same procedure was repeated for positioning the array under the right buttock.

In order to obtain the most accurate clinical data, the arrays were placed between the patient and the bed sheet over the standard hospital mattress (Tempur Pedic)[b]. This 14 cm mattress is made of a 4 cm top layer of visco-elastic polyurethane and a 10 cm bottom layer of cold foam. The whole mattress has a washable cover around it consisting of a semi-permeable layer of 80% cotton and 20% polyester.

If patients wore pyjamas or other underwear, the arrays were placed over this, thus not in direct contact with the skin. If the patient wore an incontinence pad, the array was placed between the mattress (with a sheet) and the pad. Information on what the patient was wearing at the time of measurement was recorded on the measurement data form. The patients were then instructed to put the backrest of their bed in the most comfortable position with their head on a pillow, their hands along side their body and their legs uncrossed and stretched out. The first ten minutes of the measurement were used to check if any of the sensors produced values that indicated a false measurement due to bending or malfunctioning of the sensor. In total 36 sensors measured the pressure between the patient and the mattress: 12 in the sacral area, 12 in the area under the right buttock and 12 in the area under the left buttock. The patient was asked to stay in the same position and lie as still as possible for ten minutes when measuring the TIP. During this period the actual TIP was measured continuously. The TPM III starts to measure the TIP at the sacrum followed by the left and the right buttocks. Then the cycle starts again. Each cycle is called a frame. Each *measurement* consisted of at least 11 frames. When the measurement procedure was finished, the sensor arrays were removed and cleaned. A second measurement procedure was carried out between 1 and 5 days later depending on the patient's clinical condition. The data were transported and statistically analyzed in SPSS (version 11.1).

Method of analyzing the interface pressure
The measured TIP was analyzed on the basis of the calculated Peak mean of the TIP (PIP) for each sensor. As the pressure between the patient and the mattress has a continuous character, the PIP probably has the highest impact on the patient's skin. The sensor with highest mean TIP in the array over the 11 measured frames was considered to be the PIP. All readings of a particular sensor were excluded if one or more readings were 32.8 kPa or higher. Apparently this is the highest readout of the TPM III and therefore, the accuracy is uncertain.

Statistical analysis
In order to analyze the reproducibility of the measurements, scatterplots were constructed for measurements of the PIPs for the sacrum and both the buttocks. To analyze the systematic statistical differences between the means of the two PIP values, the paired sample T-test was used. To analyze the differences of the mean PIP between the various sites (the sacrum, the left and the right buttock) within each measurement the paired sample T-test was used. The reproducibility was quantified by calculation of Intra-Class Correlation Coefficients (ICC). The reproducibility was considered satisfactory if the ICC of the two measurements was at least 0.8.

To evaluate the reproducibility of the measurement technique itself, the data from each measurement period was divided in half and was treated as if it were two separate measurements. The mean values of both (sub) measurements were compared with one another and the ICC was calculated once again.

Changes in the PIP between the first and the second measurements were evaluated with regard to the effects of body mass, height, Body Surface Area (BSA) and Body Mass Index (BMI), using the Spearman correlation test. The same was done with regard to the effect of change in backrest and Fowler's position. Changes in the use of incontinent pads, pyjamas and underwear were evaluated with regard to the change in PIP using ANOVA. Multivariate analysis of the effects of these factors on the change of PIP was done using multiple regression. The level of significance was set at $p < 0.05$ for all the statistical tests.

Results

In the period September 2000 to July 2001, 76 patients were included in the study (44 males and 32 females). An overview of the demographic characteristics of the patients is shown in Table II.

Table II. **Mean values of demographic characteristics of participating patients**

	Patients (n=76)	Range
Age (years)	59	30-95
Mass (kilograms)	76	33-114
Height (cm)	173	152-197
Body Mass Index [32]	25	10-39
Body Surface Area (cm^3)	1.88	1.4-2.4

The reliability of the measurement technique proved to be high. Comparing the mean of the first half of each individual measurement with the second half, the ICC for the measurements at the sacrum, the right buttock and the left buttock were 0.80, 0.90 and 0.88, respectively.

An overview of the position of the bed frame and the mean PIP values measured at the sacrum and the buttocks of measurements 1 and 2 are shown in Table III. The position of the bed rest could not be standardized as patients were asked to position themselves in the most comfortable position before each measurement. This position varied between the measurements as the most comfortable position of the patient changed frequently during their stay in the hospital. The mean interval between the two measurements was 4.3 (SD 2.9) days.

There appeared to be no significant difference between the two mean PIP values at all the three sites for the two measurements. Within both measurements, the mean PIP values at the three sites differed significantly between the sacrum and the right buttock (measurement 1: $p = 0.001$; measurement 2: $p = 0.008$) and between the left and the right buttocks ($p= 0.016$) in the second measurement.

Table III.   An overview of the mean Peak Interface Pressure (PIP) at measurements 1 and 2 with significance levels.

	Measurement 1 (sd)	Measurement 2 (sd)	Significance
Backrest elevation	21^0, range 0^0-47^0	21^0, range 0^0-58^0	p = .960
Fowler elevation	1.2^0, range 0^0-22^0	3.2^0, range 0^0-35^0	p = .102
PIP-Sacrum	6.6 kPa ($\pm$ 2.5)	6.0 kPa ($\pm$ 2.7)	p = .108
PIP-right Buttock	5.4 kPa ($\pm$ 2,6)	4.9 kPa ($\pm$ 2.6)	p = .214
PIP-left Buttock	6.0 kPa ($\pm$ 2.0)	5.6 kPa ($\pm$ 1.6)	p = .073

In order to assess the "between day" reproducibility of the PIP visually, measurement 1 was plotted against measurement 2 in a scatter plot. The PIP at the sacrum and both the buttocks is shown in Figure 1. The resulting ICCs for the sacrum, the right buttock and the left buttock were 0.23, 0.13 and 0.15, respectively. These results indicated a low "between day" reproducibility.

In 17 patients the second measurement was on the next day ("between day" interval 1 day). The correlation between both the measurements in this group (group A) was compared with the correlation between both measurements of the remaining 59 patients (group B). The ICCs for the sacrum was higher in group A compared with group B (0.36 versus 0.20). For the right buttock the values were 0.07 versus 0.17 and for the left buttock the values were 0.24 and 0.07.

Comparing the absolute value of the change in PIP of measurement 1 with measurement 2, a statistically significant difference was observed between the PIP at the sacrum and the PIP at the left buttock (2.4 kPa versus 1.6 kPa; p< 0.001). The other three comparisons were not significantly different.

The possible influence of extraneous variables was also analyzed. During the first measurement, the elevation of the backrest had a statistically significant positive correlation with the PIP at the sacrum and the right buttock. During the second measurement the PIP at all locations had a significant correlation with the elevation of the backrest. The level of the Fowler's position had only a significant correlation with the measured PIP at the sacrum during the first measurement. There was a correlation between the change in backrest elevation and the change of PIP at the sacrum and the left buttock (r = 0.28, p = 0.014; r = 0.27, p = 0.022, respectively).

Sacrum   Right buttock

Left buttock
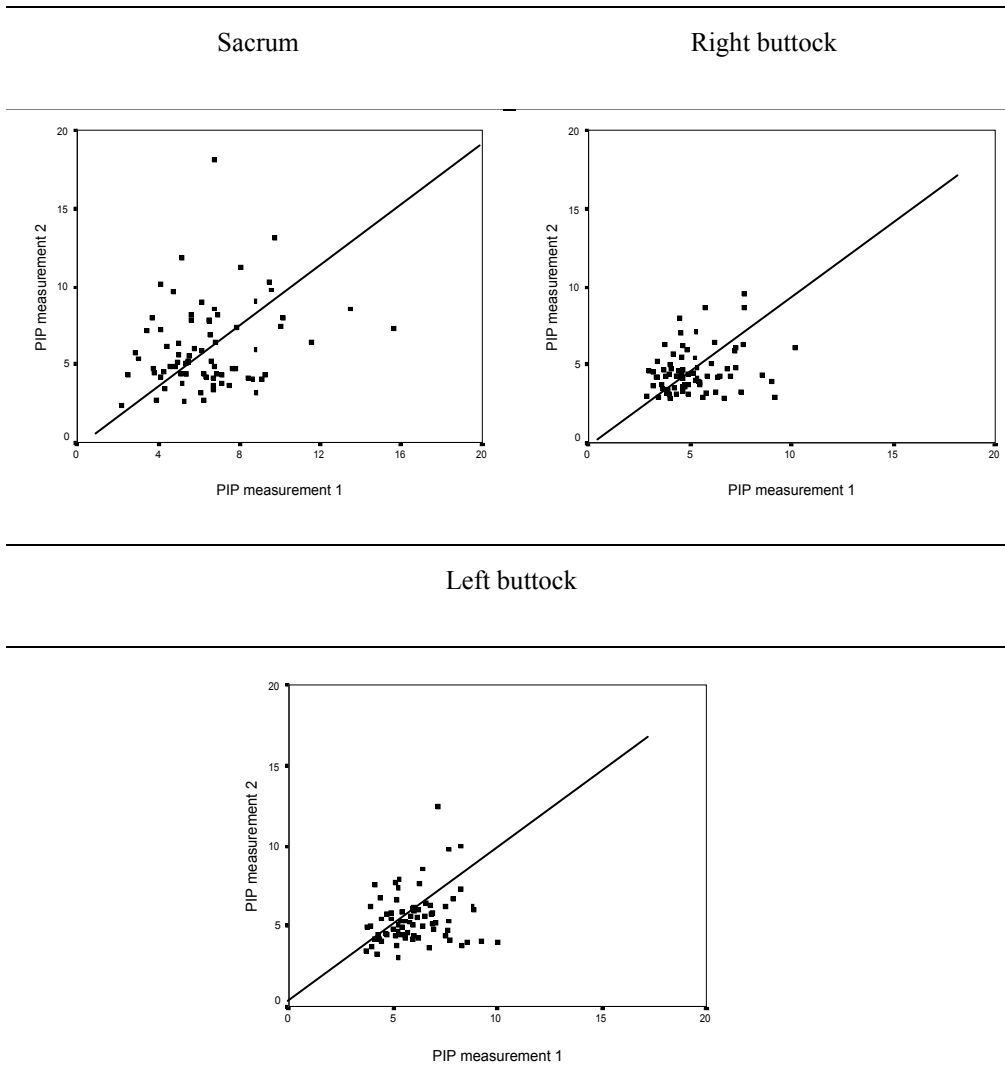
Figure I. The reproducibility of the PIP (Peak Interface Pressure) at the sacrum and the buttocks. The line in the figures represents the line of indentity

Changes in wearing pyjamas, and underwear had no statistically significant influence on the changes in PIP for each of the three sites. A change in incontinence pad however, influenced the changes in PIP between the two measurements only at the left buttock (p = 0.045). Using multiple regression it was noted that the effect of change in backrest differed between the groups who had and who had not changed the incontinence

pad (p = 0.029).In the group who had not changed incontinence pad, there was a significant positive correlation (r = 0.29, p = 0.019) between the change in the backrest and the change in the PIP at the sacrum. The patients who had changed (n = 12) incontinence pad showed a significant correlation at the left buttock (r = 0.76, p = 0.004) between the change in the PIP and the change in the backrest.

Of the patients' characteristics, only body mass showed a significantly positive correlation with the level of the PIP during both measurements at the left and the right buttocks (the right buttock measurement 1:r = 0.28, p = 0.014; measurement 2: r = 0.27, p = 0.017 ; the left buttock measurement 1: r = 0.28, p = 0.016; measurement 2: r = 0.34, p = 0.003). However, there was no statistically significant correlation between the body mass and the change in PIP over the two measurements.

Discussion

The main objective of this study was to determine the reproducibility of the TIP measurements in patients with the aim to establish whether this parameter could be used in clinical practice in a PU risk assessment model [19]. The results demonstrated that the "between day" reproducibility is very low and therefore, based on these results and this methodology, the TIP cannot be used as a parameter for this purpose.

This absence of an adequate reproducibility can be caused by the fact that the patients were allowed to place the backrest of their bed in the, for them at that moment, the most comfortable position. As being comfortable can change over the observation period, none of the backrest positions were the same in one patient during the two measurements. This finding is supported by the fact that the results also indicated that the change in the backrest elevation was strongly correlated with the change in the PIP. The actuality that the repeated measurements were carried out at different intervals probably had less influence on the reproducibility of the PIP value in the group of patients in whom the measurement was repeated after 24 hours, a low ICC was already present. It is evident from the results of this study that when patients change their position in bed (even during bed rest) it causes in a significant variation in the PIP.

Sugama *et al.* [20] developed a multi-pad pressure sensor which can be used to measure the TIP more easily in patients compared with the equipment we used in this study. Although the author admitted that there is no simple relationship between the exerted pressure and the pressure needed to constrict capillaries, after measuring the TIP at the sacrum in 79 elderly Japanese patients, they suggested that maximum pressures varying from 5.3 kPa - 6.6 kPa may be tolerated before a PU develops. The "between day" reproducibility of the measurement was not established and therefore, the value of the measured TIP for predicting PU development in clinical practice is debatable as it is unknown whether the measured TIP will vary over time.

Only a few other researchers have investigated the "between day" reproducibility of their measurements. Most of them were carried out in healthy volunteers. To date the "between day" reproducibility of the TIP has not been investigated in any other clinical study. Allen et al. [10] investigated the reproducibility of IP measurements in 6 healthy volunteers. Measurements were carried out four times a day over four consecutive days at 6 body sites (occiput, scapula, elbow, sacrum, buttock and heel) in supine position without a pillow on a foam mattress. They noted an average difference of 5.4 mmHg (0.72 kPa) in the "between day" measurements and 4.8 mmHg (0.64 kPa) in the same day between repeat measurements. They evaluated the reproducibility by comparing the means and the mean SDs of the measured TIP at group level. However, the results provided no information on the reproducibility of the actual TIP measurements in the individual patient. Based on the findings of that study, the investigators conducted 2 additional studies in which TIP measurements were carried out over four consecutive days on various mattresses [11, 12]. Unfortunately, no data in relation to "between repeats" reproducibility and "between day" reproducibility was reported.

In our study the highest PIP value was generally measured at the sacrum compared with the buttocks. No other clinical studies reported results suitable for comparison because in most studies the TIP was investigated at the sacrum, the trochanter and the heel and not at the buttocks[21]. Allen et al. [10, 11] and Ryan et al. [12] reported the opposite in healthy volunteers and measured the lowest TIP at the sacrum compared with the buttocks. Maklebust et al. [22] reported that healthy volunteers had good tone in the gluteal musculature. This tends to elevate the sacrum from the support surface on which the person is reclining resulting in low TIP readings at the sacrum. Another explanation may be the presence of the Hawthorne effect when measuring the TIP in healthy volunteers. When participating in a study, they are informed on its purpose. This can subconsciously alter their weight distribution by pressing their shoulders more in the mattress which will alter the pressure exerted by the pelvic area on the mattress. This is less likely to occur in a clinical situation as in our study because of the frequently poor physical condition of the patients. This in turn determines the way these patients position themselves in bed. In most cases, the most comfortable position is determined by the absence of pain.

The observation that the backrest elevation was significantly correlated with the level of the PIP at the sacrum and the buttocks was also reported in another study [23]. In contrast, we observed a positive correlation between the elevation of the backrest and the PIP at the sacrum and the right buttock. Defloor [24] reported no differences in the PIP measured in healthy volunteers on a polyethylene-urethane mattress with the backrest angles of 0^0, 30^0 and 60^0. The difference with our study is that Defloor et al [24] used a pressure mapping system and did not distinguish between the sites (the sacrum and the buttocks) where the pressures were measured. Therefore, the movement of peak pressures from the sacrum towards the buttocks was not identified.

The mean PIP values of measurements one and two did not differ significantly from each other. This observation supports the suggestion that TIP measurements may be

used for assessing the pressure-reducing capacity of a mattress in a varied patient population. Currently, this is carried out in healthy volunteers only [21, 25]. In a limited number of studies the TIP was measured in a patient population [26-29]. Working group 2 of the European Pressure Ulcer Advisory Panel has produced guidelines for assessing the pressure-reducing capacity of mattresses. They suggested to use mannequins as these can be easily adapted for different positions but in order to obtain reliable measurements these should be repositioned at least 6 times during 6 consecutive measurements [7]. Although this may be beneficial in producing a ranking of pressure-reducing capacity of various mattresses, a clinical evaluation with the, for example, top three mattresses must provide the ultimate evidence on the pressure-reducing capacity of the mattress.

Considering the typical patient characteristics like body mass, length, height and indexes (BMI and BSA), we observed a significant positive correlation between the body mass of a patient and the PIP at the right and the left buttocks. This is in contrast to the results by Rojas & Reynolds [30], who noted no relationship between the body mass and the generated IP. However, when the body mass of the patient was correlated to the change in the PIP, no significant correlation was noted indicating the limited value of this parameter.

A significant limitation of this study was the large diversity of patients who participated in relation to mobility and activity; using a more homogeneous patient group might produce different results. Repeating the study in patients who do not or are unable to move for longer periods (particularly intensive care patients, patients with spinal cord injuries and neonates) without changing the position of the backrest and fixed moments of measurement (12 hours, 24 hours, 48 hours and 72 hours) may produce TIP values that are more reproducible.

Conclusion

It is evident from the results of this study that the PIP value is not reproducible in a diverse patient population considering their activity and mobility. This variation in PIP is mainly caused by the degree of the backrest elevation. As the majority of patients with bed rest vary their position regularly, the TIP will also vary. Research addressing the reproducibility of TIP measurements in patient groups with similar activity and mobility is needed to investigate whether the TIP value can be used as a parameter in a pressure ulcer risk assessment model.

Analysis of the average PIP measured at the sacrum and the buttocks in patients indicated that this parameter may be used to evaluate the pressure-relieving characteristics of patient support surfaces.

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Suppliers
a   Talley Group Ltd, Premier Way, Abbey Park Industrial Estate, Romsey, Hampshire SO519AQ, England, UK
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Literature

1. CBO., Richtlijn Decubitus, tweede herziening. 2002, Utrecht: Kwaliteitsinstituut voor de gezondheidszorg (CBO). 132.
2. Xakellis GC, Jr., Frantz RA, Lewis A and Harvey P. Cost-effectiveness of an intensive pressure ulcer prevention protocol in long-term care. Adv Wound Care 1998;11:22-9.
3. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40:61-9.
4. Wywialowski EF. Tissue perfusion as a key underlying concept of pressure ulcer development and treatment. J Vasc Nurs 1999;17:12-6.
5. Reswick JB and Rogers J, Experience at Rancho los Amigos Hospital with devices and techniques to prevent pressure sores. Bedsore Biomechanics, ed. JMC R.M. Kenedi, J.T. Scales. 1976, Baltimore: University Park Press. 301-10.
6. Schoonhoven L, Haalboom JR, Bousema MT, *et al.* Prospective cohort study of routine use of risk assessment scales for prediction of pressure ulcers. BMJ 2002;325:797.
7. McLeod A. Draft guidelines for the laboratory evaluation of pressure - redistributing support surfaces. EPUAP Rev 2002;4:8-11.
8. Hobson DA. Comparative effects of posture on pressure and shear at the body-seat interface. J Rehabil Res and Dev 1992;29:21-31.
9. Allen V, Ryan DW, Lomax N and Murray A. Accuracy of interface pressure measurement systems. J Biomed Eng 1993;15:344-8.
10. Allen V, Ryan DW and Murray A. Repeatability of subject/bed interface pressure measurements. J Biomed Eng 1993;15:329-32.
11. Allen V, Ryan DW and Murray A. Measurements of interface pressure between body sites and the surfaces of four specialised air mattresses. Br J of Clin Pract 1994;48:125-9.
12. Ryan DW, Allen V and Murray A. An investigation of interface pressures in low air loss beds. Int J Clin Pract 1997;51:296-8.
13. van Dijk D, Aufdemkampe G and van Langeveld S. The QA pressure measurement system: an accuracy and reliability study. Spinal Cord 1999;37:123-8.
14. Wolsley CJ and Hill PD. Review of interface pressure measurement to establish a protocol for their use in the assessment of patient support surfaces. J Tissue Viability 2000;10:53-7.
15. Abruzzese RS. Guidelines for evaluating decubitus ulcer research. Decubitus 1989;2:28-32.
16. Bader DL and Hawken MB. Pressure distribution under the ischium of normal subjects. J Biomed Eng 1986;8:353-7.

17.   Rithalia SV, Heath GH and Gonsalkorale M. Assessment of alternating-pressure air mattresses using a time-based pressure threshold technique and continuous measurements of transcutaneous gases. J Tissue Viability 2000;10:13-20.

18.   Spijkerman DC, Terburg M, Goossens RH and Stijnen T. Effects of infaltion pressure and posture on the body-seat interface pressureof spinal cord injured patients seated on an air-filled wheelchair cushion. J Rehabil Sc 1995;8:8-12.

19.   Weststrate JT, Hop WC, Aalbers AG, Vreeling AW and Bruining HA. The clinical relevance of the Waterlow pressure sore risk scale in the ICU. Intensive Care Med 1998;24:815-20.

20.   Sugama J, Sanada H and Takahashi M. Reliability and validity of a multi-pad pressure evaluator for pressure ulcer management. J Tissue Viability 2002;12:148-53.

21.   Whittemore R. Pressure-reduction support surfaces: a review of the literature [see comments]. J Wound Ostomy Continence Nurs 1998;25:6-25.

22.   Maklebust J, Mondoux L and Sieggreen M. Pressure relief characteristics of various support surfaces used in prevention and treatment of pressure ulcers. J Enterostomal Ther 1986;13:85-9.

23.   Rondorf-Klym LM and Langemo D. Relationship between body weight, body position, support surface, and tissue interface pressure at the sacrum. Decubitus 1993;6:22-30.

24.   Defloor T. The effect of position and mattress on interface pressure. Appl Nurs Res 2000;13:2-11.

25.   Fletcher J. What can we learn from interface pressure measurements. J Wound Care 2001;10:29-32.

26.   Berjian RA, Douglass HOJ, Holyoke ED, Goodwin PM and Priore RL. Skin pressure measurements on various mattress surfaces in cancer patients. Am J Phys Med 1983;62:217-26.

27.   Clark M and Rowland LB. Preventing pressure sores: matching patient and mattress using interface pressure measurements. Decubitus 1989;2:34-9.

28.   Sideranko S, Quinn A, Burns K and Froman RD. Effects of position and mattress overlay on sacral and heel pressures in a clinical population. Res Nurs Health 1992;15:245-51.

29.   Suarez CH and Reynolds A. Pressure reduction with a hospitalized population using a mattress overlay. Ostomy Wound Management 1995;41:58-60, 2-3.

30.   Rojas MG and Reynolds A. Pressure-reducing capability of the Therarest hospital replacement mattress. J Wound Ostomy Continence Nurs 1996;23:100-4.

31.   Haalboom JR, van Everdingen JJ and Cullum N. Incidence, prevalence and classification, a survey of the literature. In The Decubitus Ulcer in Clinical Practice, P L.C., J.A. W and J.T. C, (Editors). 1997, Springer-Verlag: Heidelberg. p. 12-23.

32.   WHO, Physical status: The use and interpretation of anthropometry. 1995, World Health Organization: Geneva.

Chapter 6

A comparison of interface pressure measurements
between patients and healthy volunteers in lying
positions

Jan T.M. Weststrate[1]

Wim C.J. Hop[2]

Freerk Heule[3]

Tom Defloor[4]

Hajo A. Bruining[1]

1. Surgical Intensive Care, Erasmus MC, University Medical Center, Rotterdam
2. Department of Epidemiology and Bio-statistics, Erasmus MC, University Medical Center, Rotterdam
3. Department of Dermatology & Venereology, Erasmus MC, University Medical Center, University Hospital, Rotterdam
4. Department of Nursing Sciences, Ghent University, Gent

Abstract

The purpose of this study was to evaluate whether Tissue Interface Pressure (TIP) measured in healthy volunteers and in patients lying on the same type of mattress are equivalent. Therefore, TIP was measured at the sacrum and the buttocks in 28 patients and 30 healthy volunteers. In patients, the measurements were carried out in their preferred position at that moment and in healthy volunteers they were carried out in the three most used positions: supine, backrest elevated to 30^0 and backrest elevated to 30^0 with 22^0 Fowler's position. The body mass of both groups was equally distributed and was between 50 and 125 kg. Measurements were carried out with the Talley Pressure Monitor III (TPM III) and the values were expressed in kPa.

Results showed that the TIP values at the sacrum (7.3 kPa) and the left buttock (6.1 kPa) in patients were significantly higher than those measured in all three positions at the sacrum (4.0 kPa; 3.5 kPa; 2.5 kPa respectively for each position) and the left buttock (3.3 kPa; 4.0 kPa; 4.4 kPa, respectively for each position) in healthy volunteers ($p < 0.05$). When the maximum TIP values over the pelvic area of each patient and volunteer were compared, it became evident that averaging TIP pressures at site level may obscure high local TIP values. Again in patients the maximum TIP values were significantly higher compared with those in healthy volunteers (7.9 kPa vs 4.5 kPa, 5.1 kPa and 4.9 kPa, respectively for each position) ($p < 0.01$). In patients the maximum pressure measured at the buttocks and the sacrum was the same. In the healthy volunteers the site of maximum pressure depended on the position.

The implications of these results are that evaluation of pressure distribution ability of a mattress is best carried out in patients instead of volunteers as is currently done. Measuring the TIP at the sacrum only is also not always revealing the maximum TIP exerted in a particular patient.

Introduction

The development of a pressure ulcer (PU) in patients during their stay in hospital always has adverse consequences like pain, discomfort, prolonged hospital stay [1], increased risk of infection [2] and death [3]. For the nurses and the physicians it causes an increased workload [4, 5]. For the hospital organization it leads to increased costs [5-7]. Although prevention is not free, it is still cheaper compared with treating the accumulated burden of PU [8].

The increased risk of developing a PU is often made visible through a PU risk assessment instrument [9]. Although recent research questions the validity of such an instrument in predicting PU development, it is still used widely and provides the nurses with practical insight into the patient's risk of developing a PU [10].

The available conceptual schemes indicate that pressure and shear forces are the primary causes of PU development [11, 12]. As shear forces are currently difficult to measure clinically, over the years more attention has been paid to the influence of pressure. The most direct method to measure this parameter is by placing sensors just below the skin of the subject. As this invasive method is rather cumbersome, measuring the pressure (Tissue Interface Pressure (TIP)) at the interface of patient and the mattress was also shown to be reliable [13]. Since the human body is not flat, the TIP will vary depending on the site at which it is measured. The more protruding parts of the body, because they contain bony structures close under the skin such as the scapula, the sacrum, the buttock, the heel and the trochanter, will have higher TIP values compared with the flat parts of the body. The shape of the body can also change during a period of severe illness [14] or from being in the same position for a long time [15].

Following this concept, the basis of all PU prevention strategies is lowering the TIP at the protruding sites of the body. The nurses can decide on the basis of the PU risk level and / or inspection of the skin, when and which type of PU prevention strategy is suitable, varying from repositioning patients regularly every two hours to using a special mattress [9]. Although repositioning the patient every two hours is often the first step in PU prevention, it is often difficult to maintain such a schedule on an around the clock basis certainly in intensive care patients [16]. In addition, Clark [17] questioned the effectiveness of repositioning patients every two hours as there is hardly any scientific evidence which endorses this time interval. On the one hand, Knox [18], based on skin temperature measurements, recommended to the turn patients every one and a half hour, or even every hour, when at the end of this time frame redness occurred. On the other hand, Meijer et al. [19] concluded from their investigations that frequent repositioning may even result in applying a pressure load to a specific site that has not fully recovered from an earlier pressure load, especially in patients who have co-morbidities such as diabetes [20]. Besides this, clinical practice showed that patients who need repositioning most, are often in such an unstable clinical condition that they are less likely to be turned at all [21]. From an economic point of view Xakellis et al. [8] found that the time involved in repositioning

patients every two hours was a more expensive intervention in the prevention of PU compared with the costs of using special pressure-reducing mattresses. Defloor [22] reported that PU could be effectively prevented by repositioning the patient every four hours instead of every two hours if this was carried out in combination with the use of a polyurethane hospital mattress. All this indicates that the effectiveness of interventions such as repositioning strongly depends on the pressure-reducing ability of the mattress.

Many manufacturers of mattresses, and investigators use healthy volunteers in order to evaluate the pressure-reducing ability of a mattress by measuring the TIP. Whittemore [23] reviewed 22 studies in which TIP was measured to assess the pressure-reducing ability of a large range of mattresses. The TIP was measured in an actual patient population [24] (n=17) [25] (n=18) [26] (n=57) in only three of the 22 studies. In the other 19 studies, an average of 19 volunteers per study (range 6-64) was tested.

It is unknown whether a healthy volunteer can truly substitute a patient to evaluate the pressure-reducing ability of a mattress. Maklebust *et al.* [15] stated that healthy volunteers have good gluteal musculature tone, which tends to elevate the sacrum from the mattress. This results in a lower TIP being exerted on the skin at the sacrum, in contrast to patients who may be debilitated and lack good tone in the gluteal musculature. Following this concept Berjian *et al* [14] noted that the TIP at the sacrum and the trochanter in healthy volunteers was lower compared with cancer patients. Clark and Rowland [27] evaluated the TIP that was produced by healthy volunteers (mean age 19.8 years) and hospital patients (mean age 82.2 years) on a foam mattress and air mattress . They observed that hospital patients had a 41% higher TIP compared with the TIP that was produced by healthy volunteers on the same mattress.

As two thirds of all PUs occur in the pelvic region, in most mattress evaluation studies the TIP was measured at the sacrum and the trochanter. [14, 15, 25, 26, 28-32]. Only a limited number of investigators measured the TIP also at the ischial tuberosities [31-33]. Measuring the TIP at the ischial tuberosities becomes more relevant when the backrest of the bed is elevated. As the human body has two ischial tuberosities (left & right) and both are not always equally loaded, from a balance point of view it is important to measure both sides in order to be certain to obtain the highest TIP value. These two measurements were not carried out routinely in all mattress studies [31-33]. It is evident that all human beings have different shapes, especially in the pelvic region. Some will have a more protruding sacrum and others more protruding ischial tuberosities, but most mattresses do not have other material support for the pelvic region. Therefore, it is questionable whether the measurement at one site can be extrapolated to others and also to contra lateral sites.

Whether the TIP should be measured in patients or healthy volunteers for assessing the pressure-reducing ability of a mattress is still a matter of debate. The outcome of such an investigation may be important for both the clinical practice (users) and the manufacturers (suppliers). If such investigations show that TIP measurements in healthy volunteers can be used for assessing the pressure-reducing ability then, the manufacturers

should request independent standardization institutes to test the pressure-reducing ability of their mattresses or they should co-operate with clinicians.

In order to analyse the relevance of the TIP, we designed a study in which we compared the TIP measured in a group of healthy volunteers with those in hospitalized patients on a standard hospital mattress. As the elevation of the backrest was shown to influence the measured TIP value [34], the effect of this extraneous variable was taken into account. The influence of the Body Mass Index (BMI) of the patient or the volunteers on the magnitude of the TIP was also investigated in the past [35]. Therefore, the influence of this factor was also taken into consideration.

The following questions were formulated:

1. Is there a statistically significant difference between the maximum TIP measured in patients and volunteers at the specific body sites (the sacrum and the right and the left buttocks)?

2. Is there a statistically significant difference between the measured maximum TIP over the three sites (the sacrum, the right and the left buttock) between patients and volunteers?.

3. Which factors (age, BMI, backrest elevation) influence the TIP significantly in patients and in volunteers?

Materials and Methods

Patients

A convenient sample of 28 patients was selected for this study. All patients were admitted to the medical and surgical wards of the ErasmusMC, University Medical Center, Rotterdam, The Netherlands. Participants were selected and stratified according their body mass in one of the three categories (50kg - 74.9kg; 75kg - 99.9kg; 100kg - 125kg). The aim was to have at least 8-10 patients in each category. The category 100kg - 125kg had 8 patients. The two other categories had each 10 patients.

Participating healthy volunteers were recruited from within- and outside the center. Participants were selected and stratified according to their body mass in one of the three categories. Each category had 10 volunteers.

Patients and healthy volunteers were positioned on a standard hospital mattress (Tempur Pedic®)[1]. This is a 14 cm thick mattress made of a 4 cm top layer of visco-elastic polyurethane and a 10 cm bottom layer of cold foam. The whole mattress has a washable cover around it consisting of a semi-permeable layer of 80% cotton and 20% polyester. The study was approved by the Medical Ethics Committee of the Erasmus MC, University Medical Center.

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[1] Fagerdala, Sweden. Manufactured by Dan/Foam A-S, Denmark

Instruments

The Talley Pressure Monitor III (TPM III)[2] was used to measure TIP. The concept of this device was developed at the Oxford Orthopaedic Engineering Centre, Nuffield Orthopedic Centre, Oxford, United Kingdom. Four studies have been published using the TPM III for measuring TIP in patients as well as in and the volunteers [36-39]. The TPM III consists of three basic components: a pneumatic transducer array, a monitor and calibrating equipment. It is able to connect to eight arrays, each containing 12 connected (array) or separate (single) sensors (sensor 20 mm in diameter). Calibration data published by the developers show a maximum deviation from linearity of 3 % over the optimum range of 0-33.33 kPa [40]. We used the TPM III with three single sensors similar to that in the volunteer group reported by several other investigators [31, 32, 39, 41-45]. We used the TPM III with three arrays, each containing 12 connected sensors (3 x 4) (see appendix 3) because the patients found it more difficult to lie still during the measurements. This technique was also used by other investigators for evaluating mattresses [33, 37]. All sensors were calibrated weekly during the study.

Procedure

The arrays (AR) or single sensors (SS) were positioned under the sacrum and the right and the left buttocks of the patients and the volunteers. The position of the three AR/SS was standardized. The individual was asked to turn onto the left side. The sacrum was located by palpation and the AR/SS was held in place by the investigator's hand while the patient or the volunteer was asked to turn back into the supine position. The next AR/SS was placed under the right buttock by asking the patient or volunteer to turn onto the left side while making certain that the AR/SS placed under the sacrum remained in place. The ischial tuberosity of the right buttock was located by palpation and the AR/SS was held in place by the investigator's hand while the patient or volunteer was asked to turn back into the supine position. The same procedure was repeated for positioning an AR/SS under the left buttock. If patients wore pyjamas and / or underwear the AR were placed over this, thus not in direct contact with the skin. If the patient wore an incontinence pad, the AR was placed between the mattress (with a sheet) and the pad. Information on what the patient was wearing at the time of the measurement was recorded on the measurement data form. The patients were then instructed to put the backrest of their bed in the most comfortable position while lying with their head on a pillow, to put their hands along side their body and their legs uncrossed and stretched out. Further it was stressed to keep body movements to a minimum during the time of measurement.

The volunteers wore their own underwear and standard cotton hospital trousers. The measurements in the volunteer group were carried out in three supine positions. Position 1: backrest in 0^0, position 2: backrest in 30^0 and position 3: backrest in 30^0 with 22^0 Fowler's position. All volunteers had one pillow under their head during the measurements.

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[2] Talley Group Ltd, Premier Way, Abbey Park Industrial Estate, Romsey, Hampshire SO519AQ, England, UK.

In order to obtain the most accurate clinical measurements, the arrays were placed between the patient and the bed sheet over the standard hospital mattress.

The first ten minutes of the measurement were used to allow the measurement equipment and visco-elastic mattress to reach steady state. In the meantime it was checked if any of the sensors produced values that indicated a false measurement due to bending or malfunctioning. In the patient group it was not always possible to reposition the sensor as this involved turning the patient. This happened in four patients when the TIP was measured at the sacrum. These TIP values were excluded from analyses.

In total 36/3 sensors measured the TIP between the patient/volunteers and the mattress: 12/1 sensor(s) in the sacral area, 12/1 sensor(s) in the area under the right buttock and 12/1 sensor(s) in the area under the left buttock. While measuring the TIP, the patient or the volunteer was asked to stay in the same position and lie as still as possible for ten minutes. During this period the actual TIP was measured continuously. These TIP measurements were performed in consecutive order starting in the sacral area and ending at the left buttock. The AR/SS were removed and cleaned when the measurement procedure was finished.

As at least 10 measurement frames were performed within each procedure, the record file contained a list of 36 x 10 = 360 / 3 x 10 = 30 interface pressure values in kPa. The records were transported and statistically analyzed using SPSS (version 11.1).

The mean TIP value and the standard deviation were calculated for all arrays and single sensors at each site from each measurement. In the patient group the sensor with the highest mean TIP value at each body site was entered into the database as the Peak mean Interface Pressure (PIP-Patient). As the volunteer group used single sensors for each site the mean TIP value was calculated for each sensor at each site and entered into the database as the PIP-Volunteer.

Statistical methods

Demographic values between the two groups were compared using the Fisher exact test or the unpaired T-test. Comparison of the patient values with the volunteer values at the separate sites was done by the unpaired T-test. Comparison of PIP within subject groups (patients and volunteers) between the three sites was done using repeated measurements ANOVA. For the maximal PIP the same method was used within the volunteer group for the comparison between the three positions and to investigate the relations with BMI. The Fisher exact test was used for comparison of the difference in site of the maximum PIP between patients and volunteers. The correlation coefficients are Pearson's. $P < 0.05$ (two-sided) was considered the limit of significance.

Results

An overview of the demographics of the patient and the volunteer groups is shown in Table I. The age distribution between the volunteers and the patients differed. Although the mean height of the patients and the volunteers differed significantly there was no statistically difference in the BMI as the body mass was used to stratify the patients and the volunteers. The average elevation of the backrest in the patient group was 20.8^0 including five patients in completely horizontal position (0^0 elevation). The mean Fowler's elevation was 2.9^0 as only four patients used this position with a range of 17^0-22^0. The majority of the patients wore their underwear and pyjamas during the measurements. Only three patients wore incontinence pads during the measurement. The coefficient of variance for the mean TIP value of each sensor in the patient group was 5.2% and for the healthy volunteer group was 3.5%.

Table I.   Means (± SD) of demographic characteristics of the patients and the volunteers

	Patients	Volunteers	Sign.
N	28	30	
Male / Female	12/16	15/15	p =0.799
Age in years	62.2 (±12.3)	32.4 (±11.7)	p < 0.001
Body mass in kg	82.6 (±17.3)	87.1 (±20.6	p = 0.378
Height in cm	171.4 (±9.6)	177.9 (±10.8)	p = 0.018
BMI	28.1 (±5.4)	27.6 (±5.9)	p= 0.740
Elevation Backrest in degrees	20.8 (±14.8)	0 and 30	NT
Elevation Fowler in degrees	2.9 (±7.3)	22 and 30	NT
Presence of underwear	21	30	NT
Presence of pyjamas	9	30	NT
Presence of incontinence material	3	0	NT

NT: not tested; BMI: Body Mass Index

The mean PIP at the three sites in the patients and the volunteers are shown in Table II. Comparisons between sites within the patient group and within the three separate positions for the volunteer group are shown. The average PIP at the sacrum and the left buttock in the patients were significantly higher than those in all three positions in the volunteer group (p < 0.01). The average PIP in the patients at right buttocks was only significantly higher than the average PIP in the volunteers lying flat (0^0) (p < 0.05).

Within the volunteer group the PIP at the sacrum only in the first position was not significantly different compared with that in the second position (p = 0.106). The PIP

measured on the right buttock in the second position was not significantly different from that in the third position (p = 0.373). Further, all PIP values in the three positions differed from each other (p < 0.05).

Table II.   An overview of mean PIP, mean maximum PIP and the number of maximum PIP in patients and volunteers at three sites in different positions (± SD)

Site	Patients*	Volunteer Position 1	Volunteer Position 2	Volunteer Position 3
Sacrum (kPa)	7.3 (± 2.8)[b,c,]	4.0 (± 1.3)[c]	3.5 (± 1.6)[b]	2.5 (± 1.2)[b,c]
Buttock right (kPa)	5.2 (± 1.6)[a,c]	4.0 (± 0.7)[c]	4.7 (± 1.2)[a,c]	4.8 (± 0.8)[a,c]
Buttock left (kPa)	6.1 (± 1.7)[a,b]	3.3 (± 0.5)[a,b]	4.0 (± 0.5)[b]	4.4 (± 0.5)[a,b]
Mean maximum PIP (kPa)	7.9 (± 2.4)	4.5 (±0.9)	5.1 (±1.1)	4.9 (±0.8)
Maximum PIP sacrum (n)	19	15	6	1
Maximum PIP buttocks (n)	9	15	24	29

Note 1: * variable positions see text

Note 2: significant differences within each group between the three sites is indicated as; a: p< 0.05 versus sacrum, b: p< 0.05 versus buttock right, c: p< 0.05 versus buttock left)

Note 3: Position 1: backrest 0^0; position 2: backrest 30^0; position 3: backrest 30^0 and Fowler 22^0

As was hypothesized, if a PU will develop, it will do so at the site that endured the maximum PIP for the longest period. Therefore, the maximum PIP was selected in each patient and each volunteer for each position. The measured pressures in the three positions in the volunteer group were compared with the corresponding pressures measured in the patient group. The maximum PIP in patients differed significantly from the maximum PIP measured in volunteers for all three positions (p < 0.01). In the volunteers, the maximum PIP in position 1 differed significantly from those in the second and the third positions (p < 0.05). There was no significant difference between the maximum PIP in the second and the third positions. The maximum PIP in the majority of the patients (19/28) was at the sacrum. In the volunteers this varied according to the position. There were significantly more patients with maximum pressure at the sacrum compared with the second and the third positions in the volunteers (p < 0.01).

There was a significantly positive correlation between the maximum PIP and backrest elevation in the patients (r = 0.66; p < 0.001). No significant correlation was noted between the maximum PIP and the BMI in the patients. In the volunteers, the BMI correlated with the maximum PIP (resp. r = 0.51 (p < 0.01); r = 0.33 (p < 0.05) and r =0 .59 (p < 0.01) for the positions 1, 2 and 3 respectively). These three relations did not differ significantly (p = 0.80) from each other and the increase in PIP was 0.08 (± 0.02 sem, p < 0.001) kPa per unit of BMI (kg/m^2).

Discussion

The most noteworthy finding of this study was that TIP values in the patients were higher than those in the volunteers. This was specifically the case for the TIP values at the sacrum and the left buttock. The maximum PIPs in the pelvic area in the patients were significantly higher than those in the volunteers. This supports the view that when healthy volunteers are used for assessing the pressure-reducing ability of a mattress lower TIP values will be generated compared with those in the patients. We found one study in the literature in which the TIP at the sacrum in patients with cancer and healthy volunteers on the same type of mattress with the same type of TIP measuring instrument were investigated [14]. The investigators observed no significant difference between the TIP measured at the sacrum in the patients and those measured in the healthy volunteers.

A possible explanation for higher TIPs measured at the sacrum in the patients than the TIPs in the volunteer group was given by Maklebust *et al*. [15]. They reported that the reclining healthy volunteers showed a good gluteal musculature tone which caused the sacrum to elevate from the mattress whereby lower TIPs were generated. This is in contrast to patients who often have prolonged periods of bedrest and are generally physically weakened, whereby good gluteal muscle tone is lacking which causes the sacrum to force itself into the mattress and generate higher TIP values at the sacrum. This view is further supported by the high number of patients in our study who had the highest maximum PIP at the sacrum compared with the lower number of maximum PIPs in the volunteers in the three positions.

Maklebust *et al.* [15] also reported that TIP measurements are best performed in patients, but they are often reluctant to participate. In our study, most of the patients were co-operative and had no problem participating in the study. The reason for this may be that the TIP was measured in the patient's most comfortable position. For the patient this is also the most relevant position for assessing the pressure distributing capacity of a mattress. Furthermore, the high PIP measured at the sacrum compared with the PIP measured at the other sites may also be an explanation for the development of most PUs in the sacral area [46].

Maklebust *et al.* [15] further suggested that measuring the TIP in healthy volunteers at the trochanter will produce more reliable information on the pressure-reducing capacity of a mattress because this is a protruding part of the body that exerts more pressure on the mattress. Measuring the TIP at the trochanter indicates that patients spend considerable time on a mattress in the 90^0 lateral position. As most PUs develop in the sacral area, there is no evidence that TIP measurements at the trochanter provide more reliable information on the pressure-reducing capacity of mattresses. In addition, Seiler *et al.* [47] recommended to position patients 30^0 laterally instead of 90^0 when positioning them on alternate sides to prevent the development of a PU. In this position the pressure is not exerted on the trochanter or the sacrum but on the soft tissue between the two sites. They reported that soft tissue may tolerate up to 3.5 times as much pressure compared with that on bony sites.

Recent research has shown that the lower TIP measured at 30^0 laterally compared with 90^0 laterally also depended on the type of mattress [34]. Furthermore, placing the patients at 30^0 laterally showed not to compromise the health status of hemodynamic and respiratory unstable patients,[48] which may worsen when they are positioned 90^0 laterally.

Besides the sacrum, the TIP at the buttocks was investigated in three studies [31-33]. In all these 3 studies, higher TIPs at the buttocks than those measured at the sacrum were reported. In our study, a significantly high number (19) of the patients had a higher TIP at the sacrum compared with those at the buttocks. This is again an indication that the patients lacked good gluteal muscle tone which caused the sacrum to press more into the mattress. The PIP measurements at the right buttock in the volunteers appeared to generate the highest values compared with the values at the left buttock. This may be explained by the fact that the TPM III and the investigator were always standing on the right side of the patient. The volunteer possibly emphasized the right side by turning to the investigator and in doing so increased the pressure exerted on the right buttock unintentionally. It was not so that the patients found it more difficult to lie still during the measurement because the TPM III was placed on the different side of the patient's bed. Moreover, because of their illness, patients probably pay more attention to their physical well being than to the investigator and his equipment.

When the maximum PIP is analysed in each patient and each volunteer, the mean maximum PIP is higher than the mean PIP measured at each of the three sites separately. This indicates that by averaging the TIP for each site separately, the TIPs are artificially lowered. This provides a misleading impression on the interaction between the patient and the mattress. Besides, the study mattress has the same properties over its entire surface and is no different in the pelvic area and therefore, there is no reason for the higher pressure at the buttocks compared with the sacrum or vice versa. Thus, analysing the pressure-reducing capacity of the mid-section of a mattress is best carried out by evaluating the maximum PIP in the pelvic area (the sacrum and the buttocks).

Besides the height, the age of the volunteers was significantly lower compared with that in the patients. There appeared to be no relationship between the maximum PIP and the age. In contrast, Clark and Rowland [27] reported that elderly patients had a significantly higher TIP than the healthy young volunteers. Two important findings can possibly explain the difference with our investigation. The mean age of the elderly in the Clark and Rowland [27] study was higher than 80 years compared with 62.2 in our study. Next, the mean body mass of their elderly group was below 53.4 kg, whereas in our study this was 82.6 kg. Other investigators suggested that patients with a low BMI have less padding over bony prominences like the sacrum and therefore, generate a high TIP. There is no consensus in the literature on the influence of body mass or BMI on the TIP. Berjian et al [14] suggested that the patients with a low BMI exert higher TIPs. However, Rondorf-Klym and Langemo [24] could not find a significant difference in the TIP measured between the elderly patients with and without an ideal body mass. The results of our study showed that in the patients, there was no relationship between the maximum PIP and the BMI. In

the volunteers there was generally a significant relationship between the BMI and the maximum PIP for all the three positions. As yet, the exact influence of the BMI on the TIP remains unclear.

There was a strong correlation between the elevation of the backrest and the level of the maximum PIP in the patients. This relationship has also been reported by other investigators [26, 34, 49, 50]. This could not be evaluated in the volunteers as they were placed in three fixed positions. It was evident in the volunteers that when the backrest was elevated to 30^0 or in combination with 22^0 Fowler's position, the PIP at the sacrum was decreased and the PIP at the buttocks was increased. Specifically in position three (backrest 30^0 and 22^0 Fowler's position) there is a maximum lift of the sacrum area as the contact area increases when the legs also carry some of the weight of the pelvic area.

Apart from human test subjects (patients and volunteers), a number of other non-human test devices are mentioned in the literature that have been used to evaluate mattresses. These devices involve the use of indentors [51] and mannequins [50, 52]. The main advantage of using non- human test devices, is a high experimental reproducibility. This is not the case in patients because they are unable to act consistently over time [53]. Therefore, the European Pressure Ulcer Advisory Panel installed a working group which investigated which test device is the most appropriate for evaluating the pressure redistribution properties of mattresses relative to a standard surface [54]. They concluded that for the comparative ranking of the pressure-reducing capacity of a mattress, a human-like mannequin should be used. Measuring the TIP using a human-like mannequin enables researchers to obtain a high experimental reproducibility in the measurements with the currently used TIP measurement instruments. The main purpose of this is to show the reliability of the measurement technique and produce specific mattress-related measurements. The mannequins are not yet commercially available. Therefore, the results cannot be generalized for all types of mannequins and do not reflect the clinical situation. Human beings move and can vary the pressure on the mattress influencing the TIP. The value of TIP measurements using a human-like mannequin for clinical purpose is limited. All human beings have different shapes, weight distributions and favor different body positions on a mattress. Therefore, clinicians are more interested in the pressure-reducing capacity of mattresses expressed as the TIP measured at the most protruding parts of the body such as the sacrum and the buttocks in patients.

The results of this study clearly showed that the patients produce higher PIPs then the volunteers on the same mattress. This implies that the assessment of the pressure-reducing capacity of a mattress used for patients is only possible if the TIP is measured with patients in a clinical setting. In such an assessment, it is the best to evaluate the maximum pressure at the three sites, (the sacrum and the left and the right buttocks) in the pelvic area of each patient.

Further studies in this field should address the differences in PIP between the patients and the volunteers in population older than 70 years because of the generally increasing age of hospitalized patients. Clinical research should also address the question

whether the site with the maximum PIP is also the site at which pressure ulcers develop. In addition, the clinical implications of the discrepancy in the measurements of PIP between the patients and the volunteers should be investigated more thoroughly.

In conclusion, this study may act as a model for the manufacturers of mattresses and encourage them to test their products in patient studies prior to marketing such products. Moreover, other factors such as safety features, patient comfort and product service requirements can also be investigated.

Literature

1. Hibbs P. The economics of pressure ulcer prevention. Decubitus 1988;1:23-38.
2. Jiricka MK, Ryan P, Carvalho MA and Bukvich J. Pressure ulcer risk factors in an ICU population. Am J Crit Care 1995;4:361-7.
3. Thomas DR, Goode PS, Tarquine PH and Allman RM. Hospital-acquired pressure ulcers and risk of death. J Am Geriatr Soc 1996;44:1435-40.
4. D'Hoore W, Guisset AL and Tilquin C. Increased nursing-time requirements due to pressure sores in long-term- care residents in Quebec. Clin Perform Qual Health Care 1997;5:189-94.
5. Javitz HS, Ward MM and Martens L. Major costs associated with pressure sores. J Wound Care 1998;7:286-90.
6. Haalboom J. Rapport raad voor gezondheidszorg onderzoek: Decubitus. 1999.
7. Severens JL, Habraken JM, Duivenvoorden S and Frederiks CMA. The cost of ilness of pressure ulcers in the Netherlands. Adv skin & woundcare 2002;15:72-7.
8. Xakellis GC, Jr., Frantz RA, Lewis A and Harvey P. Cost-effectiveness of an intensive pressure ulcer prevention protocol in long-term care. Adv Wound Care 1998;11:22-9.
9. Ayello EA and Braden B. How and why to do pressure ulcer risk management. Adv skin & woundcare 2002;15:125-31.
10. Schoonhoven L, Haalboom JR, Bousema MT, *et al.* Prospective cohort study of routine use of risk assessment scales for prediction of pressure ulcers. BMJ 2002;325:797-807.
11. Defloor T. The risk of pressure sores: a conceptual scheme. J Clin Nurs 1999;8:206-16.
12. Bergstrom N, Braden BJ, Laguzza A and Holman V. The braden scale for predicting pressure sore risk. Nurs Res 1987;36:205-10.
13. Reddy PN and Cochran Gv. Evaluation of transducer performance for buttock-cushion interface pressure measurements. J Rehabil Res 1984;21:43-50.
14. Berjian RA, Douglass HOJ, Holyoke ED, Goodwin PM and Priore RL. Skin pressure measurements on various mattress surfaces in cancer patients. Am J Phys Med 1983;62:217-26.
15. Maklebust J, Mondoux L and Sieggreen M. Pressure relief characteristics of various support surfaces used in prevention and treatment of pressure ulcers. J ET Nurs 1986;13:85-9.
16. Thompson-Bishop JY and Mottola CM. Tissue interface pressure and estimated subcutaneous pressures of 11 different pressure-reducing support surfaces. Decubitus 1992;5:42-6, 8.
17. Clark M. Repositioning to prevent pressure sores-what is the evidence? Nurs Stand 1998;13:58-60, 2, 4.

18. Knox DM. Core body temperature, skin temperature, and interface pressure. Relationship to skin integrity in nursing home residents. Adv Wound Care 1999;12:246-52.

19. Meijer JH, Germs PH, Schneider H and Ribbe MW. Susceptibility to decubitus ulcer formation. Arch Phys Med Rehabil 1994;75:318-23.

20. van Marum RJ, Meijer JH, Bertelsmann FW and Ribbe MW. Impaired blood flow response following pressure load in diabetic patients with cardiac autonomic neuropathy. Arch Phys Med Rehabil 1997;78:1003-6.

21. Burman PM. Using pressure measurements to evaluate different technologies. Decubitus 1993;6:38-42.

22. Defloor T. Less frequent turning interval and yet less pressure ulcers. Tijdschr Gerontol Geriatr 2001;32:174-7.

23. Whittemore R. Pressure-reduction support surfaces: a review of the literature [see comments]. J Wound Ostomy Continence Nurs 1998;25:6-25.

24. Rondorf-Klym LM and Langemo D. Relationship between body weight, body position, support surface, and tissue interface pressure at the sacrum. Decubitus 1993;6:22-30.

25. Suarez CH and Reynolds A. Pressure reduction with a hospitalized population using a mattress overlay. Ostomy Wound Manage 1995;41:58-60, 2-3.

26. Sideranko S, Quinn A, Burns K and Froman RD. Effects of position and mattress overlay on sacral and heel pressures in a clinical population. Res Nurs Health 1992;15:245-51.

27. Clark M and Rowland LB. Preventing pressure sores: matching patient and mattress using interface pressure measurements. Decubitus 1989;2:34-9.

28. Jester J and Weaver V. A report of clinical investigation of various tissue support surfaces used for the prevention, early intervention and management of pressure ulcers. Ostomy Wound Manage 1990;26:39-45.

29. Counsell C, Seymour S, Guin P and Hudson A. Interface skin pressures on four pressure-relieving devices. J Enterostomal Ther 1990;17:150-3.

30. Weaver V and Jester J. A clinical tool: updated readings on tissue interface pressures. Ostomy Wound Manage 1994;40:34-6, 8, 40 passim.

31. Allen V, Ryan DW and Murray A. Measurements of interface pressure between body sites and the surfaces of four specialised air mattresses. Br J Clin Pract 1994;48:125-9.

32. Ryan DW, Allen V and Murray A. An investigation of interface pressures in low air loss beds. Int J Clin Pract 1997;51:296-8.

33. Holscher TG. A new low-cost anti-decubitus matress for home care; a comparison with other matresses. J Rehab Sc 1995;8:72-5.

34. Defloor T. The effect of position and mattress on interface pressure. Appl Nurs Res 2000;13:2-11.

35. Scott EM, Baker EA, Kelly PJ, Stoddard EJ and Leaper DJ. Measurement of interface pressures in the evaluation of operating theatre mattresses. J Wound Care 1999;8:437-41.

36. Hobson DA. Comparative effects of posture on pressure and shear at the body-seat interface. J Rehabil Res Dev 1992;29:21-31.

37. Swain I. Tissue viability. Assessment of support surfaces. Nurs Stand 1992;6:suppl 12-4.

38. Spijkerman DCM, Terburg M, Goossens RHM and Stijnen T. Effects of inflation pressure and posture on the body-seat interface pressure of spinal cord injured patients seated on an air-filled wheelchair cushion. J Rehabil Sci 1995;8:8-12.

39. Rithalia SV and Gonsalkorale M. Quantification of pressure relief using interface pressure and tissue perfusion in alternating pressure air mattresses. Arch Phys Med Rehabil 2000;81:1364-9.

40. Bader DL and Hawken MB. Pressure distribution under the ischium of normal subjects. J Biomed Eng 1986;8:353-7.

41. Clark M. Continuous interface pressure measurement using an electropneumatic sensor: the scp monitor. Care Sci Prac 1987;june:5-8.

42. Clark M. Comparison of the pressure redistribution atributes of a selection of bed mattresses used to prevent pressure sores. J Tissue Viability 1991;1:65-7.

43. Rithalia SV. Assessment of pressure relief characteristics in alternating pressure air cushions. Int J Rehabil Res 1997;20:205-8.

44. Rithalia SV and Gonsalkorale M. Assessment of alternating air mattresses using a time-based interface pressure threshold technique. J Rehabil Res Dev 1998;35:225-30.

45. Rithalia SV, Heath GH and Gonsalkorale M. Assessment of alternating-pressure air mattresses using a time-based pressure threshold technique and continuous measurements of transcutaneous gases. J Tissue Viability 2000;10:13-20.

46. Clark M, Bours G and DeFloor T. Summary report on the prevalence of pressure ulcers. EPUAP Review 2002;4:49-57.

47. Seiler WO, Allen S and Stahelin HB. Influence of the 30 degrees laterally inclined position and the 'super- soft' 3-piece mattress on skin oxygen tension on areas of maximum pressure--implications for pressure sore prevention. Gerontology 1986;32:158-66.

48. De Laat E. [Pressure sores in ICU patients--a literature review] Drukletsel bij IC-patienten--een literatuuronderzoek. Verpleegkunde 1997;12:4-14.

49. Fontaine R, Risley S and Castellino R. A quantitative analysis of pressure and shear in the effectiveness of support surfaces. J Wound Ostomy Continence Nurs 1998;25:233-9.

50.   Shelton F, Barnett R and Meyer E. Full-body interface pressure testing as a method for performance evaluation of clinical support surfaces. Appl Ergon 1998;29:491-7.

51.   Kernohan WG, Witherow A, Allen-Hamilton S and O'Hagan M. Evaluation of a range of hospital replacement mattresses. J Wound Care 1999;8:495-7.

52.   Bain DS, Scales JT and Nicholson GP. A new method of assessing the mechanical properties of patient support systems (PSS) using a phantom. A preliminary communication. Med Eng Phys 1999;21:293-301.

53.   Weststrate J, Goossens R, Heule F, Hop W and Bruining H. The reproducibility of interface pressure measurements in patients at risk of developing a pressure ulcer. (Submitted).

54.   McLeod A. Draft guidelines for the laboratory evaluation of pressure-redistributing support surfaces. EPUAP review 2002;4:8-11.

Chapter 7

Interface pressures in patients lying on different
mattresses during surgery

Jan T.M. Weststrate[1]

Wim C.J. Hop[2]

Freerk Heule[3]

Chris Mallios[4]

Tom Defloor[5]

Hajo A. Bruining[1]

1. Department of Intensive care, Erasmus MC, University Medical Center, Rotterdam
2. Department of Epidemiology and Biostatistics, Erasmus MC, University Medical Center, Rotterdam
3. Department of Dermatology & Venereology, Erasmus MC, University Medical Center, Rotterdam
4. Department of Anesthesiology, Erasmus MC, University Medical Center, Rotterdam
5. Department of Nursing Sciences, Ghent University, Ghent, Belgium

Abstract

Patients undergoing surgery are at an increased risk for developing a pressure ulcer. Decreased mobility and an inability to feel or react to pain are the main causes of a high incidence of pressure ulcers. Standard operating table mattresses do not effectively reduce the exerted pressure on the skin. Therefore, specific pressure-reducing mattresses are additionally required on top of the standard table. In this investigation the pressure-reducing capacity of three commonly used mattresses (Action® 1.5 cm visco-elastic dry polymer mattress, Waffle® air mattress and the ROHO® air mattress) was evaluated during surgery. Moreover, the variation of the measured Tissue Interface Pressure (TIP) during surgery was also evaluated. Sixty-three patients undergoing surgery participated in the study. Each mattress was tested in 20-22 patients.

The pressure-reducing capacity of a mattress was evaluated by measuring the TIP. Approximately every 15 minutes during surgery the TIP was measured for 5 minutes at the sacrum, the left ischial tuberosity and the right and left heel. The Peak mean TIP (PIP) out of each 5 minute measurement was the primary outcome variable.

Comparing the course of the mean PIP for each mattress during surgery, the Action® mattress produced the highest PIPs for each anatomical site compared with the PIPs measured on the Waffle® and the ROHO® mattresses ($p < 0.01$). A few patients on Waffle® and the ROHO® mattresses produced instable measurements, which were responsible for a variation coefficient of higher than 10%.

The results showed that the ROHO® and the Waffle® mattresses generated the lowest TIP compared with those measured on the Action® mattress. However, the TIP measured at the sacrum and the heels exceeded the generally accepted diastolic blood pressure level in a number of patients. Therefore, the tested mattresses would not adequately prevent pressure ulcer development in patients during surgical procedures.

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Introduction

Patients undergoing surgery under anesthesia have an increased risk of developing a pressure ulcer (PU) varying from 12% - 66% [1-4]. Recent research in the Netherlands reported a PU incidence of 21.2% in this group of patients [5]. Most investigators explained this high incidence because of the patient's decreased mobility during the operation [6], inability to react to the pain caused by the pressure exerted on the skin [5] and the inability of the standard operating table mattresses (OTMs) to reduce the pressure effectively on the patient's skin [2, 7-11]. This is further complicated by the fact that per-operatively induced PUs become visible after a period ranging from a few hours to 5 days [4, 12, 13]. This is frustrating for the physicians and the nurses in the clinical wards as they get the impression that their hard work in preventing PUs is ineffective. It is also a serious threat to the operating theatre personnel as they falsely assume that adequate measures were taken to prevent the development of PUs [8].

The development of a PU is the result of pressure and the length of time for which the pressure is applied to the skin of the patient [13]. High pressure for a short time can be as detrimental as low pressure for a prolonged period [14]. A complicating factor is that each patient has an individual threshold in relation to the damaging effect of the applied pressure [15, 16]. This difference in threshold is mainly caused by the variation in skin tolerance. Skin intolerance is a collective term for factors, when present, adversely affect the integrity of the skin under pressure, ergo a higher risk for PU development. Defloor [17] in his conceptual model, divided these factors into two groups. Group A consists of factors that influence the integrity of the skin in relation to the pressure. Group B consists of factors that influence the integrity of the skin in relation to the change in tissue oxygenation [17]. Examples of factors in group A are weight, age, dehydration status, protein and vitamin shortage, use of corticosteroids, and the presence of stress. Factors in group B are temperature, medication, protein, being a smoker, and illnesses that cause a decreased oxygen supply, a delay in reactive hyperemia and an increased vascular occlusion.

It is evident from this conceptual model that during each surgical procedure the tolerance of the skin is adversely affected by one or more of these factors. Some of these factors (medication, nutritional status, etc) are familiar to the nurses and the physicians before the operation. Others (influence of temperature, a decreased oxygen supply to the tissues and the use of inotropics) are unclear and probably occur or change during the surgery. In particular, an increased skin temperature at the interface of the patient's body and the mattress is one of the leading contributors in the development of PUs following surgery [18, 19]. This is caused by the use of warming devices that are placed under the patient in order to minimize heat loss during surgery. These devices enhance the cell metabolism, thereby increasing the need for cellular oxygen, nutrients and the rate of waste removal. Since this area is also under pressure, blood vessels are compressed and do not function adequately to supply oxygen and remove waste products [20]. In order to prevent this from occurring, reducing the exerted pressure by the OTM becomes critical.

The performance of OTMs was investigated in a limited number of studies [8, 21-25] in contrast to mattresses for hospitalized patients [26]. Stability, firmness, pressure reduction, and the ability to distribute pressure evenly without "bottoming out" are mentioned in the literature as important characteristics of OTMs [21]. The pressure-reducing capacity of an OTM may be evaluated by measuring the Tissue Interface Pressure (TIP) at the body sites (the sacrum, the buttocks and the heels) where PUs usually develop [27].

Campell [19] measured the TIP in patients on an OTM and investigated the difference in TIP values at the sacrum, the scapula, the thoracic spine and the heel in the pre-anesthesia-, post-anesthesia- and post-surgical phase. It was concluded that the sacral TIP values increased 35% in the post-surgical phase particularly compared with the pre-anesthesia phase in patients who had undergone surgery for longer than 2.5 hours. This indicated that TIP values may also vary in patients during surgery [22]. It is therefore questionable whether volunteers can act as a true model for actual patients [22, 28], although this was done in two studies [8, 23].

Most studies, in which the pressure distribution capacity of OTMs was investigated, a set of one or two measurements or a limited number (<=5) of measurements at short intervals were performed. If the TIP does change during surgery, it may be missed when an insufficient number of measurements are performed. Therefore, we designed a study in which the interface pressure was measured every 15 minutes in patients who were placed on three different OTMs during surgery. The two questions that were addressed in this study were:

1. How does the TIP value vary in patients placed on one of the three special OTMs during surgery?
2. What are the TIP values that are generated when patients were placed on one of the three special OTMs during surgery?

Materials and Methods

Routinely, an Action® 1.5 cm visco-elastic dry polymer mattress (Action® products, Inc, Hagerstown, Md, USA) on top of the standard operating theatre table is used during surgery. The two other types of mattresses that were used for this study were the Waffle® mattress (EHOB, Inc, Indianapolis, IN, USA) and the ROHO® mattress (The ROHO group, Belleville, IL, USA). The Waffle® is a static air mattress, which has a number of punched holes that allows the patient to sink into the mattress. The ROHO® mattress consists of flexible air cells that are pneumatically connected with each other. Each cell acts as a small piston in creating the same internal pressure for each cell. The mattress is covered with a lose fitting semi-permeable cover. Both mattresses have an air tube that can be connected to a pressure gauge and an air pump in order to add or release air, depending on the air pressure inside the mattress or the position of the patient.

The TIP was measured with the Talley Pressure Monitor III (TPM III) at the sacrum, the left buttock and both the heels during surgery in patients on each type of the mattress. The TIP at the right buttock was not measured as this interfered with the work of the surgeon. The TPM III consists of three basic components; a sensor array, a pneumatic monitor and a calibration jig. The general working of the TPM was described in detail by Hobson [29].

The sensor array (Appendix 3) used for measuring the TIP at the sacrum and the left buttock had a 3x4 matrix configuration and single sensors were used for the heels (see Appendix 3). The sensor array consists of twelve 20 mm diameter cells mounted on 28 mm centers. This matrix provides an effective measurement area of approximately 8,000 mm^2. A small-diameter flexible tube that plugs into the monitor manifold switch airs each cell in the matrix. The thickness of the deflated matrix is approximately 2-3 mm. The stated pressure ranges from 0 to 32.8 kPa, with a resolution of 0.13 kPa.

A total of 63 patients participated in the study. The allocation of the mattress was based on convenience. The operating theatres that handled head and neck surgery mostly used the ROHO® or the Waffle® mattress. The Action® mattress was used for all types of surgery (head and neck, abdominal & thoracic and extremities). Care was taken that the TIP was measured in at least 20 patients on each type of the three mattresses.

The preparation of the operating theatre table was as follows. A flannel sheet was put over the table. A heating blanket was positioned on top of this followed by one of the three mattresses under investigation. The mattress under investigation was covered with a sheet and a towel in the sacral area on which the patient was positioned.

The positioning of the sensor arrays was carried out after the patient was correctly positioned on the mattress on the operating table and before anesthesia. The sensor array at the sacrum was positioned first. The patient was asked to lift the buttocks and the sensor array was manually positioned by the investigator over the sacral area by positioning the sacrum as close as possible to the center of the sensor array. The second array was placed under the left buttock. To do this the patient was asked to pull up the left knee as much as possible towards the chin and to turn approximately 30^0 to the left side, without moving the right buttock over the mattress. The investigator positioned the sensor array manually over the buttock such that the ischial tuberosity was felt as close as possible to the center of the matrix. Finally, two single sensors were placed under the left and the right heel at the point of the maximum pressure located at the center of the sensor.

The two sensor arrays and two single cells were connected to the TPM III and the first measurement was carried out after the patient was completely anesthetized and paralyzed. The TPM III starts to measure the TIP at the sacrum followed by the left buttock and then the heels. Then the cycle starts again. Each cycle is called a frame. Each *measurement* consisted of 8-9 frames and took about 5 minutes, measured every 14 to18 minutes.

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The performance of the three mattresses in this study was analysed on the basis of the calculated Peak mean of TIP (PIP) for each sensor array (sacrum, buttock) or single sensor (heel). As the pressure between the patient and the operating table mattress has a continuous character, probably the PIP has the highest impact on the patient's skin during surgery. The highest mean TIP of each sensor in the array over the 8-9 measured frames was considered to be the PIP. For the single sensors over the heels the mean pressure of one cell was considered to be the PIP. All readings of a particular sensor were excluded if one or more readings were 32.8 kPa. This is the highest readout of the TPM III and therefore the accuracy is uncertain. In most instances this was caused by a crease in the sensor array. Besides calculating the PIP for each anatomical site, it was also calculated for the pelvic area. This was defined as the highest PIP for each measurement from the two arrays positioned under the sacrum and the left buttock. The local institutional review board approved the study.

Statistical analyses
Patient demographic data between the three types of mattresses was compared using the chi-square test (sex) or one way ANOVA. The Pearson correlation test was used for analysing the relation of the PIP, versus body mass, Body Mass Index (BMI) and the age of the patients on the three types of mattresses. Comparison of the PIPs between the three types of mattresses during the observation period for each anatomical site was done using repeated measurements ANOVA. Comparison of the PIPs measured at the sacrum, the buttocks and both the heels for each type of mattress was done using the paired sample T-test. The stability of the measurements was evaluated by calculating the coefficient of variation (VC) of the first eight measurements in each patient. The level of significance was set at $p < 0.05$ for all the statistical tests.

Results

Sixty-three patients participated in the study (35 males, 28 females). The mean age for both males and females was 45 years (SD respectively: 17, 25). There was no significant difference between the male/female distribution over the three mattress groups ($p = 0.82$). A summary of the characteristics of the patients who underwent surgery on each type of the three mattresses is shown in Table I. The time the patients spent on the Action® mattress was significantly less compared with the time the patients spent on the Waffle® mattress ($p < 0.05$). The length of the average measurement interval was also significantly less in the patients on the Action® mattress compared with that on the Waffle® mattress ($p < 0.05$). There was no correlation between the mean PIP measured at each anatomical site and the body mass, BMI and age within the three types of mattresses.

Table I. **An overview of characteristics, mean PIP values and type of surgery in patients who underwent surgery on the three types of operating table mattresses (SD)**

Characteristics	ROHO®	Action®	Waffle®
Number of patients	20	21	22
Male/female	10/10	12/9	13/9
Age (yrs)	53 (± 17)	38 (± 24)	44 (± 18)
Mass (kg)	76 (± 16)	74 (± 19)	76 (± 19)
Height (m)	1.72 (± 0.1)	1.71 (±0.1)	1.75 (± 0.1)
BMI	25 (± 4.2)	25 (± 4.0)	25 (± 4.1)
Number of measurements	8 (± 2.5)	7 (± 1.9)	8 (± 3.0)
Number of cycles per measurement	9.85 (± 3.7)	9.12 (± 1.7)	9.48 (± 2.3)
Measurement interval (min)	16 (± 4.4)	14 (± 3.2)	18 (± 4.6)
Average time on the mattress (min)	137 (± 73)	90 (± 32)	156 (± 94)
Mean PIP values			
Mean PIP sacrum (kPa)	10.3 (± 2.9)	15.5 (± 4.2)	9.1 (± 4.9)
Mean PIP buttock (kPa)	6.3 (± 1.5)	11.7 (± 4.6)	5.8 (± 2.1)
Mean PIP pelvic area (kPa)	10.4 (± 2.9)	16.6 (± 3.4)	9.0 (± 4.7)
Mean PIP left heel (kPa)	10.2 (± 3.5)	15.8 (± 4.4)	9.9 (± 6.2)
Mean PIP right heel (kPa)	11.1 (± 4.8)	16.9 (± 4.1)	10.3 (± 4.9)
Type of surgery			
Head and neck (%)	95	38	73
Abdominal / thoracic (%)	5	33	14
Extremities (%)	0	29	13

Since the number of measurements depended on the duration of the surgery, it varied for each patient. Most surgery was completed within 2 hours, thus allowing 8 measurements. Therefore, the first 8 measurements were used for analysing the difference between the mean PIPs at the 4 sites in each patient for the three types of mattresses. An overview of the course of the mean PIPs for each site for each mattress is shown in Figure 1. There was a significant difference between the mean PIPs measured on the ROHO® and the Waffle® mattresses compared with the mean PIPs measured on the Action® mattress for all measurements and for each anatomical site ($p < 0.01$). There was no significant

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difference (p > 0.05) between the mean PIPs on the ROHO® mattress and the Waffle® mattress for all sites. For an impression of the variation in the gathered data, the individual course of the first eight measurements at the sacrum of all patients is shown in Figure 2. It is evident that particularly the ROHO® and the Waffle® mattresses showed some variation in the PIPs. The mean VCs of the ROHO® and the Waffle® mattresses were higher than 10% although the mean median VC was below 10 % indicating that the measurements were instable in only a few patients. The VCs of the PIPs at the buttock were all below 10 % and the VCs of the PIPs at both the heels were generally speaking higher than 10%.
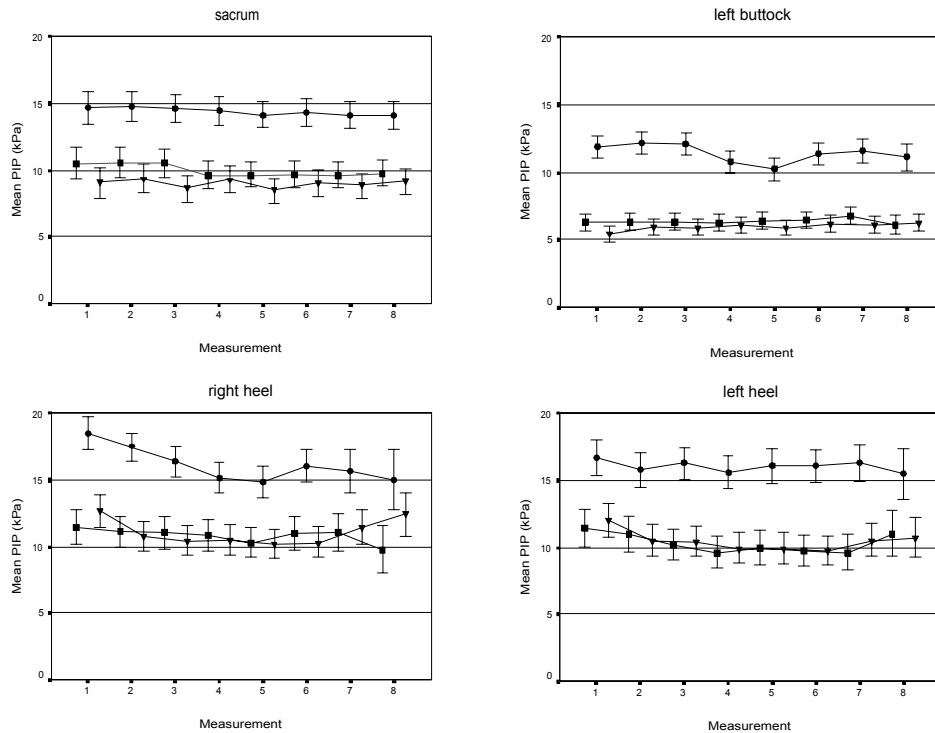

Figure 1. An overview of the different patterns of the mean PIP for each type of mattress for each site for the first 8 measurements (■: ROHO® mattress, ●: Action® mattress, ▼ : Waffle® mattress)

The highest PIP for each patient in the pelvic area was mostly observed at the sacrum (ROHO®: 93%; Action®: 75%; Waffle® 80%). The PIP measured at the sacrum was for all types of mattresses was significantly higher compared with the PIP measured at the buttocks (p < 0.001). The PIP measured at the left and the right heel was significantly the different for the ROHO® and the Action® mattresses (p = 0.018, p = 0.014 resp). The PIPs measured at the heels on the Waffle® mattress was not significantly different (p =

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0.265) when compared with those on the ROHO® and Action® mattresses. Comparing the PIPs at the heels with those measured at the sacrum and the buttocks showed that all PIPs measured at the buttocks were significantly lower than those measured at both the heels (p < 0.001). The PIPs at the left heel on the Waffle® mattress and at both the heels on the Action® mattress were not significantly higher than those measured at the sacrum. This was not the case on the ROHO® mattress compared with the sacrum on the other mattresses.
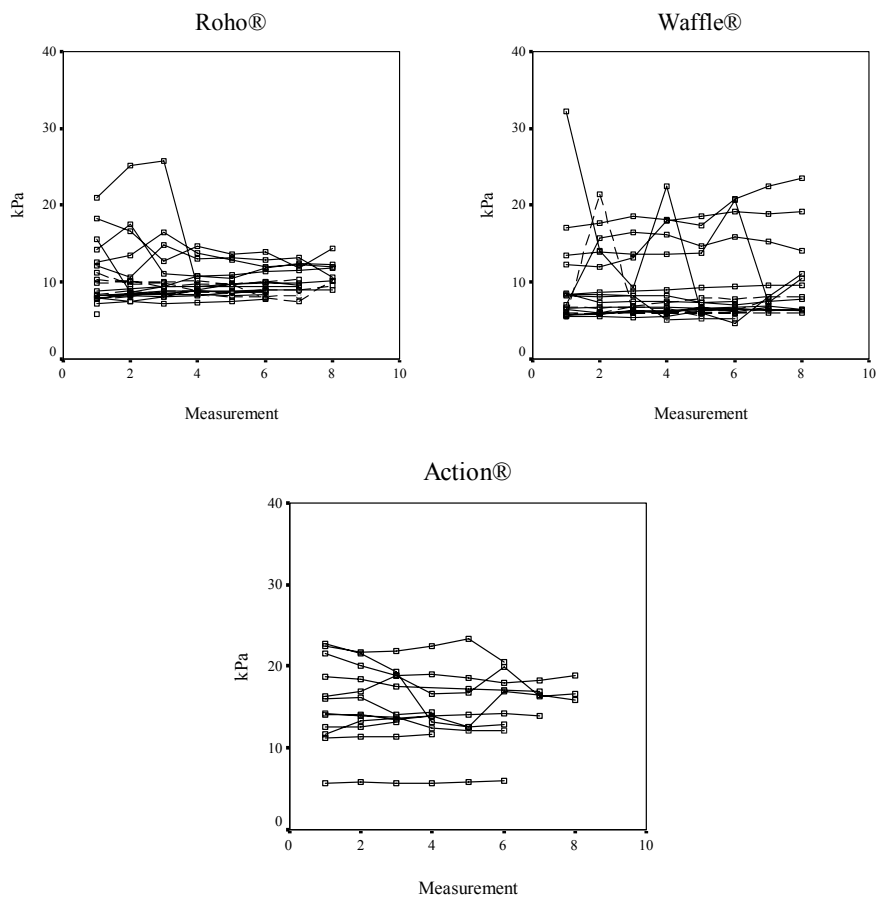

Figure 2. Individual PIP measurements (maximum of eight) at the sacrum of all patients on the three mattresses under investigation

Discussion

The results of this study clearly showed that patients who were operated on the ROHO® and the Waffle® mattresses generated lower PIPs compared with those in patients who were operated on an Action® mattress. Defloor and de Schuijmer [8] investigated the pressure-reducing capacity of 5 OTMs (standard-, polyether -, polyurethane-, foam- and gel (visco-elastic dry polymer overlay) mattresses) in patients lying in various positions. TIP was measured using the Ergocheck® which is a pressure mapping system consisting of 684 pneumatic sensors. They also observed that a visco-elastic dry polymer mattress generated the highest TIP in the supine position (5.7 kPa) compared with the other 4 OTMs. We measured a maximum PIP of 16.6 kPa in the pelvic area on the Action® mattress. This difference can be explained by the fact that different measuring devices were used in the study by Defloor and de Schuijmer and this study. Moreover, in that study [8] measurements were done in healthy volunteers, whereas we measured in patients. Nevertheless, this suggested that a visco-elastic polymer mattress increased the risk for developing PUs when compared with the ROHO® and the Waffle® mattresses. In contrast, Nixon *et al.* [24] noted that using a visco-elastic dry polymer mattress during surgery decreased the development of PUs by 50% when compared with a standard OTM. Hoshowsky and Schramm [21] investigated the effect of three OTMs (standard 2 inch mattress, 2 inch foam and gel mattress and a visco- elastic dry polymer mattress overlay) in preventing the development of PUs. Their results also indicated that the visco-elastic dry polymer mattress overlay in combination with the standard mattress prevented the development of PUs more effectively than the standard mattress alone.

The results of those two studies [21, 24] clearly indicated that a standard OTM alone during surgery is ineffective in preventing the development of PUs at the sacrum or the buttocks. Compared with the results of this study, it also indicated that the additional use of a Waffle ® or a ROHO® mattress reduced the TIP even more and thus reduced the risk of developing PUs even further.

The fact that the TIP was measured every 14 to 18 minutes during surgery made it possible to evaluate the variation in the measurements. It is evident from Figure 3 that some patients on the ROHO® and the Waffle® mattresses showed a significant variation in PIPs at the sacrum. This was mainly because of manipulations by the surgical team during surgery. This also indicated that a single measurement in each patient is not sufficient to obtain a correct impression of the pressure-reducing capacity of an OTM. The high variability at the heels can be explained by the fact that only one sensor was used to measure the TIP. Slight movements of the legs during the time of measurement and a slight change in the position between the measurements may cause this variation.

On the average about 80 % of the highest PIPs in the pelvic area were measured at the sacrum. The range of the mean PIPs at the sacrum on the ROHO® and the Waffle® mattresses varied between 8.7 kPa and 10.7 kPa for a period of at least 2 hours. In most cases this value was above the diastolic blood pressure indicating a higher risk of decreased

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tissue perfusion [30]. The PIPs measured at the sacrum and the buttocks were higher than those reported in other studies [8, 23, 25]. Blaylock and Gardner [25] evaluated two different foam mattresses and measured the TIP at the scapula, the sacrum and the heels in 20 patients. At the sacrum they measured mean pressures of 5.1 kPa. Other investigators used other measuring devices for measuring the TIP that enable measurements of the interface pressure over a specific area instead of a specific site. Scott et al. [23] used the Force Sensing Array (FSA) (Vista Medical®) to measure the TIP in volunteers in two positions on four foam mattresses. In the supine position, the mean TIP at the sacrum varied between 8.3 kPa and 10.0 kPa. Defloor and de Schuijmer [8] used the Ergocheck® to measure the TIP generated in the supine position in healthy volunteers on 5 different mattresses. They observed mean TIP values of between 4 kPa and 6.7 kPa. Our mean PIP values at the sacrum on the Waffle® and the ROHO® mattresses were more in concordance with the values reported by Scott et al. [23]. Two factors that varied between the studies could have influenced the TIP. We measured the interface pressure in patients during surgery compared with measurements in healthy volunteers in the other two studies [8, 23]. Campbell [19] reported that in general the interface pressure increased by 27.5 % after anesthesia. During surgery this increased to 35 % compared with pre-anesthesia TIP values. An earlier study by Weststrate et al. [31] also reported a significant difference between the TIP measured in patients compared with healthy volunteers.

Another factor that may influence the TIP is the number of layers of material between the sensor and the skin and the sensor and the mattress. In relation to the number of layers between the sensor and the mattress, often specific techniques for draping the operating tables are used whereby layers of different materials (heating blankets, flannel sheets, incontinence pads, towels) are placed between the sensor and the mattress. Campbell [19] reported that each layer of cloth or material on top of the OTM decreased the pressure-reducing ability of the mattress and suggested to limit the number of layers between the patient and the special OTM to a maximum of four. In our investigation, a maximum of three layers was used as the heating blanket was positioned under the OTM. In relation to the number of layers between the skin and the sensor, the sensors in our study were positioned directly on the skin of the patient.

The highest pressures in our study were measured on the heels of the patients. Although the use of the ROHO® and the Waffle® mattresses did reduce the mean PIP, the actual mean still exceeded the diastolic blood pressure in most cases. Other investigators [32, 33] measured interface pressure on the heel and reported results that are comparable with the results of this study. Therefore, a pressure-reducing OTM to lower the pressure on the heel is not sufficient. If possible, pillows supporting the lower leg and lifting the heel from the mattress should be used to ensure that the heel is free of pressure.

In conclusion, the ROHO® and the Waffle® mattresses generated the lowest interface pressures at the sacrum, the buttocks and the heels during surgery compared with the Action® mattress. The mean PIP measured specifically at the sacrum and the heels on the ROHO® and Waffle® mattresses often exceeded the average diastolic blood pressure.

Therefore, the tested mattresses would not prevent pressure ulcer development in patients during surgical procedures.

Literature

1.   Kemp MG and Krouskop TA. Pressure ulcers: reducing incidence and severity by managing pressure. J Gerontol Nurs 1994;20:27-34, 52.
2.   Waterlow J. Operating table. The root cause of many pressure sores? Br J Theatre Nurs 1996;6:19-21.
3.   Aronovitch SA, Wilber M, Slezak S, Martin T and Utter D. A comparative study of an alternating air mattress for the prevention of pressure ulcers in surgical patients. Ostomy Wound Manage 1999;45:34-40, 2-4.
4.   Vermillion C. Operating room acquired pressure ulcers. Decubitus 1990;3:26-30.
5.   Schoonhoven L, Defloor T and Grypdonck MH. Incidence of pressure ulcers due to surgery. J Clin Nurs 2002;11:479-87.
6.   Bader DL, Pressure Sores- Clinical Practice and Scientific approach., ed. DL Bader. 1990, Basingstoke U. K.: Macmillan Press Scientific & Medical.
7.   Scott EM. Hospital-acquired pressure sores in surgical patients. J Wound Care 1998;7:76-9.
8.   Defloor T and de Schuijmer JD. Preventing pressure ulcers: an evaluation of four operating-table mattresses. Appl Nurs Res 2000;13:134-41.
9.   Schoonhoven L, Incidentie van decubitus op de operatietafel (incidence of pressure ulcers on the operating table) 1998, Utrecht, Academisch Ziekenhuis Utrecht: Verplegingswetenschap.
10.   Versluysen M. How elderly patients with femoral fracture develop pressure sores in hospital. Br Med J (Clin Res Ed) 1986;292:1311-3.
11.   Schultz A, Bien M, Dumond K, Brown K and Myers A. Etiology and incidence of pressure ulcers in surgical patients. AoRN J 1999;70:434, 7-40, 43-9.
12.   Daniel RK, Priest DL and Wheatley DC. Etiologic factors in pressure sores: an experimental model. Arch Phys Med Rehabil 1981;62:492-8.
13.   Kosiak M. Etiology of decubitus ulcers. Arch Phys Med Rehabil 1961;42:19-29.
14.   Reswick JB and Rogers J, Experience at Rancho los Amigos Hospital with devices and techniques to prevent pressure sores, in Bedsore Biomechanics, RM Kenidi, Cowden JM and Scales JT, Editors. 1976, Baltimore. University park Press p. 301-10.
15.   Meijer JH, Germs PH, Schneider H and Ribbe MW. Susceptibility to decubitus ulcer formation. Arch Phys Med Rehabil 1994;75:318-23.
16.   van Marum RJ, Meijer JH and Ribbe MW. The relationship between pressure ulcers and skin blood flow response after a local cold provocation. Arch Phys Med Rehabil 2002;83:40-3.
17.   Defloor T. The risk of pressure sores: a conceptual scheme. J Clin Nurs 1999;8:206-16.
18.   Grous CA, Reilly NJ and Gift AG. Skin integrity in patients undergoing prolonged operations. J Wound Ostomy Continence Nurs 1997;24:86-91.

19.   Campbell K. Pressure point measures in the operating room. J Enterostomal Ther 1989;16:119-24.

20.   Kokate JY, Leland KJ, Held AM, *et al.* Temperature-modulated pressure ulcers: a porcine model. Arch Phys Med Rehabil 1995;76:666-73.

21.   Hoshowsky VM and Schramm CA. Intraoperative pressure sore prevention: an analysis of bedding materials. Res Nurs Health 1994;17:333-9.

22.   Neander KD and Birkenfeld R. Decubitus prophylaxis in the operating theatre? J Tissue Viability 1991;1:71-3.

23.   Scott EM, Baker EA, Kelly PJ, Stoddard EJ and Leaper DJ. Measurement of interface pressures in the evaluation of operating theatre mattresses. J Wound Care 1999;8:437-41.

24.   Nixon J, McElvenny D, Mason S, Brown J and Bond S. A sequential randomised controlled trial comparing a dry visco-elastic polymer pad and standard operating table mattress in the prevention of post-operative pressure sores. Int J Nurs Stud 1998;35:193-203.

25.   Blaylock B and Gardner C. Measuring tissue interface pressures of two support surfaces used in the operating room. Ostomy Wound Manage 1994;40:42-4, 6, 8.

26.   Whittemore R. Pressure-reduction support surfaces: a review of the literature. J Wound Ostomy Continence Nurs 1998;25:6-25.

27.   Clark M, Bours G and Defloor T. Summary report on the prevalence of pressure ulcers. EPUAP Rev. 2002;4:49-57.

28.   Abruzzese RS. Guidelines for evaluating decubitus ulcer research. Decubitus 1989;2:28-32.

29.   Hobson DA. Comparative effects of posture on pressure and shear at the body-seat interface. J Rehabil Res Dev 1992;29:21-31.

30.   Defloor T. Tissue tolerance and the risk of decubitus. Verpleegkunde 1996;11:131-42.

31.   Weststrate JTM, Hop WCJ, Heule F, Defloor T and Bruining HA. A comparison of interface pressure measurements between patients and volunteers. submitted.

32.   Allen V, Ryan DW and Murray A. Measurements of interface pressure between body sites and the surfaces of four specialised air mattresses. Br J Clin Pract 1994;48:125-9.

33.   Ryan DW, Allen V and Murray A. An investigation of interface pressures in low air loss beds. Int J Clin Pract 1997;51:296-8.

Chapter 8

General discussion

8.1   Introduction

Two critical aspects of the concept of pressure ulcer development as described by Defloor are investigated in this thesis [1]. In the first three studies (chapters 2, 3 and 4) the value of using a pressure ulcer (PU) risk assessment instrument which quantified the tissue tolerance (intrinsic factors) with regard to the applied pressure in patients was investigated. The manner in which TIP measurements may be relevant for clinical practice was investigated in the studies described in chapters 5 ,6 and 7. In this discussion the relevance of the studies on both aspects is dealt with separately. Suggestions on further research are also mentioned.

Traditionally PU development is considered as a sign of "bad" nursing [2]. This implies that nurses are to blame when a PU develops. This also insinuates that nurses are the primary group of healthcare workers who should do something about it and that other healthcare staff have no or less significant responsibility in this matter. It is the opinion of the author that nowadays PU development is a multidisciplinary issue and its prevention can only be successful if it is viewed as a multidisciplinary problem. This implies that each professional who is involved in the patient rehabilitation process is responsible for preventing PU development through effective collaboration with other involved disciplines.

8.2   The value of Pressure Ulcer risk assessments

It is evident from chapters 2 and 3 that PU development in intensive care patients is a significant problem in the local and European context. In a recent national study in intensive care patients, Bours *et al* (2002) reported almost the same prevalence (28.7%) as we did [3]. This indicates that PU development is an actual problem in patients in the ICU. In order to establish a strategy for preventing the development of PU, we investigated whether the Waterlow PU risk assessment scale could be used daily for identifying patients who had a particularly high risk of developing a PU. The investigation was not carried out with the aim to test its predictive accuracy as this can only be done if no preventive interventions (intentional or unintentional) are carried out by the medical and nursing staff during the time the patients are in the ICU [4]. As this is unethical and does not enhance the patient's rehabilitation, testing its predictive accuracy by diagnostic testing is not the method of choice for evaluating a PU risk assessment instrument [5]. Therefore, it is debatable whether a specific pattern can be predicted for each individual patient. In relation to the prediction of PU development it resembles more to the application of the chaos theory applied in the meteorology meaning that only predicting PU development may be reasonably accurate in the very near future [6].

In analyzing the collected data, we incorporated the type and the frequency of preventive measures that were implemented. We also wanted to use the model in combination with the clinical experience [4, 5]. This indicates that the nurses sometimes took different decisions based on the same PU risk assessment score whether or not a special

pressure-reducing mattress was used. The results of the studies presented in chapter 4 showed that the developed prediction model efficiently provided the ICU nurses with this information on their patients. This model has been successfully used by the nurses at the local ICU for the last 10 years to prevent PU development because the percentage of PU days in the unit decreased to below 10% [7].

In general, the daily use of PU risk assessment scales is encouraged for preventing the development of PU [8, 9]. The collected data on the quality indicators at the fifth yearly national PU prevalence survey showed that half of the quality indicators were present in an average unit at a university hospital. Zooming in on the quality indicator for registering high risk patients with respect to PU development, the nursing notes showed this was done in 20% of the units in 2002 [10] and in 32% of the units in 2003. Unfortunately, whether the high risk patients were identified in the medical notes was not investigated. Therefore, the advice in the guidelines of the European Pressure Ulcer Advisory Panel (EPUAP) and the Dutch institute of healthcare improvement (CBO) is neglected. The question is whether this explains why there was no decrease in the PU prevalence during the national PU prevalence survey in the Netherlands over the period 1998- 2003 [11] . Clearly, it is not enough for physicians and nurses to be informed and have access to the clinical guidelines to change their clinical practice. This raises the question why physicians and nurses are slow in implementing the *evidence-based guidelines* on the prevention of PU.

Hunt [12] (1981) suggested five reasons why nurses neglect working according to the evidence-based guidelines. These were: nurses were unaware of the research findings, they did not understand them, they did not believe them, they did not know how to apply them and they were not allowed to use them. The item `they did not believe them` is still actual as was shown in a recent study that 50.4% of the participating nurses (n =389) did not value working according to the evidence-based principles [13]. This is certainly an issue that needs to be addressed by hospital administrators, nurse leaders, unit managers and last but not least in nursing education programs. Concerning the other four items, as more than 20 years have passed, it is expected that nurses have increased their professionalism and are aware of these items. Therefore, other issues in which physicians may play an important role as well explains for their reluctance. Five possible items are analyzed below.

The first possible reason why nurses fail to use PU risk assessment instruments to identify patients with an increased risk is that they perceive PU development as a chain event. As patients these days visit more than one department during their stay at a hospital, the nurses possibly perceive PU development as caused not during the patient's stay in their unit and therefore feel less responsible. Blaming other units does not stimulate taking responsibility. Such thinking places the healthcare staff central instead of the patient who needs good quality care. Adequate preventive measures can be taken in time when PU risk assessments are carried out daily in all patients, regardless of the unit or the department they are cared by. Not performing any PU risk assessment in such a situation also obscures the evidence of an increased risk. The studies described in chapter 4 clearly show that the daily PU risk assessment was very important for the patient.

Secondly, not every patient with an increased risk develops a PU. This decreases the confidence of the nurse or the physician in the instrument as an effective warning device. Recent developments in evaluating the predictive value of risk assessment instruments has sometimes been misinterpreted by the physicians and nurses in adopting the view that risk assessment instruments are of no use because of their poor predictive value [14]. What has actually been shown is that the current PU risk assessment instruments are not accurate in predicting the inevitable development of a PU. It is debatable whether this was the initial purpose when these instruments were developed [5]. The value of using a PU risk assessment instrument lies more in the realm of being a warning instrument and the outcome must be combined with clinical assessment by experienced nurses. In doing so a more realistic PU risk assessment emerges [4]. In stressing the importance of clinical assessment, the British Department of Health published a document which underlined that the use of a PU risk assessment instrument should never replace nurse's clinical judgment [15].

Using a PU risk assessment instrument may be compared with a traffic light. Every time a red signal is ignored does not mean that an accident is imminent, but that the more often you ignore it the higher is the risk of an accident. Therefore, daily assessment of patient's for their risk to develop a PU makes the nurse aware of the risk a patient has for developing a PU and to take the appropriate preventive measures.

Thirdly, a PU is not seen as an immediate life-threatening condition. Especially, in an ICU and an emergency room there are often a large number of life-threatening conditions that need immediate attention compared with PU preventive measures. On such occasions filling in a PU risk assessment instrument has no priority. Not only life-threatening conditions have this impact, but also the involvement of the nurse and the physician in all sorts of organizational aspects such as meetings and management tasks pushes the patient from the center of attention. Braden and Bergstrom [16] speak of a sensory overload which reshuffles the priority list regularly as besides the direct patient care other demands also need to be addressed. This results in less time and the quality of total patient care is jeopardized.

Addressing the mortality risk when having a PU, the Dutch Central Bureau of Statistics (CBS) mentioned that in 1999, 445 people died of a PU as a primary cause of death and 1396 patients died of PU as a secondary cause of death [17]. In the long-term, a PU maybe more life-threatening than one would like to think. Certain patients who often have an increased mortality risk in an ICU will benefit from an aggressive PU prevention strategy.

Fourthly, PU development is currently not viewed as an accident or a near accident and therefore, is not reportable to the hospital administration. It is also not viewed as a serious complication for the patient's rehabilitation process which is often discussed within a ward-based team. The absence of these administrative rules decreases the importance of the issue both from a quality and socio-economical point of view. Besides the patient's discomfort, the fact that yearly each hospital spends substantial amounts of

money on buying or renting special pressure-reducing mattresses should provide enough rationale for the hospital administrators to take a closer look at this issue. A hospital-wide blame-free obligation to register each PU equal to or higher than stage 2 may stimulate a process in which the nurses and the physicians view PU development as an unacceptable complication, which has a negative influence on their budgets. Ward-based analysis of the collected data may help them to find out that in most instances the PU development could have been prevented if adequate measures were taken. Regular surveillance by independent specially trained nurses should help to keep everyone alert on this issue. This issue is particularly taken seriously by the Dutch Ministry of Health which placed PU development at the top of the yearly reported list of critical care indicators [18].

A fifth reason may be that as PU development has an incubation time between a couple of hours and 5 days [19], the ICU nurses and the physicians are not always confronted with the effect of their PU prevention strategy because often the patients have already been transferred to another unit. On the one hand, this may provide a false impression that PUs do not develop in patients in ICU and therefore, the nurses do not need to use a risk assessment instrument. On the other hand, it can give the nurses in the other unit the impression that whatever they do, patients from the ICU always develop a PU and a PU risk assessment instrument is therefore not useful. Regular communication in this respect between the physicians and the nurses in various units may bring such issues to the surface. Collaboration between units shows the reality and stimulates taking responsibility for the issue involved. The same applies to the healthcare personnel in other units in which patients stay for a short time (e.g. emergency department, operating theatre, radiology and nuclear medicine department).

In conclusion the following recommendations can be made for using PU risk assessment instruments.

> Nurses and physicians in general and specifically in the ICU should follow the national guidelines on the prevention and the treatment of PU. This should be recorded in the medical and the nursing notes [10].

> Daily reporting of the risk a patient has to develop a PU and the condition of the skin at PU risk areas in the nursing notes.

> Weekly measurement of the PU prevalence in a randomized fixed number of patients in each ward at the hospital. The outcome of such an exercise keeps the nurses, the physicians and the hospital administrators alert and can act as an objective measure of the quality of the supplied care.

> Each PU development equal to or higher than stage 2 should be recorded blame-free in a central hospital register. Regular surveillance of the ward by specially trained independent nurses must ensure compliance. Monthly multidisciplinary reflective discussions between the physicians and the nurses of various units with cases of patients with PU may help to prevent future occurrences. Benchmarking with other similar types of units with

identical patient mix will also help to identify the best practice models in this area of care [15].

Future research must be directed at the influence of using a risk assessment instrument on the suggested interventions with and without the clinical assessment by the nurse. This would clarify the role the nurse's expertise and experience play in the process of risk assessment and which factors (education, experience, knowledge, attitude, etc) influence its accuracy [20].

8.3   The value of interface pressure measurements

In the studies in the second part of this thesis (chapters 5, 6 and 7) the value of Tissue Interface Pressure (TIP) measurements performed in patients admitted to the hospital were investigated. When the aim of measuring the TIP is to predict PU development on the basis of the intensity of the pressure, the results of the study in chapter 5 showed that this was not a reliable parameter. The Intra-Class Correlation Coefficient (ICC) had low values, indicating a large variation between the two measurements. It is suspected that the primary cause is that patients did not need to be in the same position for each measurement. To ask this from patients would not reflect the reality of daily life. Patients move regularly in bed during the day and also change their positions according to their experienced comfort during the course of their hospital stay. It was calculated for this patient group that it would take nine measurements on nine consecutive days in order to get a reliable impression on the average TIP for a particular patient. With the purpose of the study in mind this is not practical as most PU develop within a time span of a couple of hours to five days [19] depending on the intensity of the pressure and the tissue tolerance of the patient to withstand the pressure.

Selecting more specific patients with an increased risk for developing a PU who do not move in bed would be better for investigating the reliability of this parameter. Therefore, patients in the ICU who are unable to move or are heavily sedated or comatose are probably better for answering this question.

An alternative method to analyze the reliability of the TIP for a more mobile patient group is to use a different method for measuring the TIP. In chapter 5, the method used for measuring the TIP with a sensor array in patients on two occasions with at least an interval of 24 hours is described. The measurement itself took about 10 minutes after which the sensor array was removed. It is unknown how often the patient changed position between the two measurements. This can be of great importance as relief of pressure allows oxygen deprived tissues to get rid of their metabolic waste products and be re-oxygenated. A new measuring device known as the Forced Sensor Array (FSA) from Vista Medical® provides the opportunity to measure the TIP with a fixed interval and a remote control. Hereby the quality and the quantity of the patient's movement can be detected and

incorporated into the analysis of the data. The FSA sensor array has 256 sensors distributed over an area of 6466 cm^2. This makes it possible to register both the alterations in position more accurately and the time patients spend in a specific position. This was investigated earlier for the reproducibility of the TIP in wheelchair patients [21].

As the TIP in patients is an unreliable parameter to predict their risk of developing a PU, the focus of interest shifted towards assessing the pressure-distributing capacity of our hospital mattress with the TIP (chapters 6 and 7). The reason why we investigated the possible difference in the TIP between patients and healthy volunteers was inspired by the fact that most of the research in this area was carried out in healthy volunteers. This amazed us because most of mattresses in hospitals are used by patients with a variety of physical disabilities. The assumption that the mattress has the same effect on patients as compared with healthy volunteers was contradicted by the results of the studies that were pursued in chapter 6.

Besides the TIP, there are a number of other selection criteria, which should be considered before purchasing a mattress. Krouskop and Van Rijswijk [22] mentioned three main purposes of a support surface (provide comfort, facilitate posturing and redistribute pressure) and developed a method to assess the nine related criteria; 1: skin moisture control, 2: skin temperature control, 3: redistribution of pressure, 4: patient / product friction, 5: life expectancy, 6: inflammability, 7: safety, 8: infection control and 9: product service requirements. These criteria may be divided into three categories namely product efficacy, product safety and product cost / benefit ratio. Manufacturers design their mattresses using these criteria to make the performance of the product transparent for health care professionals and administrators in their decision making process. The issues related to product efficacy should be assessed by nurses and physicians; the product safety related issues by technicians and the product cost / benefit by hospital administrators [23].

Currently, there is no legal requirement for the manufacturers of mattresses to provide proof that the marketed mattresses are beneficial in preventing PU development in patients compared with a standard hospital mattress. At the moment the manufacturers only require a CE (Conformité Européenne) marking as a sign that the product is safe for use in a hospital. This CE marking has four risk classes. The manufacturer decides the risk class his product falls under. Most if not all mattresses fall under the risk class I, which indicates a low risk and only requires a technical report showing that the product is safe for use [24]. Receiving a CE marking is no guarantee that the product is effective in what it is supposed to achieve. A suggestion for the near future would be that each request for a CE marking should be accompanied by a report from an independent agency (University hospital, technical universities or other technical institutions), which supplies (scientific) evidence that the new mattress meets the efficacy and the safety criteria satisfactorily. Such a report can only be demanded by the Ministry of Health supported by leading experts in this area. In order to compare different mattresses, a protocol detailing the research including the methodology, the analyses and the format of the reports should be compiled as a guideline for the manufacturers. The CBO may co-ordinate such a process. Only such an incentive

will separate the chaff from the corn and the mattresses that are marketed would adequately serve the needs of the patients.

A positive (side) effect of this, which should not be underestimated, is that the manufacturers must collaborate with healthcare institutions for the required research. This would help them to focus on the needs of the patients in an early phase of product development. For the healthcare institutions this would be an opportunity not only to be a consumer but also to be a partner with responsibility for developing a product for their patients. Such a formal partnership would benefit the patients, the manufacturers and the hospitals. As mattress manufacturers place their mattresses in CE class I there is no need to use dummies or healthy volunteers to investigate the pressure-reducing capacity of such mattresses. Therefore, manufacturers have no reason for hesitating to let patients participate in any research project they claim their product to be safe for use in a hospital. More complex mattress systems should be assigned to the CE class II and up, but require further investigation. The comparison with drug evaluation in the laboratory may be relevant, both in healthy volunteers and in patients.

Today, there are many different types of mattresses on the market and it is tempting to work according to a general consumer principle to get the best value for the lowest price. This raises the question of what is the best, as besides the TIP we can evaluate 8 other criteria [22]. What is the best for one patient group does not need to be the best for another group. This dilemma is illustrated by the investigations described in chapter 7. Judging what is best according to the measured TIP did not result in any significant difference between the ROHO® and the Waffle® mattresses. This raises the question of whether selecting the best pressure distributing mattresses for our patients is the most effective strategy. Springle suggested working according to the principle of selecting the inappropriate mattresses based on research rather than selecting the appropriate ones based on their efficacy [25]. This would provide a ranking of mattresses that are effective. Such a ranking must be based on research. Such lists may be compiled for specific PU risk (low, medium and high) groups of patients. Lists may also be compiled according to the three clustered criteria (product safety, product efficacy and product cost / benefit). This would provide healthcare institutions with a choice as to the most important criteria in relation to the types of patients at their institution.

Ranking the performance of the mattress in a specific patient group with a risk level (high, medium, low) of developing a PU binds the two halves of this thesis. Daily PU risk assessment is essential for determining the patient's risk for PU development. Specific categories of pressure distributing mattresses can be used based on this risk level. Information on the anticipated efficacy that can be achieved is obtained when the pressure-reducing capacity of each type of mattress has been evaluated in a clinical trial.

Future research has to focus more on continuous TIP measurements in tandem with routine skin inspection [25]. This enables the researchers to find general TIP threshold valvues for particular patients or even patient groups. This would provide a strong evidence-based recommendation for clinical practice. For this purpose, collaborative efforts

by nurses, physicians, scientists and statisticians must help in designing research and implementation protocols.

In general terms, the future research in the area of PU prevention must shift towards a new paradigm. The research over the last 50 years has highlighted much isolated and fragmented knowledge in a large number of healthcare disciplines. The anticipation that PU development would not be a clinical issue in the year 2000 has not been achieved in spite of the large number of research articles that have been published in this field. According to Kuhn, there is no scientific progress when only knowledge is accumulated over time, but occurs when a shift in paradigm takes place because the old paradigm is unable to answer the relevant clinical and research questions [26]. The new paradigm for preventing PU development could well be that clinically oriented research should always be multi-centered and carried out by a multidisciplinary team. This implies that the physicians and the nurses are always involved in the research, and in implementing its results. The multi-center aspects would also prevent the possible bias of single organization issues affecting the results. Although this work involves a lot of complicated planning and preparation, it may be anticipated that both the prevalence and the incidence of PU will decrease by implementing research-based PU prevention strategies.

Literature

1. Defloor T. The risk of pressure sores: a conceptual scheme. J Clin Nurs 1999;8:206-16.

2. Dealey C. The size of the pressure-sore problem in a teaching hospital. J Adv Nurs 1991;16:663-70.

3. Bours GJ, De Laat E, Halfens RJ and Lubbers M. Prevalence, risk factors and prevention of pressure ulcers in Dutch intensive care units. Results of a cross-sectional survey. Intensive Care Med 2001;27:1599-605.

4. Halfens RJ. Risk assessment scales for pressure ulcers: a theoretical, methodological, and clinical perspective. Ostomy Wound Manage 2000;46:36-40, 2-4.

5. Waterlow JA. Reliability of the Waterlow score [letter; comment]. J Wound Care 1995;4:474-5.

6. Lorenz E, The essence of Chaos. 1993, Washington: University of Washington Press.

7. Weststrate JTM and Drost NMC. The potential of computers in pressure ulcer care (for the elderly). In 3rd EPUAP Open Meeting report. 1999. Amsterdam.

8. CBO, Richtlijn Decubitus, tweede herziening. 2002, Utrecht: Kwaliteitsinstituut voor de gezondheidszorg (CBO). 132.

9. EPUAP, Pressure ulcer prevention guidelines, European Pressure Ulcer Advisory Panel, 2004, www.epuap.org.glprevention.html

10. Bours GJJW, Halfens RGJ and Wansink SW, Landelijk prevalentie onderzoek decubitus: resultaten vijfde jaarlijkse meting. 2002. Maastricht. Universiteit van Maastricht, sectie Verplegingswetenschap.

11. Bours GJJW, Halfens RGJ and Wansink SW, Landelijk prevalentie onderzoek decubitus: resultaten zesde jaarlijkse meting. 2003. Maastricht. Universiteit van Maastricht, sectie Verplegingswetenschap.

12. Hunt J. Indicators for nursing practice: the use of research findings. J Adv Nurs 1981;6:189-94.

13. Speet M and Franck AL, Individuele professionalisering van verpleegkundigen in de beroepsopleiding en in de praktijk. 2004. Utrecht. NIVEL.

14. Schoonhoven L, Haalboom JRE, Bousema MT, et al. Prospective cohort study of routine use of risk assessment scales for the prediction of pressure ulcers. Br Med J 2002;325:797-800.

15. British Department of Health, The essence of care. 2001. British Department of Health.

16. Braden B and Bergstrom N. Risk assessment and risk-based programs of prevention in various settings. Ostomy Wound Manage 1996;42 (suppl 10A):6s-12s.

17. RIVM, Decubitus, omvang van het probleem [Pressure Ulcer, extent of the problem],RIVM,2004, www.rivm.nl/vtv/data/kompas/gezondheidstoestand/ziekte/decubitus/decubitus_omvang.html

18. Dutch Ministry of Health, Evaluation of quality law Dutch healthcare institutions [Evaluatie kwaliteitswet zorginstellingen]. 2003. 1-6.

19. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med & Rehab 1959;40:62-9.

20. Moore Z. Pressure ulcer prevention: nurses' knowledge, attitudes and behaviour. J Woundcare 2004;13:330-4.

21. Bain DS and Ferguson-Pell M. Remote monitoring of sitting behaviour of people with spinal cord injury. J Rehabil Res Dev 2002;39.

22. Krouskop T and van Rijswijk L. Standardizing performance-based criteria for support surfaces. Ostomy Wound Manage 1995;41:34-45.

23. Heule F, van der Eijk D and Doeff A. An European public tender for anti-decubitus mattress material in an academic hospital center: a challenging process. In 7th European Pressure Ulcer Advisory Panel Open Meeting. 2003. Tampere, Finland: EPUAP.

24. Dutch Ministry of Health, CE marking of medical aids. 1998. Rijswijk. Dutch Ministry of Health.

25. Springle P. Effects of forces and the selection of support surfaces. Top Geriatric Rehabil 2000;16:47-62.

26. Kuhn TS, The structure of scientific revolutions. 1962, Chicago: The University of Chicago Press.

Summary

In the two most cited conceptual models on the development of pressure ulcers (PUs) the exerted pressure (influenced by extrinsic factors) and the tissue tolerance (influenced by intrinsic factors) are described as two critical parameters. However, PU will not develop if there is no pressure even when tissue tolerance is decreased. In contrast, when pressure is exerted, the level of tissue tolerance determines the timeframe in which the PU will develop.

Qualitative measurement techniques have been adopted for both the main aspects. Numerous devices for measuring the pressure between the skin of the patient and the adjoining surface (interface pressure) have been developed over the last decades. PUs may develop at any site on the human body, but the favored sites are where a thin layer of skin covers bony prominences like the sacrum, the buttocks, the scapulae and the heels. Most research on measuring the interface pressure has been done at these sites.

The tissue tolerance is quantified by using risk assessment instruments. The original purpose was to use these as risk assessment instruments in order to assess the individual risk a patient has in PU development. Recent research has indicated that flipping a coin is just as accurate in predicting PU development compared with using a risk assessment instrument. Not withstanding this assumption, the main purpose of the risk assessment instrument is to act as a warning for the physicians and the nurses so that the patients at a high risk for developing a PU can be identified early. This in combination with the clinical judgment of the nurses determines if and which preventive interventions are necessary.

Three investigations in relation to the prevalence of PU and the use of risk assessment instruments in an intensive care setting are described in this thesis. The repeatability of tissue interface pressure (TIP) measurements in patients, the difference between the TIP measured in patients and volunteers and the evaluation of the pressure-reducing capacity of three operating room table mattresses in patients during surgery are described in the other three investigations.

The clinical characteristics when pressure is applied to the skin followed by the current state of the art of what is known on the etiology of pressure ulcer development is described in **chapter 1**. Next a historical background on how the TIP measurement technique has developed over the last decades is given. Following this, different types of TIP sensors and their behavior in a clinical setting are discussed and evaluated. Subsequently, the different methods for analyzing the gathered data are discussed. The decision to use the Talley Pressure Monitor III (TPM III) for our research program was based on the outcome of the analyses of previous tests.

The second part of this chapter deals with the use and the interpretation of PU risk assessment instruments. A number of risk assessment instruments that are

specifically related to the risk factors that intensive care patients have are discussed. Finally, the motivation as to why the Waterlow risk assessment scale was selected for assessing the risk of PU development in intensive care patients is given.

The results of a frequently carried out PU point prevalence at a surgical intensive care unit (ICU) (short- and long-stay unit) are described in **chapter 2**. Besides the number and the stage of PU, the risk of developing a PU and the use of the Waterlow PU risk assessment instrument was assessed and the number of implemented preventive measurements was investigated. The results showed that the patients who stayed longer at the ICU had a higher PU prevalence compared with patients whose stay was shorter. The PU prevalence varied significantly over the days. This was mainly caused by the high turnover of patients who were admitted and discharged daily. The average prevalence at the short-stay unit was 13.6% and at the long-stay unit was 42.1%. The Waterlow risk assessment score in patients with PU grade 0 was statistically significant lower compared with those with grade III. This indicated certain sensitivity in detecting differences in PU risk assessments between the various PU grades in patients. There was a positive correlation between the number of preventive measurements and the PU grade indicating that ICU nurses were primarily alerted by the visual signs of PU development and not by the score of the risk assessment scale. It was concluded that PU development is a serious problem in intensive care patients and that using a risk assessment instrument like the Waterlow PU risk assessment scale can help intensive care nurses to identify high risk patients early.

The results of a questionnaire study on PU prevention strategies at a number of critical care units in Europe are given in **chapter 3**. The questionnaire consisted of two parts; the first with questions related to the organizational issues with regard to PU prevention strategies and the second with a number of questions related to the individual patients present on a particular day at the unit. In total 44 units in 4 countries (Denmark, Italy, Germany and the Netherlands) participated in the study. The main results were that most units used a PU prevention and treatment protocol and had a nurse who specialized in this area working in the ward. At about 70% of the units, a PU risk assessment scale was used. The other 30% used their clinical judgment to assess the patient's risk of developing a PU.

In total 299 patients were evaluated in the study. The average PU prevalence (grade II and higher) was 27%. This varied considerably between the countries from 4% - 49%. The main risk factor was described as decreased mobility followed by decreased activity, increased sensitivity and the use of vasoactive medication. The use of a pressure reducing mattress in relation to the PU risk level of patients had varied significantly between the countries. It was concluded that there is a need in Europe for standardization of protocols regarding PU prevention

strategies in critical care patients. Organizations like EPUAP, EFCCNa and the ESICM can play an important role in this area.

A study in which the clinical usefulness of the Waterlow PU risk assessment scale for intensive care patients was evaluated for creating a PU risk profile is dealt with in **chapter 4**. This prospective study was carried out at a surgical ICU. Data was collected from 594 patients who had stayed for longer than 24 hours at the ICU. Each patient was assessed daily with respect to their Waterlow PU risk score and the presence of a PU (grade II and higher) in the sacral region. Actuarial statistical methods were used to analyze the predictive value of the PU risk profile based on the Waterlow risk assessment scale. The PU incidence was 7.9%. There was a significant difference between the sex, the stay at the ICU and the Waterlow score between patients who did and did not develop a PU. The results showed that as long as patient's Waterlow PU risk score stayed below the score of 15, they did not develop a PU, whereas if patients had a Waterlow PU risk score of above 25, their risk profile in developing a PU increased sharply. The results also demonstrated that the PU risk assessment of the last 24 hours is the best indicator for the development of a PU within the next 24 hours. Further, it was shown that relying only on the clinical assessment of the patient's skin for initiating PU preventive measures did not prevent the PU development, but often only confined the extent of damage. It was concluded that the risk profile model based on the Waterlow risk assessment scale could be used in the ICU. However, as the risk profile model is based on the number and the frequency of standard PU preventive interventions, it cannot be translated without considering the context to other intensive care units as they may use other PU prevention protocols and interventions. Consequently each unit has to develop its own model.

A study in which the reproducibility of TIP measurements is evaluated in a patient population is presented in **chapter 5**. The significance of this study is based on the fact that although the exerted pressure is the main cause of PU development, PU risk assessment instruments used in clinical practice are only based on factors that influence the tissue tolerance. Combining the value of the individually generated TIP with the value of the PU risk instrument may produce a more accurate PU risk profile of patients during their stay in hospital. For the use of the TIP value in this way, its reproducibility must be evaluated.

The "between day" reproducibility of the TIP at the sacrum and the buttocks was assessed with the TPM III in 76 surgical patients who had bed rest at least for a period of 4 days. The reproducibility of the TIP measurements, with an interval of at least 24 hours, was assessed by calculating the Intra-Class Correlation Coefficient (ICC) of the Peak Interface Pressure (PIP) over the two measurements. The main results showed a low ICC (range 0.13-0.23) over the two measurements. Of all

extraneous variables, only the elevation of the backrest had a significant influence on variation of the PIP. It was concluded that TIP measurements in a patient population are not reproducible after a minimum time interval of 24 hours. Therefore, the patient's individually generated TIP value cannot be used in creating an individual PU risk profile model in combination with the value of the PU risk assessment instrument.

An investigation purpose to compare the measured TIP between patients and volunteers lying on the same type of mattress is described in **chapter 6**. The relevance of this study was based on the fact that the pressure-reducing capacity of mattresses is mostly evaluated by measuring the TIP in healthy volunteers. Patients can generate different TIPs compared with those generated by the volunteers due to pain and other uncomfortable conditions. Therefore, it is important to investigate if the generated TIPs in patients are in the same range compared with those generated by the volunteers.

The TIP was measured with the TPM III at the sacrum and the buttocks in 28 patients and 30 healthy volunteers. The TIP measurements in patients were carried out in their most preferred position. The TIP measurements in volunteers were carried out in the three most used positions: supine, backrest elevated to 30^0 and backrest elevated to 30^0 with 22^0 Fowler's position. The results showed that the PIPs measured at the sacrum and the left buttock in the patients were significantly higher than those measured in the healthy volunteers in all three positions. It was concluded that the patients generated higher PIPs than those generated by the healthy volunteers. Therefore, the results of an evaluation into the pressure-reducing capacity of mattresses carried out in healthy volunteers should be interpreted with caution when the mattresses are meant for use in patients.

A study in which the pressure-reducing capacity of three types of operating table mattresses was evaluated in 63 patients undergoing surgery is presented in **chapter 7**. The purpose of the study was to evaluate which type of mattress reduced the exerted pressure most effectively and is suitable for use on operating tables. The pressure-reducing capacity was evaluated by measuring the TIP with the TPM III at the sacrum, the left buttock and both the heels. The TIP was measured every 15 minutes for a period of 5 minutes, so that the stability of the measurement during the time of surgery could also be evaluated. The PIP within each 5 minutes of the measurement was the primary outcome parameter. The three types of mattresses that were evaluated were the Action® visco-elastic dry polymer mattress, the Waffle® air mattress and the ROHO® air mattress.

The results showed that the Action® mattress produced significantly higher PIPs compared with the other two. There were no significant differences in PIP between the Waffle® and the ROHO® mattresses at any of the body sites.

Therefore, it was concluded that the Waffle® and the ROHO® mattresses were best suited for use on the operating table during surgery because they distributed the exerted pressure adequately in the patients.

The implications of the studies described in this thesis for clinical practice are discussed in **chapter 8**. After decades of research, PU development is still a significant clinical complication that on the average occurs in one out of every 5 patients admitted to hospital. This is even more frequent in ICU patients. The investigations described in this thesis show that this complication is an international problem and not only a problem for the patients in the Netherlands. Daily clinical assessment of the patient's risk for developing a PU is a critical component in its prevention. The use of a risk profile model that can provide insight not only into the risk over 24 hours but also beyond can be of considerable value. Further research in this area should focus on the type and frequency of PU preventive measurements on the model. Incorporating the average measured PIP of a standard and pressure-reducing mattress may also be of considerable value for the clinical practice.

Evaluating the pressure-reducing capacity of mattresses is an important item as each year new types of mattresses come on the market. In order to separate the chaff from the corn measuring the generated TIPs in a patient population provides valuable information. Next to evaluating a number of other critical characteristics, a rank order can be given to the mattresses varying from good to poor effectiveness. By removing the inappropriate type of mattress in combination with providing the best possible care for the patient, the development of PUs can be reduced significantly in the future.

As mentioned in the title, this thesis has been written from a nursing perspective. Therefore, it should also include a specific set of recommendations for the nurses with emphasis on the important role and the position they occupy in the care of the patients. The prevention of PUs is a multidisciplinary responsibility; the healthcare chain is as strong as its weakest link. To make sure that this is not the case for the nurses the following recommendations are made.

In terms of responsibility, the nurses should at least carry out daily PU risk assessments in combination with a physical assessment of the patient's skin. The results of this should be documented in the nursing notes. This also provides a clear report of the patient's condition when there are transfers of patients between different units within the hospital or between different healthcare institutions. As the nurses in the hospital observe the patient almost continuously, they are the pre-eminent discipline for initiating uni- and multidisciplinary collaborative consultations around this issue.

In terms of knowledge, the nurses should commit themselves to continuous schooling in this area by being involved in clinical research. Participation in reporting the incidence and the prevalence helps to face the reality of the PU

problem and generates relevant questions for clinical researchers. As there are still numerous unanswered (research) questions in this field, initiating or participating in new research stimulates an interest in finding answers to those questions.

In terms of attitude, it is important that nurses commit themselves to following the national and international PU prevention guidelines. The use of process parameters as quality indicators instead of outcome criteria facilitates the nurses to evaluate their own performance. Combined with participating in benchmarking projects, the nurses are encouraged to learn from each other when differences are noted.

Samenvatting

In de twee meest geciteerde conceptuele modellen met betrekking tot het ontstaan van decubitus zijn de uitgeoefende druk (beïnvloed door de combinatie van extrinsieke factoren) en de weefseltolerantie (beïnvloed door het totaal van intrinsieke factoren) beschreven als de twee kritische parameters. Alhoewel, zelfs als er geen druk wordt uitgeoefend en met een verlaagde weefseltolerantie zal zich er geen decubitus ontwikkelen. Wanneer er echter wel druk wordt uitgeoefend, zal de mate van weefseltolerantie de tijd bepalen waarbinnen zich de decubitus zal ontwikkelen. Kwantitatieve meettechnieken zijn voor beide belangrijke aspecten ontwikkeld. De laatste jaren zijn er verscheidene instrumenten ontwikkeld die de druk tussen de huid en het onderliggend matras meten (interfacedruk). Hoewel decubitus zich in principe op iedere locatie van het lichaam kan ontwikkelen, heeft het wel een aantal voorkeursplaatsen. Dit zijn met name locaties waar een dunne huidlaag zich over een benig uitsteeksel plooit zoals het geval is bij het sacrum, het zitbeen, de schouderbladen, de hielen etc. Het meeste onderzoek met betrekking tot meten van de interface druk vindt dan ook plaats op deze locaties.

De weefseltolerantie wordt gekwantificeerd met behulp van risicobeoordeling-instrumenten. Het oorspronkelijke doel van deze instrumenten was om het individuele risico op het ontstaan van decubitus bij patiënten in kaart te brengen. In een later stadium heeft men getracht deze instrumenten te gebruiken om het ontstaan van decubitus te voorspellen. Recent onderzoek laat zien dat het opgooien van een munt hierin een net zo nauwkeurig antwoord geeft als het instrument.

Het belangrijkste doel van een dergelijk instrument is om artsen en verpleegkundigen de mogelijkheid te geven een patiënt met een verhoogd risico op decubitus vroegtijdig te identificeren. Dit, in combinatie met een klinische beoordeling van de patiënt door de verpleegkundige, bepaalt wanneer en welke preventieve interventies noodzakelijk zijn.

Drie onderzoeken in relatie met de preventie van decubitus en het gebruik van risicobeoordelinginstrumenten op de intensive care afdeling zijn beschreven in dit proefschrift. De reproduceerbaarheid van de interfacedruk gemeten in patiënten, het verschil in interfacedruk gemeten bij patiënten en vrijwilligers en de evaluatie van de drukreducerende capaciteit van drie operatietafelmatrassen bij patiënten tijdens een chirurgische ingreep zijn beschreven in drie andere onderzoeken.

De klinische verschijnselen van druk op de huid en de huidige "state of the art" wat betreft de etio-pathologie van decubitus is beschreven in **hoofdstuk 1**. Vervolgens wordt een overzicht gegeven, hoe de techniek van het meten van de interfacedruk zich de laatste jaren heeft ontwikkeld. Hierna wordt het gebruik van verschillende sensoren en hun gedrag in een klinische setting bediscussieerd en geëvalueerd,

waarna verschillende analysemethoden van de verzamelde data worden besproken. Het besluit om de Talley Interfacedruk Monitor (TPM III) te gebruiken voor deze onderzoeken is gebaseerd op deze analyse.

Het tweede gedeelte van het hoofdstuk beschrijft het gebruik en de interpretatie van decubitus risicobeoordelinginstrumenten. Een aantal van deze instrumenten waarin specifieke risicofactoren zijn opgenomen die relevant zijn voor de ICU patiënten worden hier besproken. Vervolgens wordt beargumenteerd waarom het Waterlow risicobeoordelinginstrument is gekozen om het risico op decubitus bij ICU patiënten te beoordelen.

De resultaten van een frequent uitgevoerde decubitus puntprevalentie studie op een chirurgische intensive care unit (kort en langdurig verblijf) staat beschreven in **hoofdstuk 2**. Naast het aantal patiënten met decubitus, de gradering, het risico op het ontwikkelen van decubitus en het gebruik van het Waterlow decubitus risicobeoordelingsinstrument werd ook het aantal en type decubitus preventieve maatregelen onderzocht. De resultaten laten zien dat patiënten die langer op de intensive care verbleven een hogere prevalentie van decubitus hadden in vergelijking met diegenen van wie het verblijf kortdurend was. Tevens verschilde de prevalentie van decubitus significant tussen deze dagen dat gemeten werd. Dit werd met name veroorzaakt door het grote aantal patiënten dat dagelijks werd opgenomen en ontslagen. De gemiddelde prevalentie op de kortverblijf-unit bedroeg 13.6% en op de langverblijf-unit was dit 42.1%. De Waterlow decubitus risicoscore bij patiënten die geen tekenen van decubitus hadden was statistisch significant lager in vergelijking met diegenen die decubitus graad III hadden. Dit laat een zekere sensitiviteit zien in de verschillen van risico op decubitus tussen patiënten met verschillende gradaties van decubitus. Tevens was er een positieve correlatie tussen het aantal uitgevoerde preventieve maatregelen en de gradatie van decubitus wat liet zien dat verpleegkundigen op de ICU hoofdzakelijk door de klinische verschijnselen hun beleid laten bepalen en niet door de hoogte van het decubitus risicobeoordelinginstrument. Geconstateerd werd dat de ontwikkeling van decubitus bij patiënten op de intensive care een serieus probleem is en dat gebruik van een risicobeoordelinginstrument als de Waterlow decubitus risicoschaal verpleegkundigen kan helpen om patiënten met een hoog risico te identificeren.

De uitkomsten van een enquête naar decubitus preventieve maatregelen op een aantal intensive care afdelingen in Europa staan vermeld in **hoofdstuk 3**. De enquête bestond uit twee delen. Het eerste deel bestond uit vragen met betrekking tot de organisatie van de afdeling in relatie tot decubitus preventieve maatregelen. Het tweede gedeelte bestond uit vragen die betrekking hadden op het risico op decubitus van de individuele patiënten die op een bepaalde dag aanwezig waren op de ICU. In totaal deden 44 afdelingen mee verdeeld over vier landen (Denemarken, Italië,

Duitsland, en Nederland). De belangrijkste resultaten waren dat de meeste afdelingen een decubitus preventie- en behandelprotocol gebruikten en dat er een verpleegkundige op de afdeling aanwezig was die gespecialiseerd was op dit gebied. Op ongeveer 70 % van de afdelingen werd een decubitus risico beoordelingsinstrument gebruikt. De resterende 30% gebruikte alleen de klinische blik om het risico op decubitus bij de patiënt in te schatten.

In totaal werden 299 patiënten geëvalueerd in de studie. De gemiddelde prevalentie van decubitus in de studie (graad II en hoger) bedroeg 27%. Dit getal varieerde behoorlijk tussen de vier landen (4%-49%). De belangrijkste risicofactor werd omschreven als een verminderde mobiliteit gevolgd door een verminderde activiteit en het gebruik van vasoactieve medicatie. Het gebruik van een drukverlagend matras in relatie met de decubitus risicoscore van patiënten varieerde ook sterk tussen de verschillende landen. Concluderend werd gesteld dat er in Europa behoefte is aan een zekere standaardisatie van protocollen in relatie tot decubitus preventieve maatregelen bij intensive care patiënten. Organisaties als de EPUAP, EFCCNa en de ESICM kunnen hierin een belangrijke rol spelen.

Een studie waarbij de klinische bruikbaarheid van een risicoprofiel op basis van het Waterlow decubitus risicobeoordelinginstrument is geëvalueerd bij patiënten op de intensive care afdeling staat beschreven in **hoofdstuk 4**. Deze prospectieve studie is uitgevoerd op een chirurgische intensive care afdeling. Data werden verzameld van 594 patiënten die langer dan 24 uur op de intensive care afdeling verbleven. Iedere patiënt werd dagelijks beoordeeld met betrekking tot de Waterlow decubitus risicoscore en de aanwezigheid van de decubitus (graad II of hoger) op de stuit. Actuariële statistische methoden zijn gebruikt om de voorspellende waarden van het decubitus risicoprofiel op basis van de Waterlow decubitus risicobeoordelingschaal te analyseren.

De incidentie van decubitus tijdens de gehele studie was 7,9%. Er bestond een significant verschil tussen de patiënten die wel en geen decubitus ontwikkelden met betrekking tot geslacht, de verblijfsduur op de intensive care en de Waterlow decubitus risicoscore. De resultaten lieten zien dat, zolang een patiënt's Waterlow decubitus risicoscore onder de 15 punten is deze geen decubitus ontwikkelde op de intensive care afdeling. Dit in tegenstelling tot patiënten met een score van 25 punten of hoger. Hun risico op het ontwikkelen van decubitus steeg bijzonder snel. Tevens laten de resultaten zien dat de risicobeoordeling van decubitus over de laatste 24 uur de beste indicator is voor het al dan niet ontwikkelen van decubitus in de volgende 24 uur. Het werd duidelijk dat het inzetten van preventieve maatregelen alleen op basis van een klinische beoordeling van de huid de ontwikkeling van decubitus niet tegenging maar alleen mogelijk de omvang van de schade beperkte. De conclusie van het onderzoek was dat een risicoprofiel gebaseerd op het Waterlow decubitus risicobeoordelinginstrument effectief kan worden ingezet op de intensive

care afdeling. Een aantekening hierbij is dat het risicoprofiel omdat het mede gebaseerd is op het type en de frequentie van standaard decubitus preventieve maatregelen, niet zomaar op een andere intensive care afdeling gebruikt kan worden, omdat zij mogelijk andere decubitus preventie maatregelen en interventies gebruiken. De conclusie is dan ook dat iedere unit zijn eigen risicoprofiel zal moeten ontwikkelen.

Een onderzoek waarin reproduceerbaarheid van de interfacedruk op de huid (TIP) is geëvalueerd in een patiëntenpopulatie staat beschreven in **hoofdstuk 5**. Het belang van deze studie is gebaseerd op het feit dat hoewel de uitgeoefende druk de belangrijkste oorzaak is voor het ontstaan van decubitus de decubitus risicobeoordelinginstrumenten die in de praktijk worden gebruikt alleen maar gebaseerd zijn op factoren die de tolerantie van de huid ten aanzien van de druk beïnvloeden. Een combinatie van de individueel gemeten TIP gecombineerd met de waarde van het decubitus risicobeoordelinginstrument kan een meer nauwkeurig decubitus risicoprofiel van de patiënt geven gedurende zijn verblijf in het ziekenhuis. Om de TIP op een dusdanige manier te gebruiken moet de reproduceerbaarheid van de meting eerst aangetoond worden.

De TIP op de stuit en de bil werd op twee momenten met een tijdsinterval van tenminste 24 uur ("tussendag" reproduceerbaarheid) gemeten met de TPM III bij 76 chirurgische patiënten die bedrust hadden voor een periode van tenminste vier dagen. De reproduceerbaarheid van de twee TIP-metingen werd beoordeeld met behulp van de Intra-Class Correlation (ICC) van de Piek Interface Druk (PIP) van iedere meting. De belangrijkste uitkomsten laten een lage ICC zien (bereik 0,13-0.23) over de twee metingen. Van alle andere factoren had alleen de elevatie van de hoofdsteun significante invloed op de variatie van de PIP. De conclusie van het onderzoek was dan ook dat de PIP in een patiëntenpopulatie niet reproduceerbaar is bij een minimum tijdsinterval van 24 uur. Hierdoor kan de individueel berekende PIP van de patiënt niet gebruikt worden om een individueel decubitus risicoprofiel te vervaardigen in combinatie met de waarde van het decubitus risicobeoordelinginstrument.

Een onderzoek met als doel het vergelijken van de TIP tussen patiënten en gezonde vrijwilligers liggend op hetzelfde type matras staat beschreven in **hoofdstuk 6**. Het belang van deze studie is gebaseerd op het feit dat de drukverlagende capaciteit van matrassen vaak wordt geëvalueerd met behulp door de TIP te meten bij gezonde vrijwilligers. Ten gevolge van pijn en andere oncomfortabele omstandigheden kunnen patiënten andere TIP-waarden produceren. Het is dan ook van belang om te onderzoeken of de gemeten TIP-waarden bij patiënten in dezelfde range liggen in vergelijking met de TIP waarden gemeten bij vrijwilligers. De PIP werd berekend op basis van de gemeten TIP met de TPM III aan de stuit en bil bij 28 patiënten en

30 vrijwilligers. De TIP-metingen bij patiënten werden uitgevoerd in hun meest comfortabele positie van dat moment. De metingen bij vrijwilligers werden uitgevoerd in de drie meest gebruikt posities n.l. plat op bed, hoofdsteun op 30^0 en hoofdsteun op 30^0 gecombineerd met de positie van Fowler in 22^0. De resultaten laten zien dat de PIP aan de stuit en de linker bil bij patiënten hoger uitvalt in vergelijking met die bij gezonde vrijwilligers in alle drie de posities. De conclusie was dan ook dat patiënten een hogere PIP genereren in vergelijking met gezonde vrijwilligers op een zelfde type matras. Als conclusie kan dan ook worden gezegd dat de resultaten van een matrasevaluatie m.b.t. het drukreducerend vermogen vastgesteld met behulp van gezonde vrijwilligers kritisch moeten worden bestudeerd wanneer het matras voor een patiënten populatie gaat dienen.

Een onderzoek waarin het drukreducerend vermogen van drie operatietafelmatrassen werd beoordeeld is beschreven in **hoofdstuk 7**. Het doel van de studie was om te bestuderen welk type matras de uitgeoefende druk het meest effectief kon reduceren en geschikt was voor gebruik op de operatietafel. De drukreducerende capaciteit werd bestudeerd door het meten van de TIP met behulp van de TPM III aan de stuit en de linker bil en beide hielen. De TIP werd iedere vijftien minuten gedurende vijf minuten gemeten zodat tevens de stabiliteit van de meting kon worden geëvalueerd. De PIP over iedere 5 minuten was de primaire uitkomst variabele. De drie matrastypes die zijn geëvalueerd waren de Action® Visco-Elastische droge polymer matras, de Waffle® luchtmatras en de Roho® matras.

De resultaten laten zien dat de Action® matras significant hogere PIP-waarden produceerde in vergelijking met de andere twee matrassen. Er bestonden geen significante verschillen in PIP waarden tussen de Roho® en Waffle® matras op alle drie de lichaamslocaties. De conclusie van het onderzoek was dan ook dat de Waffle® matras en de Roho® matras het meest de druk bij patiënten reduceerde en hierdoor het meest geschikt waren voor gebruik op de operatietafel.

De gevolgen van de onderzoeksresultaten voor de klinische praktijk worden besproken in **hoofdstuk 8**. Na tientallen jaren van onderzoek naar de ontwikkeling van decubitus is het nog steeds een belangrijke klinische complicatie die gemiddeld bij één op de vijf patiënten in het ziekenhuis voorkomt. Bij patiënten opgenomen op de ICU ligt dit getal zelfs hoger. De studies die in dit proefschrift staan beschreven en de referenties laten zien dat deze complicatie een internationaal probleem is en dus niet alleen bij patiënten in Nederland voorkomt. Dagelijkse klinische beoordelingen naar het risico op het ontstaan van decubitus bij patiënten is een kritische component in de preventie ervan. Het gebruik van een risicoprofielmodel kan niet alleen inzicht geven in de hoogte van het risico over de eerste 24 uur maar kan ook op langere termijn van waarde zijn. Verder onderzoek moet zich richten op de invloed van decubitus preventieve maatregelen op dit model. Het opnemen van

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de gemiddelde PIP-waarde van een standaard matras en een drukreducerend matras kan van betekenis zijn voor de praktijk. Het evalueren van de drukreducerende capaciteit van matrassen is een belangrijk gegeven omdat jaarlijks tal van nieuwe matrassen op de markt verschijnen. Om het kaf van het koren te scheiden kan het meten van de TIP in een patiënten populatie waardevolle informatie opleveren. Naast het meten van een aantal andere kritische parameters kan dan een rangorde worden samengesteld variërend van geschikt tot ongeschikt voor gebruik bij patiënten. Door de ongeschikte matrassen te verwijderen in combinatie met het geven van de best mogelijke zorg aan de patiënt kan in de toekomst de aanwezigheid van decubitus belangrijk worden teruggedrongen.

Zoals in de titel staat vermeld is dit proefschrift geschreven vanuit een verpleegkundig perspectief. Daarom ook zijn aanbevelingen ook op hun plaats die het belang van de rol en positie van verpleegkundigen in de zorg voor patiënten m.b.t. dit onderwerp benadrukken. De preventie van decubitus is een multidisciplinaire verantwoordelijkheid. De zorgketen hiervan is dan ook zo sterk als de zwakste schakel. Verpleegkundigen kunnen ervoor zorgen dat deze schakel tenminste niet in hun domein ligt door de volgende aanbevelingen vorm te geven in hun dagelijkse praktijk.

In termen van verantwoordelijkheid zouden verpleegkundigen tenminste dagelijks een decubitus risicobeoordeling moeten uitvoeren in combinatie met een klinische inspectie van de huid. De bevindingen hiervan dienen te worden gedocumenteerd in het verpleegkundige dossier. Een dergelijke verslaglegging geeft tevens een heldere beschrijving van de conditie van de patiënt wanneer deze wordt overgeplaatst naar een andere afdeling of instelling. Aangezien verpleegkundigen de patiënt bijna continu observeren vormen zij de discipline bij uitstek om het initiatief te nemen in mono- en multidisciplinaire consultaties op dit gebied.

In termen van kennis kunnen verpleegkundigen zich voortdurend scholen door betrokken te zijn bij klinisch wetenschappelijk onderzoek op het gebied van decubitus preventie. Deelname in het rapporteren van de incidentie en prevalentie helpt hen om de realiteit van het probleem onder ogen te zien. Tevens genereert dit het stellen van relevante vragen voor klinisch onderzoekers.

In termen van houding is het van belang dat verpleegkundigen het gebruik van nationale en internationale richtlijnen voor de preventie van decubitus onderschrijven. Het gebruik van proces parameters of uitkomstindicatoren laat verpleegkundigen hun eigen gedrag evalueren. Dit gecombineerd met deelname aan benchmarkprojecten stimuleert het leren van elkaar met name wanneer er verschillen aanwezig zijn.

Dankwoord

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in de onderzoekgroep op de heelkunde intensive care hebben gezeten en jullie betrokken waren bij de dataverzameling bij patiënten. Ik bedank Leo Tegelaar voor het onderschrijven van het belang van onderzoek door verpleegkundigen bij patiënten op de intensive care. Ik zie je nog de knoop doorhakken in de deurpost van kamer 1004. Dit proefschrift is mede door deze beslissing tot stand gekomen. Met jou bedank ik alle intensive care verpleegkundigen van de afdeling die betrokken zijn geweest bij het verzamelen van de data. Ook ben ik dank verschuldigd aan Coos Jabbaaij voor zijn altijd aanwezige bereidheid om acute technische problemen op te lossen. Steven Buijk, Karan Kanhai en Jan Pompe bedank ik voor steun en bereidheid om kritisch naar teksten te kijken. Tevens voor het plezier dat we als onderzoeksgroep met elkaar hadden.

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*In memoriam A.Weststrate (3 februari 2005)

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Curriculum Vitae

Jan Weststrate werd in 1956 geboren te Wemeldinge. In 1973 behaalde hij het M.A.V.O. diploma aan de Johannes Calvijn school te Gouda. In 1974 startte hij met de inservice opleiding tot Verpleegkundige aan de Prinses Margriet school te Rotterdam en was hij werkzaam als leerling verpleegkundige in het Van Dam Ziekenhuis aan de Westersingel te Rotterdam. Onderbroken door de militaire dienstplicht behaalde hij in 1981 zijn diploma aan de School voor Verpleegkunde in Gouda. Begin 1982 verhuisde hij met zijn gezin naar Engeland en werkte daar twee en een half jaar in het st Peters Hospital te Chertsey, Surrey. Na een jaar op een algemeen chirurgische afdeling, heeft hij de resterende tijd op de algemene intensive care afdeling doorgebracht en daar de eerste ervaringen met intensive care verpleegkunde opgedaan. Terug in Nederland begon hij eind 1984 met de opleiding tot intensive care verpleegkundige in het huidige Erasmus Medisch Centrum. In 1986 behaalde hij zijn diploma waarna hij ging werken op de Heelkunde intensive care afdeling. Vanaf 1992 tot 1998 maakte hij deel uit van de onderzoeksgroep van prof.dr.H.A. Bruining waar hij betrokken was bij de uitvoering van contract research en tevens de eerste stappen zette op het gebied van onderzoek naar aspecten van de verpleegkundige zorg aan de intensive care patiënt. In 1994 begon hij met de Master opleiding Verpleegkundig Onderzoek aan de huidige Hogeschool van Utrecht. Toen hij deze in 1997 had afgerond kreeg hij al snel de mogelijkheid om een promotie-onderzoek te gaan uitvoeren. Gezien zijn betrokkenheid bij het onderwerp decubitus preventie en behandeling is dit thema gekozen. Sinds 1 april 2004 werkt hij twee dagen in de week aan de Hogeschool Rotterdam, afdeling verpleegkundige opleidingen, als docent aan de opleiding Master Advanced Nurse Practitioner, twee dagen als zorgonderzoeker binnen het cluster Neurologie, Neurochirurgie en KNO en één dag als project coördinator om de decubitus prevalentie meting te organiseren.

Jan Weststrate is getrouwd met Marian Lubbers en zij hebben twee kinderen Renske en Tjitze.

Publications

1. Weststrate JTM. Meting van therapeutische interventie. Intensive Care Review 1990;5:105-10.
2. Weststrate JTM. Het wassen van patiënten op de intensive care afdeling: Wanneer. Intensive Care Review 1991;6:156-57.
3. Los AL, Weststrate JTM. De open behandeling van de geïnfecteerde buik: Deel 1. Kritiek 1991;1:3-5.
4. Weststrate JTM. Los AL. De open behandeling van de geïnfecteerde buik: Deel 2. Kritiek 1991;2:2-4.
5. Weststrate JTM. Bronchiaal toilet, deel 1: een enquête. Kritiek 1992;3:21-26.
6. Weststrate JTM. Bronchiaal toilet, deel 2 enquête: het wegzuigen van longsecreet. Kritiek 1992;4:17-22.
7. Weststrate JTM. Bronchiaal toilet, deel 3 enquête: het balloneren. Kritiek 1992;5:3-7.
8. Weststrate JTM. Los AL. The 'open' treatment of patients with an infected abdomen. An international forum on wound care 1995;3:7-8.
9. Weststrate JTM. Care of the open wound in abdominal sepsis. Journal of wound care 1996;5:325-8.
10. Weststrate JTM. Bruining HA. Pressure sores in an intensive care unit and related variables. A descriptive study. Intensive and Critical Care Nursing 1996;12:280-4.
11. Weststrate JTM. Open treatment of a wound in abdominal sepsis. Nursing Times 1997;2:13-4.
12. Weststrate JTM. Endotracheaal uitzuigen : Routinematig of op indicatie. Kritiek 1997;1:19-23.
13. Buijk SE, Kanhai KJ, Weststrate JTM. Bruining HA. An insidious case of streptococcal soft tissue infection. European Journal of Surgery 1998;164:153-5.
14. Weststrate JTM. De Waterlow decubitusrisicoschaal voor intensive care patienten. Kritiek 1998;6:26-7.
15. Weststrate JTM. Hop WCJ, Aalbers AGJ, Vreeling AWJ, Bruining HA.The clinical relevance of the Waterlow pressure sore risk scale in the ICU. Intensive Care Medicine 1998;24:815-20.
16. Weststrate JTM. Drost NMC. Automatisering van de Waterlow decubitus risicoschaal: een voorbeeld van samenwerking tussen It-ers en verpleegkundigen. Informatie & zorg 1999;28(2):50-4.
17. Weststrate JTM. Heule F. Prevalence of pressure ulcers, risk factors and use of pressure relieving mattresses in ICU patients. Connect 2001;1(3):77-82.

Appendix

Appendix 1. The Waterlow Pressure Sore Risk Scale (adapted for ICU situation). The *column gives the weighing factor of each element

Build/weight for height	*	Skin type visual risk areas	*	Sex / Age	*	Special Risks	*
Average	0	Healthy	0	Male	1	Tissue Malnutrition	*
Above average	1	Tissue paper, dry, clammy, edematous	1	Female	2	Terminal cachexia	8
Obese	2	Discolored	2	14-49	1	Cardiac failure or catecholamines	5
Below average	3	Broken spot	3	50-64	2	Peripheral vascular disease	5
Continence		Mobility		65-74	3	Anemia	2
Complete catheterized	0	Fully mobile	0	75-80	4	Smoking	1
Occasional incontinence	1	Restless or fidgety	1	81 +	5	Neurological deficit	
Catheter and fecal incontinence	2	Apathetic	2	Appetite		Diabetes, MS[1], CVA[2]	4-6
Incontinent for feces and urine	3	Restricted	3	Average	0	Motor / sensory paraplegia or femoral fracture	
		Inert/ traction	4	Poor	1	Major surgery / trauma	
		Chair bound /complete bed rest	5	NG[3] tubes and fluids only	2	Orthopedic: below the waist or spinal cord	5
				NBM[4] / anorexia	3	Operation time > 2 h	5
						Medication	
						Chemotherapy, high doses steroids, anti-inflammatory drugs	4

[1]. Multiple sclerosis; [2]. Central Vascular Accident, [3]. : Naso Gastric, [4]. : Nil By Mouth

Appendix 2. Description of the various pressure sore stages as defined by the Dutch National Pressure Ulcer Advisory Panel

Stage I   Non-blanchable erythema of intact skin; the heralding lesion of skin ulceration.

Stage II   Partial thickness skin loss involving epidermis and/or dermis; the ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage III   Full-thickness skin loss involving damage or necrosis of subcutaneous tissue which may extend down to, but not through, underlying fascial layer; the ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.

Stage IV   Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g. tendon, joint capsules, etc.)

Appendix 3. An overview of the 3x4 sensor array of the Talley Pressure Monitor III
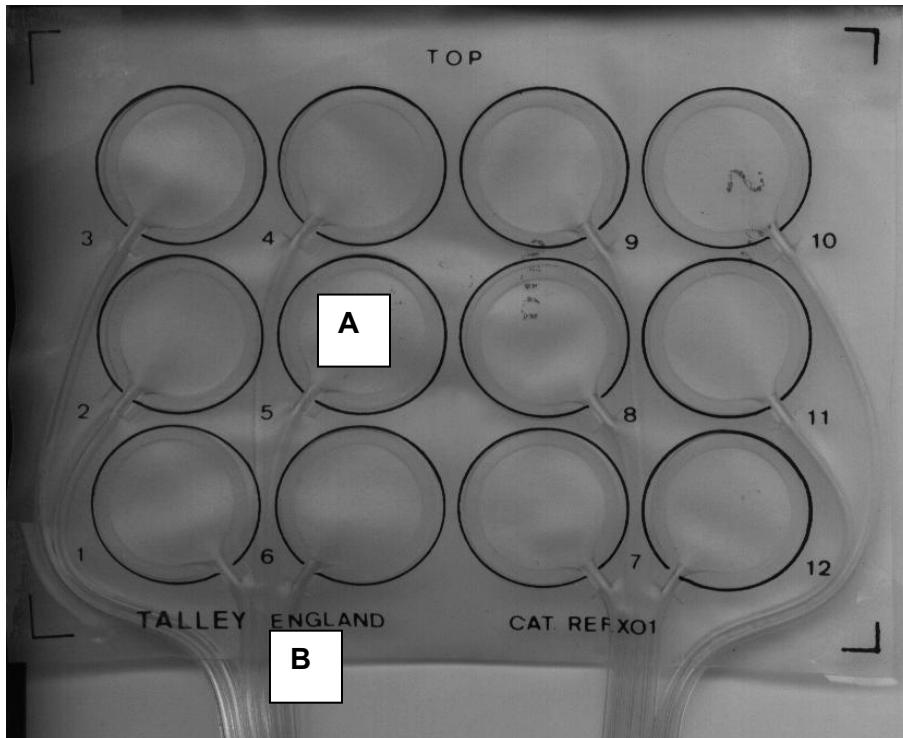
A: The actual sensor
B: Connection tubes to TPM III and monitor