PRESSURE ULCER PREVENTION: FUNDAMENTALS FOR BEST PRACTICE

MARK COLLIER

Nurse/Consultant – Tissue Viability United Lincolnshire Hospitals NHS Trust
c/o Pilgrim Hospital, Boston, Lincolnshire, UK

This introduction has highlighted both the complex nature of the aetiology of pressure ulcer development and the complex nature of the assessment process intended to identify those patients who are or might be at an enhanced risk of pressure ulcer development. The latter statement assumes that all patients cared for in any healthcare setting are vulnerable to pressure ulcer development. Whilst it is acknowledged that the use of a risk assessment tool can be important in an overall pressure ulcer prevention strategy, it is important that the limitations of these tools are acknowledged and that they are not an finite assessment in themselves and that they should be used by a practitioner with a fundamental breadth of relevant knowledge and an appreciation of the range of appropriate preventative equipment/techniques available and the role of the multi-disciplinary team in the prevention of all avoidable pressure ulcers.

KEY WORDS: pressure ulcer, prevention, best practice

ADDRESS FOR CORRESPONDENCE: Mark Collier
United Linkolnshire Hospitals NHS Trust
c/o Pilgrim Hospital
Sibsey Road
Boston, Linkolnshire, PE21 9QS
E-mail: mark.collier@ulh.nhs.uk

INTRODUCTION

In view of an increasingly ageing population and the associated demographic changes throughout Europe, it could be argued that the need for all clinicians to be both aware of the Aetiology of Pressure Ulceration and a variety of Prevention Techniques is paramount for the delivery of evidence based practice by all members of the multi-disciplinary team - for the benefit of all of our patients. This is in spite of the fact that there have been an increased number of articles/copy pages in journals and other publications that have been dedicated to issues relevant to the prevention of pressure ulceration, especially during the past decade. Previous prevalence studies undertaken throughout Europe (1) indicate that there is still much work to be undertaken and that pressure ulceration is a real problem both for patients and the healthcare systems in which those patients are being cared for (table 1).

Table 1.

Prevalence of pressure ulceration by county involved in study (ibid)	
Belgium	21%
Italy	8%
Portugal	12%
Sweden	23%
United Kingdom	22%

Note: A total of 5,947 patients were included in the study across five countries and identified percentages have been rounded to the nearest whole figure. It is also important to note that this study was a pilot project to test methods for measuring prevalence across healthcare boundaries.

More recently, it has been reported that at least 500,000 patients a year will develop at least one pressure ulcer whilst in the care of the National Health Service (NHS – United Kingdom) and that one in twenty patients (5%) who are admitted to hospital with an acute illness through an Accident and Emergency Department will also develop a pressure ulcer. This incidence figure rises to as much as 99% of patients admitted through Accident and

Emergency Departments who are acutely ill and who have been kept within the A&E department for over two hours prior to transfer to another clinical setting (2).

Furthermore the cost of managing a pressure ulcer and the effects on both the patient and healthcare setting can be summarised as follows;

- Additional treatment / management costs associated with a patient with one Category 4 Pressure Ulcer equals…up to £40,000 (€46,170) (3)
- from £1,214 (€1,402) Category 1 to £14,108 (€16,296) Cat 4 (4)

Additional 'hidden costs' that can be incurred by the healthcare system can be summarised as;

- Cost of treatment is 3.6 times more expensive than prevention (5) - think cost effectiveness before all of your actions/interventions!
- Additional resource burdens for Healthcare setting? Increased staffing and treatment costs / extended length of stay / dissatisfaction of patients / effects on staff morale.
- Increased incidence of Wound Infections -> Gangrene -> Septicaemia -> Amputation -> Death, resulting in increased pharmacological / product and surgical costs, as well as increased mortality and associated litigation costs!

The effects on a patient's Health Related Quality of Life (HRQoL) have been previously summarised as;

- Physical – a wound is present; it may be painful; it further/reduces mobility
- Psychological – cannot get my favourite shoes on; will it ever heal? when can I return to normal activities?
- Social – impairs social interaction (unable to access or due to associated symptomatology)
- Emotional – negative effects on wellbeing due to persistent pain
- Spiritual – affects access to long established groups important to affected persons such as Church (6).

A *pressure ulcer* can be defined as 'ulceration of the skin due to the prolonged effects of pressure in association with a number of other variables' (7) (8) and *prevalence* may be defined as 'the number of people/patients with a pressure ulcer as a proportion of the entire patient population, at a specific period of time' e.g. an identified day (9).

It could therefore be argued that whenever patients - in whatever healthcare setting - are being assessed as requiring a patient support surface with known pressure reducing or relieving properties, these should be chosen by an individual with both a knowledge of the patient's pressure ulcer 'at risk status' and the forces likely to be encountered by the patient's skin whilst on the same and the effects that these may have over time - as transmitted through tissues at various anatomical sites.

Pressure reduction is the constant reduction in the amount of external pressure being exerted on a patient's anatomy whilst at rest, whereas *pressure relief* is the intermittent lowering of the external pressure being exerted on the patient's anatomy (10).

PRESSURE DEFINED

Bennett and Lee defined pressure as a perpendicular load or force exerted on a unit of area such as the sacrum (11). This gravitational force is also often referred to as compression. The average pressure exerted on the skin can be calculated using the following formula:

$$\text{Pressure} = \frac{\text{Body weight}}{\text{Skin contact area}}$$

or by the use of pressure sensitive equipment (7).

In addition to the overall concept of pressure, other differing forms of pressure have also been highlighted - those of shear and friction.

Shear: (A stretching force). A mechanical stress that is parallel to a plane of interest (11). When a high level of shear is present, then the amount of external pressure necessary to produce vascular occlusion is only about half the amount when shear is not present (12). When trying to describe various clinical examples of shear, many authors have noted that when the head of the bed is elevated there is automatically a greater compressive force placed on the sacral tissues than when the bed is in the flat position (13)(14)(15). It is thought that the shear ulcer may typically develop as a result of the patient's sacral skin adhering to their bed linen (in the sitting position), the deep fascia moves in a downwards direction with the skeletal structure as a result of gravitational forces, whilst at the same time the sacral fascia remains attached to the sacral dermis. This effect can be minimised if the patient support surface is covered with a vapour permeable two-way stretch cover that helps to reduce moisture build up at the interface (16). However if the effects of shear are prolonged or exacerbated by the presence of moisture, regional stretching of the microcirculation of the skin may occur. If this is left unchecked it can lead to the avulsion of local capillaries and arterioles, increasing the possibility of the development of some localised tissue necrosis.

Although shear can be differentiated from that of pressure (compression), it has been previously highlighted that it is difficult to create pressure without shear and shear without pressure (17).

Friction: Friction occurs when two surfaces move across one another (18) for example when a patient under-

takes a sliding transfer from a bed to a wheelchair. Friction itself is not thought to be a primary factor in the development of pressure ulcers, however it is a contributory factor as this force can exacerbate the stripping of broken epidermis or be the cause of an initial break in the skin, which may then be further compounded by the effects of pressure and shear forces. If the surface on which the patient is being supported is moist, it has been previously highlighted that the friction coefficient will rise and if great enough will actually lead to adherence of the patient's skin to the damp surface (19) thereby resulting in an increase in any associated shearing effects.

In summary then, the physical parameters that must be considered when thinking of the aetiology of pressure ulcers are:

* Pressure / Compression
* Shear
* Friction
* Humidity of the patient's skin (may increase risk of adherence as previously described).

TRANSMISSION OF PRESSURE

Any external pressure measured at an interface will be transmitted from the body surface (the skin) to the underlying skeletal anatomy (the bone) compressing all of the intermediate tissues. The resultant pressure gradient has been described as the McClemont 'cone of pressure' (20), in which external pressures can increase by three to five times at the point of greatest pressure experienced, such as at a bony surface. For example, an external interface pressure of 50mmHG could rise to as much as 200mmHg at a bony prominence such as an ischial tuberosity.

With pressure being distributed in this way it should become apparent that any external skin blemishes/discolouration, however minor, identified as a result of the use of a pressure ulcer / wound classification tool such as those of Torrance (21) and Collier (22) amongst others (23), may be indicating that necrosis of the underlying tissue is already becoming established. It is therefore important that all practitioners are able to distinguish between a normal and abnormal physiological response and if the latter is suspected that they initiate the further assessment of the same with available technology such (24) (25).

THE NORMAL PHYSIOLOGICAL RESPONSE TO PRESSURE

The previous information should be considered in the light of research (26) that has shown that the pressure in the capillary bed in healthy medical student volunteers ranges between 12mmHg and 32mmHg (see figure 1). Landis in 1930 suggested that a value of 32mmHg was the mean capillary pressure at its arterial inflow - using a micro injection technique - and other studies suggested that if this pressure is exceeded then capillary occlusion occurs - predisposing to tissue damage (27)(28). However, in 1941 Landis revised his work - using an amended technique - identifying that a more realistic figure to be considered as the capillary closing pressure should be between 45mmHg to 50mmHg, over which threshold damage was likely to ensue. However, it should be remembered that any pressures measured may have different effects on different parts of the body depending on the local bone, muscle and skin structure.

This information becomes clinically relevant when interface pressures between the skin and the standard hospital mattress (a single block of foam) have been reported as between 70mmHg and 100mmHg over the main bony prominences and the interface pressure between the skin and a commercially available pressure reducing replacement mattresses (7) has been shown to be between 30mmHg and 40mmHg when measured on an 'average' individual lying in the supine position (29).

The capillary loops in the skin run vertically to the surface and are coiled at their bases, thereby limiting the risk of occlusion as a result of direct pressure. However, in the subcutaneous tissue, the blood vessels lie mainly in the parallel planes of the deep fascia and follow the paths of ligaments and nerves. This renders them very vulnerable to distortion and occlusion as a result of pressure from both external sources and the underlying bony structures (30). Prolonged pressure may cause ischaemic changes at and around the point of occlusion. If this occlusion is prolonged, the result is both anoxia and a build-up of circulating metabolites. A release of pressure however produces a large and sudden increase in blood flow, as the anoxia and metabolites act on structures within the circulatory system, such as pre-capillary sphincters. This increase in blood flow may be as much as 30 times the resting value and the bright red flush, which is often noted, is referred to as reactive or blanching hyperaemia (31) a normal response! As little as five seconds of external pressure can provoke a physiological reaction that may last between one third and three-quarters of the period of ischaemia (32). If the lymphatic vessels of the dependant tissue remain intact and excess interstitial fluid is removed, then it is said that permanent tissue changes will not progress (18). *Blanching hyperaemia* has been described as the distinct skin colour change caused by reactive hyperaemia which when light finger pressure is applied will blanch (further change colour – e.g. whiten), indicating that the patient's microcirculation is generally intact. *Non-blanching hyperaemia* - an ab-

normal physiological response – is detected when the discolouration of the skin remains when light finger pressure is applied, indicating a degree of microcirculatory disruption often associated with other clinical signs such as blistering, induration (alteration in texture of the skin) and oedema (21).

Note: The vessels in the subcutaneous tissues also give rise to the perforators that also supply the skin, and so deep vessel obstruction is likely to result in both cutaneous and subcutaneous ischaemia if the period of occlusion is sustained. The results of trancutaneous oxygen assessments have suggested that perfusion of the skin is affected more greatly by subcutaneous pressure than by external interface pressures only (33).

In order to accurately recognise both blanching and non-blanching hyperaemia, it is important for the assessing practitioner not only to fully understand the definition of a pressure ulcer as highlighted earlier, but also to understand the pathophysiology of reactive hyperaemia (34).

Although the fragile nature of the microcirculation has been acknowledged, this does not take account of the protective function of collagen. It appears that if tissue collagen levels are not depleted, this helps to prevent disruption to the micro- circulation by buffering the interstitial fluid from external pressures, thereby maintaining the optimum hydrostatic pressure.

FACTORS ENHANCING A CLIENT/PATIENTS RISK OF PRESSURE ULCER DEVELOPMENT

The latest published clinical guideline No.179 from NICE – Pressure Ulcer Prevention and Management, highlighted a number of risk factors that practitioners should be aware of (including their evidence base) when assessing an individuals risk of developing pressure ulcers, whether through an informal or formal assessment process (35). These risk factors included reduced acute illness; extremes of age; level of consciousness; malnutrition/dehydration; mobility or immobility; previous history of pressure damage; sensory impairment; severe chronic or terminal illness and vascular disease (see table 2).

The fragile nature of the microcirculation has been previously discussed. If this pathophysiology is clearly understood, practitioners will appreciate that pressure ulcers can develop as the result of two main processes - occlusion of the blood vessels because of external pressure and endothelial damage of arterioles and the micro- circulation due to the application of disruptive (compression) and shearing forces (36). Tissue changes will occur when occlusion is prolonged and external pressures result in damage to lymphatic vessels, leading to the squeezing out of interstitial fluid. This is important as if a sufficient volume leaves the interstitial space, cell to cell contact can occur, resulting in cell membrane rupture and the release of toxic intracellular materials (37).

Table 2. Physiological factors in the development of pressure ulcers and criteria for the selection of appropriate patient support surfaces (after 11).

Intrinsic factors:	*Selection criteria:*	*References:*
Acute illness Pyrexia Medication	Ensure that the support surface facilitates holistic care and that the effects of these factors are taken into consideration. Some areas of the body, such as the heels, may be more at risk of breakdown than others	21, 38, 39, 40, 41
Extremes of age	The choice of equipment to be considered will be dependent upon the nature of the patient's skin type and tolerance to pressure	42, 43
Level of consciousness	Ensure that the chosen support surface is able to manage the patient's body weight and facilitate appropriate care interventions	44, 45
Malnutrition/dehydration	Ensure that the additional features on the chosen support surface will not unintentially exacerbate the patients condition, for example any heating mechanism	
Mobility/ immobility	Assess the patient's ability to move in bed. Choose a support surface that will compensate for any loss of movement and allow access for moving and handling purposes	38, 46
Previous history of PU	Ensure that the support surface chosen facilitates enough pressure relief/reduction to minimise the risk of further tissue breakdown	
Sensory impairment	The choice of equipment to be considered will be dependent upon the presence of this factor	43
Chronic/ terminal illness	Ensure that the support surface chosen facilitates enough pressure relief/reduction to minimise the risk of further tissue breakdown	
Vascular disease	Ensure that the support surface can be profiled or adapted to take account of the patient's condition	
Extrinsic factors:		
Pressure/ shear/friction	Choose a support surface that is designed to alleviate the appropriate force(s), is comfortable for the patient and is covered by a vapour permeable two way stretch cover	7, 11, 12, 13, 17, 18, 20
Exacerbating factors:		
Bacterial contamination	Ensure that the support surface can and is decontaminated as per an approved protocol	
Skin moisture	Ensure that the cover of the support surface is easily cleaned and vapour permeable.	47, 48
Spinal shock	Special equipment may be required during the acute phase of patient management	
Sleep	Ensure that the support surface chosen maximises the patient's comfort and that any electrical equipment is as quiet as possible whilst in use	49, 50, 51

Although it is generally accepted that the use of appropriate patient support surfaces will assist with planned care interventions intended to prevent/minimise the risk of pressure ulcer development, it is also generally accepted that there is no support surface on

which a patient will not need to be repositioned on a regular/frequent basis and that if the need for a pressure reducing/relieving surface has been identified it needs to be available 24 hours a day (38)(35), for example when a patient is both in bed and or sitting out on a chair. This may necessitate the provision of pressure reducing/relieving cushions as well as mattresses. Patient turning regimes can never be absolute and should always be based on an individual assessment of risk (34). The frequency of repositioning should be determined by the results of skin inspection and individual needs, and should also take into consideration other relevant matters, such as the patients medical condition, their comfort, the overall plan of care and the current support surface on which they are being nursed (35).

RISK ASSESSMENT TOOLS: AN AIDE TO THE ASSESSMENT PROCESS?

Throughout Europe a variety of risk assessment tools are used - at least nineteen (52), although there is little evidence in the literature to suggest that one tool is superior to any other. However, it is generally accepted that the incorporation of a risk assessment tool in the formal assessment process will help to inform a practitioners next planned care intervention (53) (54). The tools most frequently observed in clinical use are those of Norton (55), Waterlow (56) and Braden (57).

Table 3. Some advantages and disadvantages of the three most frequently used tools.

Assessment Tool	Advantages	Disadvantages
Norton	Simple and easy to use. Has been used widely used in a number of different care settings.	Too simplistic, does not take account of the increasing complexity of the patients health status.
Waterlow	Assesses most risk factors considered relevant, that can be supported by the literature. Includes a five-stage ulcer classification tool, guidelines on the use of preventative equipment and a management system for wound care as well as the risk assessment tool. Most widely taught assessment tool in Nurse Education programmes - in particular throughout the United Kingdom.	Numerical scores derived as a result of the assessment process often tend to be on the high side. This has been observed to result in an increase in pressure on resources - especially when the score is being used in order to identify preventative equipment to be used. The sensitivity and specificity has been questioned (58).
Braden	This scale has demonstrated greater sensitivity and specificity than the majority of other assessment tools (59) (60). Widely used throughout the United States of America and Europe.	Difficult for practitioners to understand/ utilize when initially introduced to the same.

Most of the research currently available, in relation to pressure ulcer risk assessment tools, focuses on the sensitivity and specificity of the tools being analysed. *Sensitivity* may be defined as the ability of a risk assessment tool/scale to correctly identify those patients who will go on to develop a pressure ulcer and *specificity* can be defined as the ability of a pressure ulcer tool/scale to correctly identify those who will not develop a pressure ulcer. However, it should be remembered that the use of any preventative measures; the length of the observation period and the nature of the group of patients being studied will influence both sensitivity and specificity. It could therefore be argued that these measures alone should not be used to judge the effectiveness of a risk assessment tool.

By implication, the appropriate use of preventative measures for the management of patients assessed as at risk of pressure ulcer development should have the effect of lowering that individuals risk, whereas if preventative measures are not initiated or are stopped, this would have the effect of increasing an individuals risk status.

When practitioners are deciding which at risk tool to incorporate into their assessment plan they should consider a number of factors, of which the following are just a few.

Is the risk assessment tool?

- *Valid.* Face validity relates to the authors knowledge of the subject matter being assessed whereas content validity relates to the factors that the assessment tool considers in identifying a patients risk – these should be supported by the literature.
- *Reliable.* Relates to the consistency and accuracy of the tool in measurement. For example, would two practitioners using the same assessment tool for the same individual get similar or the same results.
- *Applicable to the patient group being assessed?*
- *A subjective or objective assessment tool.* It has previously been reported that, 'assessing risk involves making subjective decisions and assigning them a numerical value' (61).
- *User Friendly.* Is the assessment tool easy for the user to understand?
- *Useful.* Having completed the assessment process, does the use of the tool lead to an alteration in subsequent patient interventions? This should not simply be the choice of a particular piece of equipment based on a numerical score. *Note:* It is important to remember that some tools denote increased risk by descending scores e.g. Norton whereas others identify increased risk by ascending scores e.g. Waterlow.

NICE guidelines (35) reinforce the statements published by the Department of Health in relation to Pressure Ulcer Benchmarks (62) that suggest that assessing an individual's risk of developing a pressure ulcer should involve both informal and formal assessment procedures. That risk assessment should be carried out by personnel who have undergone appropriate training to recognise the risk factors that contribute to the development of pressure ulcers and know how to initiate and maintain correct and suitable preventative measures. That the timing of risk assessments should be based on each individual's case and that all formal assessments of risk should be documented / recorded and made accessible to all members of the multidisciplinary team. Finally, but most importantly, risk assessment tools should only be used as an aide memoire and should not replace clinical judgement (35).

SO HOW IS THE EUROPEAN WOUND MANAGEMENT ASSOCIATION (EWMA) HELPING TO PROMOTE PRESSURE ULCER PREVENTION?

EWMA and the European Pressure Ulcer Advisory Panel (EPUAP) have recently agreed to work together to establish a joint engagement for the promotion of Pressure Ulcer Prevention and Patient Safety agenda's at the European level, as well as at a national level in some selected European countries. This joint project has been approved by both executive committees/organisations and appropriate financial resources have been allocated for the same. A joint working group has been established and representatives from both organisations identified.

It has been agreed that initially an epidemiological overview (desk top) based on existing European data will be undertaken and National pressure ulcer prevalence and incidence data will be collected. After this, further actions will be agreed as applicable.

On completion of the project, it is anticipated that both EWMA and EPUAP will be able to report to relevant individuals/organisations/healthcare settings/providers information about current Pressure Ulcer prevention activities across Europe. It is also hoped that the project will stimulate increased collaboration between wound care experts and patient safety organisations in selected European countries. Continuous monitoring of EU policies and initiatives will take place, with the objective of placing PU prevention, patient safety and wound care on the European health care agenda. Finally, increased collaboration between two of the largest and most influential European wound care associations should be beneficial not only for their members but also for the patients that the members influence the care of/care for on a daily basis.

Visit the two organisations websites when you can for regular updates on progress of this important project – *see useful contacts below*.

REFERENCES

1. Clark M, Bours G, De Flour T. Summary report on the prevalence of pressure ulcers. EPUAP Review 2002; 4: 49-56.
2. RCEM. Quality Care for Older People with Urgent and Emergency Care Needs. Royal College of Emergency Medicine, 2012, UK.
3. Collier M. Pressure Ulcer prevention: fundamentals for practice.In: Pressure Ulcer Prevention: A guide to product selection. (Supp) J Wound Care Therapy Weekly 2004; May 3-7.
4. Dealey C, Posnett J, Walker A. The cost of pressure ulcers in the United Kingdom J Wound Care 2012; 21: 261-4.
5. Santamaria N, Liu W, Gertz M et al. The cost-benefit of using soft silicone multi-layered foam dressings to prevent sacral and heel pressure ulcers in trauma and critically ill patients. Int Wound J 2013; ISSN 1742 - 4801
6. Collier M, Franks P. Quality of life: the cost to the individual. Chapter in Morison M and Braden B, eds. The Prevention and Management of Pressure Sores. Edinbourgh: Harcourt Brace 2001.
7. Collier M. Pressure Reducing Mattresses. J Wound Care 1996; 5: 207-11.
8. EPUAP. Proceedings of the First European Pressure Ulcer Advisory Panel Meeting. European Pressure Ulcer Advisory Panel, Oxford, 1997.
9. Gallagher S (1997). Outcomes in clinical practice: pressure ulcer prevalence and incidence studies. Ostomy Wound Manag 1997; 43: 28-32.
10. Agency for Health Care Policy and Research. Treatment of Pressure Ulcers: Clinical Practice Guideline No.15. USA: AHCPR, 1994.
11. Bennett L, Lee B. Shear versus pressure as causative factors in skin blood flow occlusion. Arch Phys Med Rehab 1986; 60: 309-14.
12. Bennett L, Lee B. Pressure versus shear in pressure sore formation. In: Lee B, ed. Chronic Ulcers of the Skin. New York: McGraw Hill, 1985, 39-55.
13. Berecek K. Etiology of pressure sores. Nurs Clin North Am 1975; 10: 157.
14. Brown S. Nursing innovation for prevention of decubitus ulcers in long-term facilities. Plast Surg Nurse 1985; 5: 57.
15. Reichel SM. Shearing forces as a factor in decubitus ulcers in paraplegics. JAMA 1958; 166-172.
16. Collier M. Fundamental concepts. Resource File: Mattresses and Beds. 1- 8. London: EMAP, 1999.
17. Bridel J. The aetiology of pressure sores. J Wound Care 1993; 2: 230-238.
18. Krouskop T. Mechanisms of decubitus ulcer formation - a hypothesis. Med Hypothesis 1976; 4: 37-9.
19. Lothian P, Cowden J, Scales J. Underpads in the prevention of decubitus. In: Kenedi R, ed. Bedsore Biomechanics. USA: University Park Press, 1976.

20. McClemont E. Pressure Sores. Nursing 1984; Supp. 2.
21. Torrance C. Pressure Sores: Aetiology, Treatment and Prevention. London: Croom Helm, 1983.
22. Collier M. Assessing a wound - RCN Nursing Update Unit 29. Nursing Standard. 1994; 8(49) Supp. 3-8.
23. EPUAP. Guide to Pressure Ulcer Grading. EPUAP Rev.2001; 3: 75.
24. Collier M. Pressure ulcer development and principles for prevention. In: Glover D, Miller M, eds. Wound Management: theory and practice. NT Books, 1999.
25. Longport Incorporated Applications of Ultrasound Biomicroscopy in Wound Care. 2003. www.longportinc.com
26. Landis E. Microcirculation studies of capillary blood pressure in human skin. Heart. 1930; 15: 209-28.
27. Barton A, Barton M. The Management and Prevention of Pressure Sores. Faber, 1981.
28. Daniel R, Priest D,Wheatley D. Etiologic factors in pressure sores: an experimental model. Arch Phys Med Rehab 1981; 62: 492-8.
29. Scales J, Lowthian P, Poole A, Ludman W. Vaperm patient support system: a new general-purpose hospital mattress. Lancet 1982 2:50-2.
30. Bliss M . Aetiology of pressure sores. Clin Gerontol 1993; 3: 379-97.
31. Lamb J, Ingram C, Johnson T, Pitman R. Essentials of Physiology. London: Blackwell Scientific, 1980.
32. Lewis T, Grant R. Observations upon reactive hyperaemia in man. Heart 1925; 4: 37-9.
33. Sangeorzan B, Harrington R, Wyss C et al. Circulatory response of skin to loading. J Orthop Res 1989; 7: 425-31.
34. Collier M. Blanching and non-blanching hyperaemia. J Wound Care 1999; 8: 63-4.
35. National Institute for Clinical Excellence. Pressure Ulcer Prevention: Clinical Guideline No.179. London: NICE, 2014.
36. Barton A, Barton M. The Management and Prevention of Pressure Sores. Faber, 1981.
37. Krouskop M. A synthesis of the factors which contribute to pressure sore formation. Med. Hypothesis 1983;11: 255-67.
38. Collier M. Taking the pressure off: twenty-four hours a day. Huntleigh Healthcare Clinical Report. NHE. Luton, 1997..
39. Malone C. Intensive pressures. Nursing Times. 1992; 88: Suppl. 3-8.
40. McSorley P, Warren D. The effects of propanolol and metoprolol on the peripheral circulation. BMJ 1978; 2: 1598-1600.
41. Barrow T, Sikes C. Decubitus ulcers in rheumatic fever treated with cortisone. JAMA 1951; 147: 41-2.
42. Reswick J, Rogers I. Experience at Rancho Los Amigos Hospital with devices and techniques to prevent pressure sores. In: Kenedi R, Cowan J, Scales J,eds. Bedsore Biomechanics. University Park Press, 1976, 301-10.
43. Raney J. A comparison of the prevalence of pressure sores in hospitalised ALS and MS patients. Decubitus 1989; 2: 48-9.
44. Summer W, Curry P, Haponikm E, Nelson S, Elston R. Continuous mechanical turning of intensive care unit patient's shortens length of stay in some diagnostic-related groups. J Critical Care 1989; 4: 45-53.
45. Philips P. Obesity and weight reduction programmes. Geriatr Med 1981; 11: 53-57.
46. David J. The size of the problem of pressure. Care Sci Pract 1981; 1: 10-13.
47. Flam E. Skin maintenance in the bedridden patient. Ostomy Wound Management. 1990; May/June: 48-54.
48. Norton D, McLaren R, Exton Smith A. An Investigation of Geriatric Nursing Problems in Hospital. Edinburgh: Churchill Livingstone, 1975.
49. Torrance C. Sleep and wound healing. Surg Nurse. 1990: 3: 16-20.
50. Kelly M, Coverdale S, Williams S et al. Easing the pressure. Nursing Times 1995;. 91: 72-6.
51. Grindley A, Acres J. Alternating pressure mattresses: comfort and quality of sleep. Br J Nursing. 1996; 5: 1303-10.
52. EPUAP. Pressure Ulcer Risk Assessment: A draft EPUAP position statement. EPUAP Rev. 2001; 3: 46-52.
53. Goodridge D, Sloan J, LeDoyen Y, Mckenzie J, Knight W, Gayari M. Risk assessment scores, preventative strategies and the incidence of pressure ulcers among the elderly in four Canadian health care facilities. Can J Nursing Res 1998; 30: 23-44.
54. Collier M. NT essential guide to wound assessment. London: EMAP, 2001.
55. Norton D, McLaren R, Exton-Smith A). An investigation of Geriatric Nursing Problems in Hospital. Edinburgh: Churchill Livingstone, 1962.
56. Waterlow J. A risk assessment card. Nursing Times.1985; 81: 49-55.
57. Bergstrom N, Braden B, Laguzza A et al. The Braden Scale for predicting pressure sore risk. Nursing Res 1987; 36: 205-10.
58. Defloor T, Grypdonck M. Risk assessment scales, a critique. Proceedings of the 4th European Pressure Ulcer Advisory Panel Open Meeting, Pisa, 2000.
59. Braden B, Bergstrom N. Clinical utility of the Braden Scale for predicting tissue sore risk. Decubitus 1988; 2: 34-8.
60. Flanagan M. Predicting Pressure Sore Risk. J Wound Care 1993; 2: 162-7.
61. Scott E. The prevention of pressure ulcers through risk assessment. J Wound Care; 2000; 9: 69-70.
62. Do H. The Essence of Care. London: Department of Health, 2001.

Useful contacts:

- European Wound Management Association – www.ewma.org
- European Pressure Ulcer Advisory Panel – www.epuap.org.uk
- Tissue Viability Society – www.tvs.org.uk

SAŽETAK

PREVENCIJA TLAČNOG VRIJEDA: TEMELJI ZA NAJBOLJU PRAKSU

M. COLLIER

United Linkonshire Hospitals NHS Trust, c/o Pilgrim Hospital, Boston, Linkolnshire, UK

Na početku ovog rada naglašena je kompleksna etiologija razvoja tlačnog vrijeda i kompleksan proces promjene s namjerom da se prepoznaju oni pacijenti koji jesu ili bi mogli biti rizični za razvoj tlačnog vrijeda. Ova zadnja tvrdnja pretpostavlja da svi pacijenti koji su zbrinuti u bilo kojoj zdravstvenoj ustanovi mogu razviti tlačni vrijed. Smatra se da korištenje alata procjene može biti važno u jednoj općenitoj strategiji prevencije vrijeda. Važno je da se ograničenja tih alata uzmu u obzir i da oni nisu sami po sebi procjena. Njih treba koristiti praktičar s temeljnom širinom relevantnog znanja i uz raspoloživi raspon odgovarajućih ekipa/tehnika prevencije; naglašena je uloga multidisciplinarnog tima u prevenciji svih tlačnih vrijedova koji se mogu spriječiti.

KLJUČNE RIJEČI: tlačni vrijed, sprječavanje, osnove najbolje prakse