Evaluation of antidecubitus mattresses

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Abstract—*Pressure sores are a current problem in hospitals and care of the elderly, leading to protracted hospital stays and a high care burden. The trauma for the patients is severe, and the cost of pressure sore prevention and treatment, is considerable. Antidecubitus mattresses are used for prevention and in treatment, but they also contribute to the cost of treating pressure sores. The problem highlighted in the review is that the mattresses' effectiveness in preventing and treating pressure sores has not been sufficiently evaluated. When antidecubitus mattresses are evaluated, it is often only with regard to aspects of the interface pressure and the mattresses' ability to redistribute the pressure. The review points out the important observation that, to be able to evaluate the efficacy of the antidecubitus mattress, the mattress's effect on tissue viability needs to be studied. The parameters that ought to be considered when evaluating a support surface are: interface pressure, pressure and blood flow distribution, temperature and humidity in the skin-support surface interface. The authors propose that the effect on tissue viability of external loading can be assessed by simultaneous measurement of the interface pressure and tissue perfusion.*

Keywords—*Pressure sores, Tissue perfusion, Interface pressure, Antidecubitus mattress*

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1 Introduction

PRESSURE SORES are a current problem in hospitals and in elderly care facilities (BRIENZA *et al.*, 2001; LINDGREN *et al.*, 2004). Because of increased life expectancy and advances in medicine, the number of patients at risk of developing pressure sores is increasing. This results in protracted hospital stays and a high care burden.

A study from 2002 shows a cumulative, 5 month pressure sore incidence of 43% in two long-term facilities in the USA (LYDER *et al.*, 2002). Studies from medical and surgical wards in a health care area in Sweden, involving 530 patients between 1996 and 1998, revealed an incidence of 11.7%, including non-blanching hyperaemia (LINDGREN *et al.*, 2004). The cost of pressure sores is considerable. In a Swedish county, the cost of dressing changes for pressure sores, in respect only to nursing time, was equal to ten full-time nurses (LINDHOLM *et al.*, 1999). Pressure sores also involve great suffering for the patients affected (GIBSON, 2002; LANGEMO *et al.*, 2000).

Clinical strategies for pressure sore prevention include, for example, the use of risk assessment scales (BERGSTROM *et al.*, 1987; LINDGREN *et al.*, 2002; NORTON *et al.*, 1979), education of patients (LANGEMO *et al.*, 2000) and personnel (GUNNINGBERG *et al.*, 2000), good hygiene practice (LEVINE *et al.*, 1989), redistribution and relief of the forces acting on the tissue by change of position (GUNNINGBERG *et al.*, 2000; KOSIAK, 1959; LEVINE *et al.*, 1989) and the use of antidecubitus mattresses for prevention and in treatment (LINDGREN *et al.*, 2000).

This review summarises the factors that contribute to pressure sore formation and suggests which parameters should be measured when evaluating antidecubitus mattresses. Also, the principles of measurement techniques and problems related to the measurement systems and the test procedures are discussed.

2 Pressure sore aetiology

A pressure sore is defined as an area of tissue damage appearing after a prolonged period of ischaemia in the tissue (EK, 1987*a*; KOSIAK, 1959). There are several factors contributing to the formation of pressure sores. These factors can be divided into two groups, extrinsic and intrinsic. The extrinsic factors are externally applied pressure, shear forces and increases in surface temperature and humidity. The intrinsic factors consider the individual's condition: for example, grade of immobility and activity (LINDGREN *et al.*, 2004), age (BERGSTROM and BRADEN, 1992; GUNNINGBERG *et al.*, 2000; LINDGREN *et al.*, 2004), nutrient intake (CHRISTENSSON *et al.*, 1999; MARUM *et al.*, 2001), skin condition (EDSBERG *et al.*, 2001), loss of sensation (KETT and LEVINE, 1987), incontinence (EK and BOMAN, 1982; LYDER *et al.*, 2002), low blood

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pressure (BERGSTROM and BRADEN, 1992; LINDGREN *et al.*, 2004; SCHUBERT, 1991), general physical condition (EK, 1987*b*) and body constitution (ALLMAN *et al.*, 1995).

Young, healthy people react differently to external loading from geriatric or paralysed persons (CLARK and ROWLAND, 1989*a*; EK, 1987*a*; LINDAN, 1961), because of better coverage of muscle tissue that distributes the pressure more evenly (BERJIAN *et al.*, 1983; CLARK and ROWLAND, 1989*a*; MAKLEBUST *et al.*, 1986; NOLA and VISTNES, 1980; SACHSE *et al.*, 1998; SCHUBERT and FAGRELL, 1989). Normal, healthy tissue is more resistant to pressure-induced ischaemia and decreases in oxygenation than soft tissue in paraplegic individuals (DANIEL *et al.*, 1981; 1985; LIU *et al.*, 1999).

The mechanisms underlying the aetiology of pressure sores are not well understood. Most researchers agree that the primary cause of pressure sores is externally applied pressure, leading to tissue ischaemia (DINSDALE, 1973; ERIKSSON, 1980; LE *et al.*, 1984). There is no consensus on the tissue layer at which pressure sores start to develop. One theory is that the initial pathological changes occur in the deep muscle and progress upwards, called bottom-to-top sore formation (DANIEL *et al.*, 1981; SALCIDO *et al.*, 1994). This theory is supported by the fact that muscles are more susceptible to ischaemia than skin and subcutaneous tissue, as muscle tissue has a higher metabolism (DANIEL *et al.*, 1981; NOLA and VISTNES, 1980). The other theory is that the sore formation first occurs in the epidermis and upper dermis and then progresses downwards if the pressure is not relieved, i.e. top-to-bottom sore formation (BRIDEL, 1993; WITKOWSKI and PARISH, 1982). This theory has similarities with how the definitions of the different grades of pressure sores are built up (EPUAP, 1999).

Often, external pressure is considered to be the only external force acting on the tissue. However, the loading in the interface between the skin and support surface is a combination of normal and shear forces (BRIENZA *et al.*, 2001). Shear forces are always present when the surface and the external force are not perpendicular. Shear forces act parallel to the skin and exist owing to pressure and friction (BENNETT *et al.*, 1979). Dinsdale demonstrated that friction contributes to the formation of pressure sores through mechanical damage (DINSDALE, 1974). It has also been shown that shear forces affect the tissue perfusion by stretching the skin in relation to underlying structures and can even damage the vascular structure (BENNETT *et al.*, 1979; BENNETT and LEE, 1988; WYWIALOWSKI, 1999; ZHANG and ROBERTS, 1993). Others claim that shear forces exert their effect on the large vessels in the deeper tissue layers and cannot be seen when attempts are made to measure then at the skin surface (WITKOWSKI and PARISH, 1982).

It has been demonstrated that less external pressure is needed for blood flow occlusion when shear forces are present (BENNETT *et al.*, 1979). Also, a decrease in skin blood flow (ZHANG and ROBERTS, 1993) and oxygen tension (GOOSSENS *et al.*, 1994) can be seen with increased shear forces. With paraplegic and hospitalised elderly people, median shear forces about three times greater have been measured at the interface with the seat, compared with healthy individuals (BENNETT *et al.*, 1984). Some propose that the deformation of the tissue, rather than tissue ischaemia, is a contributing factor to pressure sore formation (ALLEN *et al.*, 1994; BRIENZA *et al.*, 2001; KETT and LEVINE, 1987).

Loading time and magnitude play a major role in pressure sore development (BENNETT and LEE, 1988; DANIEL *et al.*, 1981; 1985; DINSDALE, 1974; ERIKSSON, 1980; HUSAIN, 1953; KOSIAK, 1959; 1961; LINDAN, 1961). Studies have shown an inverse relationship between pressure and time with regard to tissue damage (DINSDALE, 1974; KOSIAK, 1959), but there is no consensus about at what pressure level and application time tissue damage will be caused. External pressure above the value of the capillary blood pressure could cause pressure sores (KOSIAK, 1959), but pressures lower than those causing capillary closure can also lead to tissue ischaemia (LE *et al.*, 1984; XAKELLIS *et al.*, 1991). Whether the external pressure applied will cause any damage depends on the individual's physiological condition (BERJIAN *et al.*, 1983; CLARK and ROWLAND, 1989*a*; DANIEL *et al.*, 1985; EK, 1987*b*; FRANTZ and XAKELLIS, 1989; FRANTZ *et al.*, 1993; RONDORF-KLYM and LANGEMO, 1993) and the body part and type of tissue structure that is subject to the external loading (CLARK and ROWLAND, 1989*a*; EK *et al.*, 1987; LE *et al.*, 1984; SCHUBERT and FAGRELL, 1989). In healthy individuals, an external pressure of at least 120 mm Hg is required for blood flow occlusion, compared with 11–30 mm Hg in geriatric hospitalised patients (BENNETT *et al.*, 1981; EK *et al.*, 1987).

Because of the tissue's inhomogeneous structure, external loading creates different levels of pressure inside the tissue and therefore blood flow in the tissue will be affected to varying degrees. The pressure at the skin could be lower than the capillary closing pressure, whereas the pressure near the bony prominences could be as high as five times the externally applied pressure (LE *et al.*, 1984) and thus enough to cause ischaemia. This could be why tissue over bony prominences is especially prone to pressure sore development. Individuals' different physiological conditions and the unpredictable effect on the inner tissue make it difficult to stipulate a safe threshold interface pressure level (BADER and GANT, 1988; CLARK and ROWLAND, 1989*b*; FLETCHER, 2001; MCLEOD, 1997).

When external forces are present, the blood flow will decrease, as the vascular network is compressed, which will affect the tissue oxygenation and nutrition and waste product transportation. There is no simple relationship between interface pressure and skin blood flow perfusion (RITHALIA and GONSALKORALE, 2000) or oxygen tension (FELDMAN *et al.*, 1993; XAKELLIS *et al.*, 1993). Therefore interface pressure measurements alone are not sufficient as a sign of tissue status (BRIENZA and GEYER, 2000; MAYROVITZ and SIMS, 2002; WHITTEMORE, 1998). External pressure can also lead to poor lymph flow (GUNTHER and CLARK, 2000; HUSAIN, 1953; KROUSKOP *et al.*, 1978; MILLER and SEALE, 1981) and interstitial fluid flow (REDDY *et al.*, 1981*a*; *b*), contributing to pressure sore formation.

In healthy individuals, irrespective of their age, the dermal blood flow increases when low levels of external pressure are applied (FRANTZ and XAKELLIS, 1989; HERRMAN *et al.*, 1999; PATEL *et al.*, 1999; XAKELLIS *et al.*, 1993), whereas the response pattern for elderly patients at risk of pressure sores is inconsistent (FRANTZ *et al.*, 1993). Upon relief of pressure, debilitated individuals demonstrate an impaired and delayed tissue recovery compared with healthy individuals (BADER, 1990). It has been shown that patients who increased their skin blood flow during surgery did not develop pressure sores, whereas those in whom blood flow decreased to below pre-operative level during surgery developed pressure sores. The patients who did not develop pressure sores also showed greater reactive hyperaemia (SANADA *et al.*, 1997).

A general, higher body temperature is often seen in individuals who develop pressure sores (BERGSTROM and BRADEN, 1992). Some elderly patients also demonstrate an impaired ability to increase skin blood flow in response to a thermal stimulus, compared with healthy persons of the same age (EK *et al.*, 1984). This impairment in the peripheral circulation could increase the risk of development of pressure sores. When a subject is lying upon a mattress or sitting in a wheelchair, the skin temperature could increase or decrease, depending on which type of support surface is used (COCHRAN and

PALMIERI, 1980; NICHOLSON *et al.*, 1999; STEWART *et al.*, 1980). A tissue temperature increase of 1°C gives an approximately 12% increase in metabolism (GUYTON, 1996), meaning that the tissue will need more nutrition and oxygen, therefore requiring an increased blood flow.

The relative humidity in the skin–support surface interface could be raised (COCHRAN and PALMIERI, 1980; STEWART *et al.*, 1980) owing to perspiration, urine and faeces. Moderate moisture increases the skin friction, whereas a high degree of moisture decreases the friction (SULZBERGER *et al.*, 1966), but makes the skin more sensitive to damage from rubbing (WYWIALOWSKI, 1999). Prolonged skin wetness increases the vulnerability to pressure-induced blood flow reduction (MAYROVITZ and SIMS, 2001). This effect appears to be caused mainly by the wetness, but urine could aggravate the effect. Thus the support surface has an important role in the dissipation of heat and moisture away from the skin–surface interface, to maintain an acceptable microclimate (NICHOLSON *et al.*, 1999). Using plastic covers to protect mattresses in hospital is common practice; however, the plastic cover limits the mattress's ability to maintain a good microclimate.

To summarise this Section, the extrinsic parameters we consider most essential to measure when evaluating antidecubitus mattresses are interface pressure in combination with blood flow and microclimate. Ideally, these parameters ought to be measured in conceivable users, i.e. individuals prone to pressure sore formation. It is also important to take into consideration the individual conditions that differ between subjects.

3 Parameters to identify and evaluate

3.1 *Interface pressure and shear forces*

Interface pressure is determined by body weight and the size of the body's contact area with the underlying surface. Tissue compliance has an effect on the size of the contact area (ALLEN *et al.*, 1993*b*). Pressure measurement can be made with a single pressure sensor or with a pressure sensitive mat. The types of pressure sensor used for studying the aetiology of pressure sores and evaluation of mattresses are: electro-pneumatic (MAKLEBUST *et al.*, 1986), strain gauges (BENNETT *et al.*, 1979) and load cells (EK *et al.*, 1987). Commercial pressure-sensitive mats use piezoresistive or capacitive measuring principles. Shear forces have been measured with strain gauges (GOOSSENS *et al.*, 1993) and using a capacitive sensor principle (GOOSSENS *et al.*, 1997), but, as shear forces are dependent upon pressure to exist, the possibility of measuring shear force alone without the influence of pressure can be discussed.

Several measurement points are needed, as the pressure over a small area can be highly variable (LE *et al.*, 1984), and low interface pressure at one anatomical site does not exclude a much higher interface pressure at other sites (ALLEN *et al.*, 1993*b*; 1994; CLARK, 1994). If a single sensor is used, it has to be moved between measurements to obtain the pressure levels under several bony prominences, which could physiologically stress the tissue and the person. In addition there is always a risk of the sensor being misplaced before the measurement. A pressure-sensitive mat solves these problems and visualises the pressure distribution, but has the disadvantage of limited accuracy (HOCHMANN *et al.*, 2002).

In mattress evaluation, it is important that the performance of the mattress is not affected by the measuring system (BENNETT *et al.*, 1984; BLISS, 1993; FERGUSON-PELL and CARDI, 1993; HOLLEY *et al.*, 1979). It has been shown that the mattress characteristics and the skin–support surface interface affect the accuracy of the interface pressure measurement (ALLEN *et al.*, 1993*a*; REDDY *et al.*, 1984). The size (FERGUSON-PELL and CARDI, 1993; KROUSKOP and GARBER, 1990; REDDY *et al.*, 1984), frictional properties and flexibility of the sensor affect the pressure distribution (FERGUSON-PELL and CARDI, 1993) and thereby the result. Thus a pressure-sensitive mat is preferable, as it is more flexible and therefore affects the measurements less than a rigid pressure sensor.

3.2 *Peripheral blood flow and oxygenation*

By measuring tissue blood flow or tissue oxygenation, the physiological effect of externally applied pressure on the tissue can be assessed (BADER, 1990; BADER and GANT, 1988; FRANTZ *et al.*, 1993; GOOSSENS *et al.*, 1994; JACOBS, 1989; MAYROVITZ and SIMS, 2002; NEWSON *et al.*, 1981; RITHALIA and GONSALKORALE, 2000; SEILER and STÄHELIN, 1979). Methods of measuring tissue blood flow are laser Doppler flowmetry, laser Doppler imaging and photoplethysmography. Transcutaneous oxymetry, radioisotope clearance and video microscopy have also been used to study tissue viability, see Table 1.

Through use of different wavelengths of light and different distances between the light source and detector, laser Doppler methods and photoplethysmography can reflect the blood flow at different tissue depths. In general, laser Doppler flowmetry and imaging can penetrate the tissue to an approximate depth of 235 μm (JAKOBSSON and NILSSON, 1993; WÅRDELL *et al.*, 1993). Thus laser Doppler reflects the blood flow from superficial tissue structures. A somewhat greater tissue depth is achieved with photoplethysmography. Studies indicate

Table 1 Methods used to study effect of external loading and unloading in tissue

Method	References
Transcutaneous oxymetry	BADER, 1990; BADER and GANT, 1988; BALDWIN, 2000; FELDMAN *et al.*, 1993; GOOSSENS *et al.*, 1994; LIU *et al.*, 1999; NEWSON *et al.*, 1981; RITHALIA *et al.*, 2000; RITHALIA and GONSALKORALE, 2000; SACHSE *et al.*, 1998; SANADA *et al.*, 1995; XAKELLIS *et al.*, 1991
Laser Doppler flowmetry and velocitometer	EK *et al.*, 1987; 1984; FELDMAN *et al.*, 1993; FRANTZ and XAKELLIS, 1989; FRANTZ *et al.*, 1993; HERRMAN *et al.*, 1999; JACOBS, 1989; MAYROVITZ and SIMS, 2002; MAYROVITZ *et al.*, 2003; MAYROVITZ *et al.*, 1997; MEINDERS *et al.*, 1996; PATEL *et al.*, 1999; SACHSE *et al.*, 1998; SANTOS *et al.*, 2003; SCHUBERT, 1991; SCHUBERT and FAGRELL, 1989; SCHUBERT and HÉRAUD, 1994; SCHUBERT *et al.*, 1995; XAKELLIS *et al.*, 1991; 1993
Laser Doppler imager	MAYROVITZ *et al.*, 1999; MAYROVITZ and SMITH, 1998; MAYROVITZ *et al.*, 1997; THORFINN *et al.*, 2002
Photoplethysmography	BENNETT *et al.*, 1979; 1981; 1984; LEE *et al.*, 1979; MURRAY and MARJANOVIC, 1997
Video microscopy	NIITSUMA *et al.*, 2003
Radioactive isotope clearance	BOLUND and HOLSTEIN, 1976; DINSDALE, 1974; KLEMP *et al.*, 1983; MILLER and SEALE, 1981

that muscle blood flow from muscles at an average depth of 5.4 mm can be measured by photoplethysmography (SANDBERG *et al.*, 2005). Through the use of three different wavelengths in photoplethysmography, significant differences in occlusion pressure have been shown between individuals and from different tissue depths (MURRAY and MARJANOVIC, 1997).

However, attempts have been made to use new algorithms and near-infrared light in the laser Doppler technique to allow measurements from a greater tissue depth (BINZONI *et al.*, 2003). Transcutaneous oxymetry measures the condition of the tissue at a depth of about 100–300 μm (FELDMAN *et al.*, 1993). It is only possible to use video microscopy on thin tissue structures, e.g. an ear lobe (NIITSUMA *et al.*, 2003), which limits the use of this method. Radioactive isotope clearance is an invasive method that involves injections of radioactive substances and access to a gamma camera for imaging.

Significant spatial blood flow differences in tissue with apparently homogenous perfusion have been demonstrated (TENLAND *et al.*, 1983; WÅRDELL *et al.*, 1994) and correlate well with the tissue's microvascular architecture (BRAVERMAN *et al.*, 1990). Because of this spatial variation and problems with the position of the probe, it is difficult to draw any conclusions about perfusion from a single site (WÅRDELL *et al.*, 1994). Therefore it is preferable to use technologies that assess the spatial variation in blood flow when evaluating antidecubitus mattresses. With current equipment for measuring tissue perfusion, simultaneous measurement in tissue under external loading and investigation of the spatial variation cannot be achieved. With laser Doppler flowmetry, photoplethysmography and standard transcutaneous oxymetry, blood flow or tissue oxygenation can only be assessed at a single point or at a few points. To assess the spatial variation in perfusion as well, a laser Doppler imager can be used. This technology, however, has the disadvantage that it needs free access to the tissue (WÅRDELL *et al.*, 1993) and therefore cannot be used to evaluate support surfaces.

The laser Doppler flowmeter is very sensitive to movement of the laser Doppler probe or tissue motion relative to the probe (SHEPHERD and ÖBERG, 1990). The lack of a physiological zero can be an issue with laser Doppler when measuring low blood flow velocities that could be present under externally applied pressure (SACKS *et al.*, 1988).

Despite this, for the purpose of studying tissue viability under external loading, laser Doppler and photoplethysmography seem to be more suitable methods than oxygen tension, as they directly reflect the blood perfusion. In addition, laser Doppler and photoplethysmography involve measurement at a greater depth and are less hazardous compared with transcutaneous oxygen tension, which requires heating of the skin (RITHALIA *et al.*, 2000).

3.3 *Temperature and humidity*

Different types of support surface have different capacities for handling heat and humidity at the skin–support surface interface, making the evaluation of these parameters important (STEWART *et al.*, 1980). The support surface performance in maintaining a good microclimate can be studied with subjects on the mattress (STEWART *et al.*, 1980) or with help of a system exposing the mattress to environmental changes (NICHOLSON *et al.*, 1999). When measuring temperature and humidity, one or a few measurement points in the skin–support surface interface and one control site for skin temperature and environmental conditions are preferred (STEWART *et al.*, 1980). The temperature can be measured with a thermocouple (MAYROVITZ and SIMS, 2001; STEWART *et al.*, 1980) or thermistor (EK *et al.*, 1984; FISHER *et al.*, 1978). One study, which evaluated relative humidity, used both an electro-humidity sensor and a chemical sensor (STEWART *et al.*, 1980).

4 Discussion

The challenge in evaluating antidecubitus mattresses is to assess tissue viability during external loading. This is vital information for health care personnel in order for them to purchase or choose a suitable and effective mattress for patients with, or at risk of developing, pressure sores. Information about mattress efficiency could also contribute to greater cost-effectiveness within the public health service.

Antidecubitus mattresses are used both for prevention and in treatment. A mattress for an individual is chosen according to the person's weight and, when used in treatment, also according to the severity of the pressure sore. For example, the air pressure in alternating mattresses is determined from the person's weight, although individuals with equal weight might not tolerate the same pressure on the tissue. When deciding which mattress to purchase, several more properties have to be considered, e.g. comfort, durability, flammability, static electricity and cost-effectiveness (CLARK and ROWLAND, 1989*b*; JAY, 1995).

Today, the evaluation of antidecubitus mattresses is often performed by suppliers and is mostly based on interface pressure, as there are no suitable methods for evaluating the mattress's influence on the patient's tissue (RITHALIA and KENNEY, 2000). In addition, the evaluations are also mostly performed with healthy individuals. With increased knowledge of the effects on tissue of mechanical loading, the efficiency of different support surfaces can be evaluated (MAYROVITZ and SIMS, 2002). This would also facilitate the development of new antidecubitus mattresses (RITHALIA and KENNEY, 2000).

The few evaluations that have been performed have not been standardised with regard to any aspect. Standardisation is needed with regard to the measurement system (BRIENZA *et al.*, 2001; FERGUSON-PELL and CARDI, 1993; KROUSKOP and GARBER, 1990; WHITTEMORE, 1998), test procedure (CLARK, 1994) and the subject's position (BRIENZA *et al.*, 2001). Through the use of standardised procedures, comparisons of different measurement results could be achieved (KROUSKOP and RIJSWIJK, 1995), the problem of biased evaluations would be solved, test results would be strengthened, and the selection of an appropriate mattress would be facilited (COCHRAN and PALMIERI, 1980; FERGUSON-PELL and CARDI, 1993; KROUSKOP and RIJSWIJK, 1995). An extended, and to some extent standardised, evaluation of an antidecubitus mattress's efficiency ought to be performed for mattresses already on the market. Of course, the optimum solution for each individual at risk of developing pressure sores would be individual testing using the different mattresses available.

This review has pointed out that several parameters play a role in pressure sore formation. We consider it to be important to measure four of them when evaluating antidecubitus mattresses: interface pressure, blood flow, temperature and humidity in the skin–surface interface. However, there is a fifth factor of importance that ought to be regarded as a parameter in pressure sore formation, i.e. shear forces. It is difficult to separate the shear forces from other forces, and thus it is complicated to measure the shear forces alone. With the known measurement techniques, only tensile forces in the upper tissue are measured, and the true shear forces emanating from deeper within the tissue cannot be assessed.

Owing to the great variation in individuals' tissue viability, as well as their varying responses to external loading, it is important to use test subjects who are prone to developing pressure sores when evaluating antidecubitus mattresses. For the same reason, the rationale for measuring pressure only, in the search for a threshold level for blood flow occlusion, ought to be questioned. Assessing the blood flow in the tissue under loading is a better way of evaluating mattresses. Because of the large spatial variations in blood flow between adjacent, to

all appearances homogenous, areas, it is preferable to measure the variation in tissue perfusion over a greater area. To understand further the formation of pressure sores and assess the effect on the tissue in response to external loading, measurement of blood flow at different tissue depths is of interest.

5 Conclusions

This review shows the limitation of only measuring interface pressure when evaluating antidecubitus mattresses. To evaluate the effect of different mattresses on tissue viability thoroughly, the authors propose assessment of a multitude of parameters. Interface pressure distribution, blood flow distribution, temperature and humidity in the skin–support surface interface are all essential parameters. The interface pressure and blood flow distribution should be measured simultaneously, and the blood flow sensor should allow measurement at different tissue depths.

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References

ALLEN, V., RYAN, D. W., LOMAX, N., and MURRAY, A. (1993*a*): 'Accuracy of interface pressure measurement systems', *J. Biomed. Eng.*, **15**, pp. 344–348

ALLEN, V., RYAN, D. W., and MURRAY, A. (1993*b*): 'Repeatability of subject/bed interface pressure measurements', *J. Biomed. Eng.*, **15**, pp. 329–332

ALLEN, V., RYAN, D. W., and MURRAY, A. (1994): 'Measurements of interface pressure between body sites and the surfaces of four specialised air mattresses', *Br. J. Clin. Pharmacol.*, **48**, pp. 125–129

ALLMAN, R. M., GOODE, P. S., PATRICK, M. M., BURST, N., and BARTOLUCCI, A. A. (1995): 'Pressure ulcer risk factors among hospitalized patients with activity limitation', *JAMA*, **273**, pp. 865–870

BADER, D. L., and GANT, C. A. (1988): 'Changes in transcutaneous oxygen tension as a result of prolonged pressures at the sacrum', *Clin. Phys. Physiol. Meas.*, **9**, pp. 33–40

BADER, D. L. (1990): 'The recovery characteristics of soft tissues following repeated loading', *J. Rehabil. Res. Dev.*, **27**, pp. 141–150

BALDWIN, K. M. (2000): 'Transcutaneous oximetry and skin surface temperature as objective measures of pressure ulcer risk', *Adv. Skin Wound Care*, **14**, pp. 26–31

BENNETT, L., KAVNER, D., LEE, B. K., and TRAINOR, F. A. (1979): 'Shear vs pressure as causative factors in skin blood flow occlusion', *Arch. Phys. Med. Rehabil.*, **60**, pp. 309–314

BENNETT, L., KAVNER, D., LEE, B. Y., TRAINOR, F. S., and LEWIS, J. M. (1981): 'Skin blood flow in seated geriatric patients', *Arch. Phys. Med. Rehabil.*, **62**, pp. 392–398

BENNETT, L., KAVNER, D., LEE, B. Y., TRAINOR, F. S., and LEWIS, J. M. (1984): 'Skin stress and blood flow in sitting paraplegic patients', *Arch. Phys. Med. Rehabil.*, **65**, pp. 186–190

BENNETT, L., and LEE, B. Y. (1988): 'Vertical shear existence in animal pressure threshold experiments', *Decubitus*, **1**, pp. 18–24

BERGSTROM, N., BRADEN, B. J., LAGUZZA, A., and HOLMAN, V. (1987): 'The Braden scale for predicting pressure sore risk', *Nursing Res.*, **36**, pp. 205–210

BERGSTROM, N., and BRADEN, B. (1992): 'A prospective study of pressure sore risk among institutionalized elderly', *J. Am. Geriatr. Soc.*, **40**, pp. 747–758

BERJIAN, R. A., DOUGLASS, H. O., HOLOKE, E. D., GOODWIN, P. M., and PRIORE, R. L. (1983): 'Skin pressure measurements on various mattress surfaces in cancer patients', *Am. J. Phys. Med.*, **62**, pp. 217–226

BINZONI, T., LEUNG, T. S., BOGGETT, D., and DELPY, D. (2003): 'Non-invasive laser Doppler perfusion measurements of larger tissue volumes and human skeletal muscle blood RMS velocity', *Phys. Med. Biol.*, **48**, pp. 2527–2549

BLISS, M. R. (1993): 'Aetiology of pressure sores', *Rev. Clin. Gerontol.*, **3**, pp. 379–397

BRAVERMAN, I. M., KEH, A., and GOLDMINZ, D. (1990): 'Correlation of laser Doppler wave patterns with underlying microvascular anatomy', *J Invest Dermatol.*, **95**, pp. 283–286

BRIDEL, J. (1993): 'The aetiology of pressure sores', *J. Wound Care*, **2**, pp. 230–238

BRIENZA, D. M., and GEYER, M. J. (2000): 'Understanding support surface technologies', *Adv. Skin Wound Care*, **13**, pp. 237–243

BRIENZA, D. M., GEYER, M. J., KARG, P., and JAN, Y.-K. (2001): 'State of the science white paper on tissue integrity management'. *A State of the Science Conf. on Seating Issues for Persons with Disabilities*, Orlando, Florida, US

CHRISTENSSON, L., UNOSSON, M., and EK, A.-C. (1999): 'Malnutrition in elderly people newly admitted to a community resident home', *J. Nutr. Health Aging*, **3**, pp. 133–139

CLARK, M., and ROWLAND, L. B. (1989*a*): 'Comparison of contact pressures measured at the sacrum of young and elderly subjects', *J. Biomed. Eng.*, **11**, pp. 197–199

CLARK, M., and ROWLAND, L. B. (1989*b*): 'Preventing pressure sores: Matching patient and mattress using interface pressure measurements', *Decubitus*, **2**, pp. 34–39

CLARK, M. (1994): 'Problems associated with the measurement of interface (or contact) pressure', *J. Tissue Viability*, **4**, pp. 37–42

COCHRAN, G., VAN, B., and PALMIERI, V. (1980): 'Development of test methods for evaluation of wheelchair cushions', *Bull. Prosthet. Res.*, **17**, pp. 9–30

DANIEL, R. K., PRIEST, D. L., and WHEATLEY, D. C. (1981): 'Etiologic factors in pressure sores: An experimental model', *Arch. Phys. Med. Rehabil.*, **62**, pp. 492–498

DANIEL, R. K., WHEATLEY, D., and PRIEST, D. (1985): 'Pressure sores and paraplegia: An experimental model', *Ann. Plast. Surg.*, **51**, pp. 41–49

DINSDALE, S. M. (1973): 'Decubitus ulcers in swine: Light and electron microscopy study of pathogenesis', *Arch. Phys. Med. Rehabil.*, **54**, pp. 51–56

DINSDALE, S. M. (1974): 'Decubitus ulcers: Role of pressure and friction in causation', *Arch. Phys. Med. Rehabil.*, **55**, pp. 147–152

EDSBERG, L. E., NATIELLA, J. R., BAIER, R. E., and EARLE, J. (2001): 'Microstructural characteristics of human skin subjected to static versus cyclic pressures', *J Rehabil. Res. Dev.*, **38**

EK, A.-C., and BOMAN, G. (1982): 'A descriptive study of pressure sores: the prevalence of pressure sores and the characteristics of patients', *J. Adv. Nurs.*, **7**, pp. 51–57

EK, A.-C., LEWIS, D. H., ZETTERQVIST, H., and SVENSSON, P.-G. (1984): 'Skin blood flow in an area at risk for pressure sore', *Scand. J. Rehabil. Med.*, **16**, pp. 85–89

EK, A.-C. (1987*a*): 'Prevention, treatment and healing of pressure sores in long-term care patients', *Scand. J. Caring Sci.*, **1**, pp. 7–13

EK, A.-C. (1987*b*): 'Prediction of pressure sore development', *Scand. J. Caring Sci.*, **1**, pp. 77–84

EK, A.-C., GUSTAVSSON, G., and LEWIS, D. H. (1987): 'Skin blood flow in relation to external pressure and temperature in the supine position on a standard hospital mattress', *Scand. J. Rehabil. Med.*, **19**, pp. 121–126

EPUAP (1999): 'European Pressure Ulcer Advisory Panel Guidelines on treatment of pressure ulcers', *Epuap Rev.* **1**, pp. 31–33

ERIKSSON, E. (1980): 'Etiology: microcirculatory effects of pressure', in CONSTANTIAN, M. B. (Ed.): 'Pressure ulcers: principles and techniques of management' (Little Brown & Co., 1980), pp. 7–14

FELDMAN, D. L., SEPKA, R. S., and KLITZMAN, B. (1993): 'Tissue oxygenation and blood flow on specialized and conventional hospital beds', *Ann. Plast. Surg.*, **30**, pp. 441–444

FERGUSON-PELL, M., and CARDI, M. D. (1993): 'Prototype development and comparative evaluation of wheelchair pressure mapping system', *Assist. Technol.*, **5**, pp. 78–91

FISHER, S. V., SZYMKE, T. E., APTE, S. Y., and KOSIAK, M. (1978): 'Wheelchair cushion effect on skin temperature', *Arch. Phys. Med. Rehabil.*, **59**, pp. 68–72

FLETCHER, J. (2001): 'What can we learn from interface pressure measurements?' *J. Wound Care*, **10**, pp. 29–32

FRANTZ, R., and XAKELLIS, G. C. (1989): 'Characteristics of skin blood flow over the trochanter under constant, prolonged pressure', *Am. J. Phys. Med. Rehabil.*, **68**, pp. 272–276

FRANTZ, R., XAKELLIS, G. C., and ARTEAGA, M. (1993): 'The effects of prolonged pressure on skin blood flow in elderly patients at risk for pressure ulcers', *Decubitus*, **6**, pp. 16–20

GIBSON, L. (2002): 'Perceptions of pressure ulcers among young men with a spinal injury', *Br. J. Commun. Nurs.*, **7**, pp. 451–60

GOOSSENS, R. H. M., SNIJDERS, C. J., DIJKE, G. A. H. V., and OUDEN, A. H. D. (1993): 'A new instrument for the measurement of forces on beds and seats', *J. Biomed. Eng.*, **15**, pp. 409–412

GOOSSENS, R. H. M., ZEGERS, R., DIJKE, G. A. H. V., and SNIJDERS, C. J. (1994): 'Influence of shear on skin oxygen tension', *Clin. Physiol.*, **14**, pp. 111–118

GOOSSENS, R. H. M., SNIJDERS, C. J., HOLSCHER, T. G., HEERENS, W. C., and HOLMAN, A. E. (1997): 'Shear stress measured on beds and wheelchairs', *Scand. J. Rehabil. Med.*, **29**, pp. 131–136

GUNNINGBERG, L., LINDHOLM, C., CARLSSON, M., and SJÖDÉN, P.-O. (2000): 'The development of pressure ulcers in patients with hip fractures: inadequate nursing documentation is still a problem', *J. Adv. Nurs.*, **31**, pp. 1155–1164

GUNTHER, R. A., and CLARK, M. (2000): 'The effect of a dynamic pressure-redistributing bed support surface upon systemic lymph flow and composition', *Suppl. J. Tissue Viabil.*, **10**, pp. 10–15

GUYTON, A. C. (1996): 'Energetics and metabolic rate', in GUYTON, A. C. (Ed.): 'Textbook of medical physiology' (W. B. Saunders Company, Philadelphia, US, 1996), p. 908

HERRMAN, E. C., KNAPP, C. F., DONOFRIO, J. C., and SALCIDO, R. (1999): 'Skin perfusion responses to surface pressure-induced ischemia: Implication for the developing pressure ulcer', *J. Rehabil. Res. Dev.*, **36**, pp. 0–0

HOCHMANN, D., DIESING, P., and BOENICK, U. (2002): 'Evaluierung der messmethoden zur bewertung des therapeutischen nutzens von antidekubitus-systemen', *Biomed. Tech.*, **47**, pp. 816–819

HOLLEY, L. K., LONG, J., STEWART, J., and JONES, R. F. (1979): 'A new pressure measuring system for cushions and beds – with a review of the literature', *Paraplegia*, **17**, pp. 461–474

HUSAIN, T. (1953): 'An experimental study of some pressure effects on tissue with reference to the bed-sore problem', *J. Pathol. Bacteriol.*, **33**, p. 347

JACOBS, M. A. (1989): 'Comparison of capillary blood flow using a regular hospital bed mattress, ROHO® mattress, and Mediscus® bed', *Rehabil. Nurs.*, **14**, pp. 270–272

JAKOBSSON, A., and NILSSON, G. E. (1993): 'Prediction of sampling depth and photon pathlength in laser Doppler flowmetry', *Med. Biol. Eng. Comput.*, **31**, pp. 301–307

JAY, R. (1995): 'Pressure and shear: Their effects on support surface choice', *Ostomy/Wound Manag.*, **41**, pp. 36–45

KETT, R. L., and LEVINE, S. P. (1987): 'A dynamic model of tissue deflection in a seated individual'. *Resna 10th Ann. Conf.*, San Jose, California, US, pp. 524–526

KOSIAK, M. (1959): 'Etiology and pathology of ischemic ulcers', *Arch. Phys. Med. Rehabil.*, pp. 62–69

KOSIAK, M. (1961): 'Etiology of decubitus ulcers', *Arch. Phys. Med. Rehabil.*, pp. 19–29

KROUSKOP, T. A., REDDY, N. P., SPENCER, W. A., and SECOR, J. W. (1978): 'Mechanisms of decubitus ulcer formation – An hypothesis', *Med. Hypotheses*, **4**, pp. 37–39

KROUSKOP, T. A., and GARBER, S. L. (1990): 'Interface pressure measurements', *J. Enterostomal Ther.*, **17**, p. 182

KROUSKOP, T. A., and RIJSWIJK, L. V. (1995): 'Standardizing performance-based criteria for support surfaces', *Ostomy/Wound Management*, **41**, pp. 34–45

LANGEMO, D. K., MELLAND, H., HANSON, D., OLSON, B., and HUNTER, S. (2000): 'The lived experience of having a pressure ulcer: A qualitative analysis', *Adv. Skin Wound Care*, **13**, pp. 225–235

LE, K. M., MADSEN, B. L., BARTH, P. W., KSANDER, G. A., ANGELL, J. B., and VISTNES, L. M. (1984): 'An in-depth look at pressure sores using monolithic silicon pressure sensors', *Plast. Reconstr. Surg.*, **74**, pp. 745–754

LEVINE, J. M., SIMPSON, M., and MCDONALD, R. J. (1989): 'Pressure sores. A plan for primary care prevention', *Geriatrics*, **44**, pp. 75–90

LINDAN, O. (1961): 'Etiology of decubitus ulcers: An experimental study', *Arch. Phys. Med. Rehabil.*, pp. 774–783

LINDGREN, M., UNOSSON, M., and EK, A.-C. (2000): 'Pressure sore prevalence within a public health services area', *Int. J. Nursing Prac.*, **6**, pp. 333–337

LINDGREN, M., UNOSSON, M., KRANTZ, A.-M., and EK, A.-C. (2002): 'A risk assessment scale for the prediction of pressure sore development: reliability and validity', *J. Adv. Nurs.*, **38**, pp. 190–199

LINDGREN, M., UNOSSON, M., FREDRIKSON, M., and EK, A.-C. (2004): 'Immobility – a major risk factor for development of pressure ulcers among adult hospitalized patients: a prospective study', *Scand. J. Caring Sci.*, **18**, pp. 57–64

LINDHOLM, C., BERGSTEN, A., and BERGLUND, E. (1999): 'Chronic wounds and nursing care', *J. Wound Care*, **8**, pp. 5–10

LIU, M. H., GRIMM, D. R., TEODORESCU, V., KRONOWITZ, S. J., and BAUMAN, W. A. (1999): 'Transcutaneous oxygen tension in subjects with paraplegia with and without pressure ulcers: A preliminary report', *J. Rehabil. Res. Dev.*, **36**

LYDER, C. H., SHANNON, R., EMPLEO-FRAZIER, O., MCGEHEE, D., and WHITE, C. (2002): 'A comprehensive program to prevent pressure ulcers in long-term care: Exploring costs and outcomes', *Ostomy/Wound Manag.*, **48**, pp. 52–62

MAKLEBUST, J., MONDOUX, L., and SIEGGREEN, M. (1986): 'Pressure relief characteristics of various support surfaces used in prevention and treatment of pressure ulcers', *J. Enterostomal Ther.*, **13**, pp. 85–89

MARUM, R. J. V., MEIJER, J. H., OOMS, M. E., KOSTENSE, P. J., EIJK, J. T. V., and RIBBE, M. W. (2001): 'Relationship between internal risk factors for development of decubitus ulcers and the blood flow response following pressure load', *Angiology*, **52**, pp. 409–416

MAYROVITZ, H. N., and SIMS, N. (2001): 'Biophysical effects of water and synthetic urine on skin', *Adv. Skin Wound Care*, **14**, pp. 302–308

MAYROVITZ, H. N., and SIMS, N. (2002): 'Effects of different cyclic pressurization and relief patterns on heel skin blood perfusion', *Adv. Skin Wound Care*, **15**, pp. 158–164

MCLEOD, A. G. (1997): 'Principles of alternating pressure surfaces', *Adv. Wound Care*, **10**

MILLER, G. E., and SEALE, J. (1981): 'Lymphatic clearance during compressive loading', *Lymphology*, **14**, pp. 161–166

MURRAY, A., and MARJANOVIC, D. (1997): 'Optical assessment of recovery of tissue blood supply after removal of externally applied pressure', *Med. Biol. Eng. Comput.*, **35**, pp. 425–427

NEWSON, T. P., PEARCY, M. J., and ROLFE, P. (1981): 'Skin surface PO_2 measurement and the effect of externally applied pressure', *Arch. Phys. Med. Rehabil.*, **62**, pp. 390–392

NICHOLSON, G. P., SCALES, J. T., CLARK, R. P., and CALCINA-GOFF, M. L. D. (1999): 'A method for determining the heat transfer and water vapour permeability of patient support systems', *Med. Eng. Phys.*, **21**, pp. 701–712

NIITSUMA, J., YANO, H., and TOGAWA, T. (2003): 'Experimental study of decubitus formation in the rabbit ear lobe', *J. Rehabil. Res. Dev.*, **40**, pp. 67–72

NOLA, G. T., and VISTNES, L. M. (1980): 'Differential response of skin and muscle in the experimental production of pressure sores', *Plast. Reconstr. Surg.*, **66**, pp. 728–733

NORTON, D., MCLAREN, R., and EXTON-SMITH, A. N. (1979): 'An investigation of geriatric problems in hospital' (Churchill Livingstone, London, UK, 1979)

ÖBERG, P. Å. (1990): 'Innovations and precautions', in SHEPERD, A. P. and ÖBERG, P. A. (Eds.): 'Laser-Doppler blood flowmetry' (Kluwer Academic Publishers, Norwell, USA), p. 94

PATEL, S., KNAPP, C. F., DONOFRIO, J. C., and SALCIDO, R. (1999): 'Temperature effects on surface pressure-induced changes in rat skin perfusion: Implications in pressure ulcer development', *J. Rehabil. Res. Dev.*, **36**, pp. 0–0

REDDY, N. P., COCHRAN, G. V. B., and KROUSKOP, T. A. (1981*b*): 'Interstitial fluid flow as a factor in decubitus ulcer formation', *J. Biomech.*, **14**, pp. 879–881

REDDY, N. P., PALMIERI, V., and COCHRAN, G. V. B. (1981*a*): 'Subcutaneous interstitial fluid pressure during external loading', *Am. J. Physiol.*, **240**, pp. R327–R329

REDDY, N. P., PALMIERI, V., and COCHRAN, G. V. B. (1984): 'Evaluation of transducer performance for buttock-cushion interface pressure measurements', *J. Rehabil. Res. Dev.*, **21**, pp. 43–50

RITHALIA, S., and KENNEY, L. (2000): 'Hospital bed mattresses: an overview of technical aspects', *J. Med. Eng. Technol.*, **24**, pp. 32–39

RITHALIA, S. V., HEATH, G. H., and GONSALKORALE, M. (2000): 'Assessment of alternating-pressure air mattresses using a time-based pressure threshold technique and continuous measurements of transcutaneous gases', *J. Tissue Viability*, **10**, pp. 13–19

RITHALIA, S. V. S., and GONSALKORALE, M. (2000): 'Quantification of pressure relief using interface pressure and tissue perfusion in

alternating pressure air mattresses', *Arch. Phys. Med. Rehabil.*, **81**, pp. 1364–1369
RONDORF-KLYM, L. M., and LANGEMO, D. (1993): 'Relationship between body weigh, body position, support surface, and tissue interface pressure at the sacrum', *Decubitus*, **6**, pp. 22–30
SACHSE, R. E., FINK, S. A., and KLITZMAN, B. (1998): 'Multimodality evaluation of pressure relief surfaces', *Plast. Reconstr. Surg.*, **102**, pp. 2381–2387
SACKS, A. H., KSANDER, G., O'NEILL, H., and PERKASH, I. (1988): 'Difficulties in laser Doppler measurement of skin blood flow under applied external pressure', *J Rehabil. Res. Dev.*, **25**, pp. 19–24
SALCIDO, R., DONOFRIO, J. C., FISHER, S. B., LEGRAND, E. K., DICKEY, K., CARNEY, J. M., SCHOSSER, R., and LIANG, R. (1994): 'Histopathology of pressure ulcers as a result of sequential computer-controlled pressure sessions in a fuzzy rat model', *Adv. Wound Care*, **7**, pp. 23–40
SANADA, H., KANAGAWA, K., INAGAKI, M., IMAE, J., NISHIMURA, M., YOSHIO, K., and HIRAMATSU, T. (1995): 'A study on the prevention of pressure ulcers: The relationship between transcutaneous PO_2 in the sacral region and predictive factors for pressure ulcer development', *Wounds*, **7**, pp. 17–23
SANADA, H., NAGAKAWA, T., YAMAMOTO, M., HIGASHIDANI, K., TSURU, H., and SUGAMA, J. (1997): 'The role of skin blood flow in pressure ulcer development during surgery', *Adv. Wound Care*, **10**, pp. 29–34
SANDBERG, M., ZHANG, Q., STYF, J., GERDLE, B., and LINDBERG, L.-G. (2005): 'Non-invasive monitoring of muscle blood perfusion by photopletysmography: – evaluation of a new application', *Acta Physiol Scand.*, **183**, pp. 335–343
SCHUBERT, V., and FAGRELL, B. (1989): 'Local skin pressure and its effects on skin microcirculation as evaluated by laser-Doppler fluxmetry', *Clin. Physiol.*, **9**, pp. 535–545
SCHUBERT, V. (1991): 'Hypotension as a risk factor for the development of pressure sores in elderly subjects', *Age Ageing*, **20**, pp. 255–261
SEILER, W. O., and STÄHELIN, H. B. (1979): 'Skin oxygen tension as a function of imposed skin pressure: Implication for decubitus ulcer formation', *J. Am. Geriatr. Soc.*, **XXVII**, pp. 298–301
STEWART, S. F. C., PALMIERI, V., and COCHRAN, G. V. B. (1980): 'Wheelchair cushion effect on skin temperature, heat flux, and relative humidity', *Arch. Phys. Med. Rehabil.*, **61**, pp. 229–233
SULZBERGER, M. B., CORTESE, T. A., FISHMAN, L., and WILEY, H. S. (1966): 'Studies on blister produced by friction', *J. Invest. Dermatol.*, **47**, pp. 456–465
TENLAND, T., SALERUD, E. G., NILSSON, G. E., and ÖBERG, P. Å. (1983): 'Spatial and temporal variations in human skin blood flow', *Int J Microcirc: Clin Exp*, **2**, pp. 81–90
WÅRDELL, K., JAKOBSSON, A., and NILSSON, G. E. (1993): 'Laser Doppler perfusion imaging by dynamic light scattering', *IEEE Trans. Biomed. Eng.*, **40**, pp. 309–316
WÅRDELL, K., BRAVERMAN, I. M., SILVERMAN, D. G., and NILSSON, G. E. (1994): 'Spatial heterogeneity in normal skin perfusion recorded with laser Doppler imaging and flowmetry', *Microvasc. Res.*, **48**, pp. 26–38
WHITTEMORE, R. (1998): 'Pressure-reduction support surfaces: A review of the literature', *J. Wound, Ostomy Continence Nurs.*, **25**, pp. 6–25
WITKOWSKI, J. A., and PARISH, L. C. (1982): 'Histopathology of the decubitus ulcer', *J. Am. Acad. Dermatol.*, **6**, pp. 1014–1021
WYWIALOWSKI, E. F. (1999): 'Tissue perfusion as a key underlying concept of pressure ulcer development and treatment', *J. Vasc. Nurs.*, **17**, pp. 12–16
XAKELLIS, G. C., FRANTZ, R. A., ARTEAGA, M., and MELETIOU, S. (1991): 'A comparison of changes in the transcutaneous oxygen tension and capillary blood flow in the skin with increasing compressive weights', *Am. J. Phys. Med. Rehabil.*, **70**, pp. 172–177
XAKELLIS, G. C., FRANTZ, R. A., ARTEAGA, M., and MELETIOU, S. (1993): 'Dermal blood flow response to constant pressure in healthy older and younger subjects', *J. Gerontol.*, **48**, pp. M6–M9
ZHANG, M., and ROBERTS, V. C. (1993): 'The effect of shear forces externally applied to skin surface on underlying tissues', *J. Biomed. Eng.*, **15**, pp. 451–456

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