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Pressure management

J. C. BARBENEL

Bioengineering Unit, University of Strathclyde, Glasgow, UK

Abstract

The prevention or minimisation of the occurrence of pressure sores is an important consideration in the rehabilitation of physically disabled people, especially for the wheelchair user with a spinal cord injury.

Although there is little definitive information on the cause of pressure sores, several intrinsic and extrinsic factors have been highlighted. Probably the most significant causative factor is the application of force to the skin surface. The relationship between the magnitude of pressure and its duration; the temperature and humidity at the interface; and the physiological effects that this has on the microcirculation and lymphatic drainage are discussed in this article.

It is suggested that a rationale for the prevention of pressure sores includes the limitation of the duration of pressures applied to the skin surface and the reduction of the peak pressures particularly at vulnerable sites. In this context the design criteria for a clinical interface pressure measurement system, and the uses and limitations of the commercially available options, are considered. The development of a structured programme of wheelchair and support surface provision, assessment and follow-up is required.

Introduction

Pressure sores are a major problem for people who have areas of tissue which lack sensation or who have limited natural mobility. These deficits affect very many who use wheelchairs and the prevention of pressure sores is a key consideration in the rehabilitation of patients who require a wheelchair and in the provision of a suitable chair and cushion.

There is little reliable information on the incidence or prevalence of sores related to the use of wheelchairs. A survey of 995 patients in the Borders Health Area in Scotland showed that 24% of the 121 wheelchair users had a pressure sore compared to to 7.4% of the 874 patients who did not use a wheelchair (Barbenel, 1985), a highly significant difference.

Spinal-cord-injured wheelchair users are at special risk of developing sores and it has been estimated that pressure sores affect 5-10% of such wheelchair users each year (Ferguson-Pell *et al.*, 1980). The same study showed that the average duration of hospital in-patient treatment for ischial sores was 70 days. These figures imply that pressure sores in wheelchair users represent a considerable financial cost and medical and nursing burden. In addition the sores are a source of major discomfort and disruption to the lives of the patients who have them and recurrent sores may make it impossible for them to gain employment.

Reliable, definitive and objective information on the cause of pressure sores remains limited and fragmentary. The prevention of sores is based both on this limited scientific evidence and empirical experience. The development of a structured programme of wheelchair and support surface provision, assessment and follow-up, within which the idea of pressure sore prevention can be implemented, is the key to the successful prevention of the majority of pressure sores.

Causation

Pressure sores are a form of non-specific damage which can be caused in many ways. The results of systematic programmes of pressure sore prevention for wheelchair users (Barbenel, 1989) suggest that there is an unavoidable minimum number of sores which may be caused by trauma during transfer, friction etc. Nevertheless, the major cause of pressure sores is the application of forces to the skin surface for prolonged periods of time. Additional extrinsic factors include the presence of abnormally high temperatures and moisture accumulation at the loading area of the skin. Intrinsic factors such as the thickness and conditions of the skin and subcutaneous tissues, the presence of scars and the nutritional status of the patient may also be of great importance. Posture may also greatly influence the magnitude and distribution of forces acting on the tissues.

All correspondence to be addressed to J. C. Barbenel, Bioengineering Unit, University of Strathclyde, Wolfson Centre, 106 Rottenrow, Glasgow G4 0NW, UK.

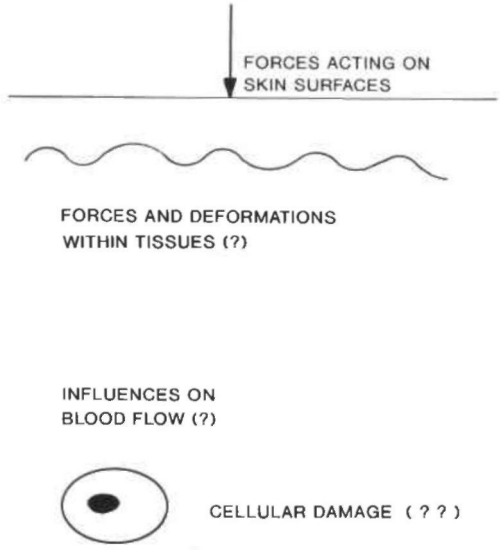

Fig. 1. Forces acting on the skin surface may produce cellular damage, but details of the effects of pressure on the tissues become increasingly uncertain (indicated by the question marks).

Ultimately, pressure sores are produced by damage to, and destruction of, cells within the tissues. There is clearly a chain of cause and effect, linking forces applied to the skin surface with cellular damage (Fig. 1), but it is also a chain of increasing ignorance. The forces applied to the skin surface can be measured with some degree of precision. The local forces and deformation which the surface forces produce within the tissues are much less well understood, and physical or finite element models which might produce detailed information are extremely difficult to validate. The effect of these local forces and deformations on the microcirculation and lymphatic drainage of the tissues have been investigated, but only the most general information has been obtained which appears to be both subject and device dependent. Finally, the response of the cells to the unfavourable mechanical and nutritional environment produced by the surface forces is particularly ill-understood. Damage can occur to a variety of target cells which may be within the muscle or overlying fat and skin or the cells of the dermal-epidermal junction, but it is not known which type of cell will be damaged by which combination of causative factors.

Pressure and time

The force applied to the surface of the skin can be resolved into two components. One acts at right angles to the skin and is generally called the pressure; the other acts parallel to the skin surface and is known as shear. Devices for measuring the forces on the skin measure the pressure (although the shear forces may affect the measurement). There is no satisfactory method of measuring the shear forces. Consequently, quantitative investigation and discussion of forces acting on the skin refer to the effects of pressure.

Pressure has been known to be a causative factor in pressure sores for many years (see historical references in Silver, 1990). Kosiak *et al.* (1958) were the first to systematically investigate the magnitude and duration of pressures required to produce tissue damage. Forces of various magnitudes were applied to skin over the femoral trochanters and ischial tuberosities of dogs for variable durations and the condition of the loaded tissues examined histologically. It was found that tissue damage occurred after the application of high forces for a relatively short time, or lower forces for longer times. The experiments have been repeated and the general conclusion that both magnitude and duration of force are critical in developing pressure sores has been verified by other workers (Lindan, 1961; Daniel *et al.*, 1981). The work was, inevitably, carried out on animals (dog, rat, rabbit and pig) and within the overall verification of the general conclusion, it is clear that there are major differences in the magnitudes and duration of forces which the various workers have reported as being necessary to produce tissue damage.

Although there have been no controlled investigations of the forces and times needed to produce tissue damage in humans, there is some limited data. Trumble (1930) found that local pressures of about 78mmHg applied to the skin of patients produced complaints of pain and he was able to show the collapse of capillaries and veins. The most clinically useful results are those of Reswick and Rogers (1976) who measured the pressures over the bony prominences of 980 seated subjects. The tolerance curve they produced (Fig. 2) confirms the inverse relationship between pressure and duration, and has been widely used. The authors themselves warn that it should, however, be used as a general guideline rather than being taken as absolute.

Physiological effects of pressure

Applying even moderate pressures to the skin surface results in blanching. This observation has led to the interaction between pressures and microcirculation being given a central role in the causation of pressure sores. There have been numerous investigations of the response of the superficial microcirculation to load. In addition, it has been suggested that the lymphatic drainage of tissues may be disrupted by pressure.

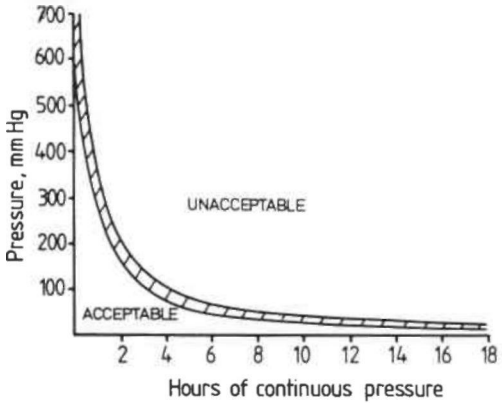

Fig. 2. Pressure-time tolerance curve, after Reswick and Rogers (1976).

The effect of external loading on the skin microcirculation has been assessed using radioisotope clearance (Daly *et al.*, 1976; Larsen *et al.*, 1979), photoplethysmography (Barbenel *et al.*, 1976; Bennet *et al.*, 1984), transcutaneous oxygen tension measurements (Newson and Rolfe, 1982; Bader, 1990) and laser Doppler flowmetry (Sacks *et al.*, 1988). Transcutaneous oxygen measurements have the major drawback that they can only be realistically carried out at elevated skin temperatures, and it is unclear whether the results of such tests can be extrapolated to more normal conditions. Optical methods have the advantage of rapid response time and allow tests to be completed in a shorter timescale.

The results reported by the individual groups are highly variable and difficult to summarise, but there is no evidence of the existence of the critical closing pressure, analogous to the capillary blood pressure, at which a marked discontinuity in the pressure-microcirculatory response occurs. The results do, however, confirm that higher pressures produce greater disruption of the microcirculation, providing an underlying causation for the inverse relationship between pressure and time required to produce tissue damage which is found experimentally. Although it has been suggested that the microcirculation in the denervated skin of paraplegic patients is more sensitive to load than normal skin, little evidence has been found to support this except during repetitive loading (Barbenel *et al.*, 1976).

The lymphatic drainage of the subcutaneous tissues of dogs has been investigated by Miller and Seale (1981) who injected Tc-99m labelled sulphur. Lymphatic clearance rates were measured with the tissue unloaded and under external compression. Clearance was found to be normal until the external pressures reached 60-70mmHg, after which the lymphatic flow rates were reduced almost to zero. A mathematical model of a lymphatic drainage and the effects of external pressure on interstitial fluid dynamics has been presented by Reddy *et al.*, (1981). The simple linear model appears to predict an inverse relationship between the magnitude of pressure and loading duration required for the interstitial fluid volume to reach any given proportion of its initial volume. The relationship is similar to that in Figure 2 and has led Reddy and co-workers to suggest that lymphatic fluid flow may play a significant role in the physiological response of tissues to load, which ultimately leads to tissue damage.

Temperature and humidity

Most wheelchair cushions are made of materials which are poor conductors of heat and which disrupt the normal heat loss from the skin surface. This results in temperature elevations in the tissues at the contact areas, which will increase the local metabolic rate and may further exacerbate the deleterious effect of pressure. The interface temperatures have been shown to depend both on the thermal properties of the support surface and the ambient temperature (Brattgard *et al.*, 1976). Materials with a high heat capacity, such as gel cushions, will take a considerably longer time to reach their equilibrium elevated temperature than will foam cushions. Movements which disrupt the contact between the skin and support surface will immediately change the interface forces, but the tissue temperature will remain elevated for some time (Barbenel *et al.*, 1978).

Temperature elevations at contact areas are commonly associated with sweating and a high local relative humidity. As with temperature, the magnitude of the effect depends both on the nature of the support surface and the environmental conditions (Brattgard *et al.*, 1976).

Tissue thickness and condition

Skin thickness can be measured using both A and B scan ultrasound. Magnetic resonance has been used to produce images of the cross-sectional anatomy of test subjects (Reger *et al.*, 1990). These images show the skin, subcutaneous tissue and muscle and allow the influence of load on tissue thickness to be evaluated.

There is a wide variety of devices for measuring the mechanical properties of the skin in vivo (Barbenel, 1987) but the complexity of the measurement and analysis and doubts about the utility of the measurements has limited such tests.

Tissue condition can be measured by a variety

of biochemcial tests and spinal injury patients have been shown to have collagen breakdown (Claus-Walker *et al.*, 1973), protein deficiency (Kermani *et al.*, 1970; Moolton, 1972) and reduced leucocyte ascorbic acid concentration (Burr and Rajan, 1972).

Measurement of interface pressure

The multiple factors associated with pressure sore formation, which have been outlined in the previous section, provide the rationale and potential for the measurement of many variables. Interface pressure is the only variable which has been routinely measured and shown to be of practical significance.

The requirements which an interface pressure sensor must satisfy have been understood in general qualitative terms for some time (Pressure and Force Measurements, 1968). More recently these have been used to provide estimates of design parameters (Ferguson-Pell, 1980), but it has proved difficult to design and construct usable devices which satisfy these guidelines.

A key problem is that placing a sensor between the skin and the soft support surface, such as a cushion, disturbs the conditions which determine the pressure and which the sensor is required to measure. Obviously a large rigid sensor will indent both surfaces and the measured pressure will depend much more on the device than on the interface pressures under the resting conditions. It is obvious that the sensor should be as thin as possible, and it appears that both the thickness and diameter of the device are important. It has been suggested that the diameter to thickness ratio should not be less than 10:1. In order to produce least disturbance of the original pressure and geometry of the interface, it is also desirable that the sensor be flexible.

The sensor of choice depends on the use to which it is to be put. For routine clinical use, sensors which provide a static pressure measurement which is essentially a snapshot of the pressure distribution at a single instant, have proved very useful. Simple pressure mapping methods using pressure-sensitive, dye-releasing capsules (Brand and Ebner, 1969) or sheets impregnated with chemicals which react at a rate modified by the applied pressure (Frisina and Lehneis, 1970) have been described. Unfortunately these very simple systems are sensitive to temperature and other factors, in addition to pressure, and this has made the pressure values obtained unreliable and limited their use.

Simple electropneumatic sensors, first described by Mooney *et al.* (1971) have been extremely useful for routine measurement of interface pressures. They are closed systems which are inflated by a standard sphygmomanometer. The sensors consist of flat capsules with thin flexible walls which have electrical contacts on the opposing internal surfaces. The sensor is placed at the patient-support surface interface and inflated until the pressure is sufficient to separate the walls of the capsules and electrical contacts, producing a signal commonly shown by the switching of a LED. The capsule is then allowed to slowly deflate until the indicator shows that the walls are once again in contact. The pressure at which this occurs is then taken as the interface pressure. The major advantage of these sensors is their simplicity and commercial availabilty (Talley Surgical Instruments Ltd., Boreham Wood, Herts, UK).

The simple elecropneumatic sensors have formed the basis of more complex systems. Multiple sensors can be constructed in sheet form to produce pressure mapping systems (Garber *et al.*, 1978; Mayo-Smith, 1980; Pratt *et al.*, 1980; Bader and Hawken, 1986). A commercial system (Talley Surgical Instruments Ltd.) has proved popular for clinical use.

Continuous output sensors are useful if the pressure-time history occurring at the patient support interface is being investigated, and also have applications for routine use. Strain-gauged diaphragm transducers for measuring hydrostatic pressure are readily available commerically, as is the necessary signal-conditioning equipment. The sensors themselves can be obtaned in very small sizes and with diameters of less than 3mm and thickness less than 1mm, although they are rigid. A major drawback for their routine use is their considerable cost.

There are few other sensors suitable for measuring interface pressure which are regularly available commerically. Numerous specially constructed devices have been described in the literature. These generally rely on pressure induced changes in either the resistance or the capacitance of materials within the sensor which produces an electrical output; both types suffer from two major defects. The pressure sensitive material in the sensor has to be relatively soft in order to obtain sufficient sensitivity. Most soft materials are however time-dependent, showing such behaviour as hysteresis and creep. The electrical output produced by such time dependent devices depends not only on the load but also previous load history to which the device has been subjected and it is extremely difficult to obtain an unambiguous measurement of pressure. Under these circumstances the devices usually have a non-linear relationship between

the output and the applied pressure and are commonly sensitive to temperature and curvature (Barbenel, 1983).

A recently described combination of a fluid filled sensor bag and pressure transducer (Barbenel and Sockalingham, 1990) has been used to measure the interface pressure beneath elastic bandages, and has considerable application in measuring interface pressures including the interface between the subject and the support surfaces.

All the sensors described above appear to be sensitive to the interface conditions and will generally not produce the same output reading under identical test conditions. This makes the quantitative comparison of the work of different groups using different sensors difficult. In addition care must be taken in interpreting the absolute values of pressure-time tolerance curves (as in Fig. 2) as these measurements will depend on the sensors used for carrying out the initial research.

Pressure sore prevention

The pressure beneath a subject seated in a wheelchair is not uniformly distributed, but is highest over the bony prominences of the ischial tuberosities and the greater trochanters, the sites at which pressure sores are most prevalent (Barbenel *et al.*, 1977). The assessment of patients, available support devices and the matching of the two are discussed in Choosing a Wheelchair System (1990) and elsewhere in this supplement. The starting point for pressure sore prevention must be the provision of a suitable wheelchair, correctly adjusted for the patient. The rationale for pressure sore prevention consists of three aims:

i) to limit the duration for which pressure acts,
ii) to reduce the peak pressures at vulnerable sites by distributing the pressure beneath the patient more uniformly or selectively unloading at-risk sites,
iii) to eliminate as far as possible such unfavourable factors as friction, poor posture and malnutrition.

Patients who can make regular movements should be trained and encouraged to provide regular pressure relief by push-ups or rocking from side to side. The preferred frequency and duration of pressure relief is largely a matter of personal opinion and values which have been suggested include ten seconds relief every ten minutes (Noble, 1981), to one or two minutes relief every hour (Watson, 1983). Measurements suggest, however, that patients actually make relief movements at irregular intervals and that the periods of relief are of variable duration (Fisher and Paterson, 1983; Barbenal *et al.*, 1984; Merbitz *et al.*, 1985; Ferguson-Pell *et al.*, 1990).

The magnitude of the peak pressures is less amenable to simple modification. Since it is not possible to make any great reduction in the weight supported by the wheelchair seat, the average interface pressures cannot be reduced to any great extent. Major alterations can only be achieved by redistributing the pressure to make it more uniform and to reduce it over the "at-risk" sites. These aims can be achieved by the provision of a suitable wheelchair cushion and the aim of any pressure sore prevention programme for seated wheelchair users must be the choice, provision and maintenance of a suitable wheelchair cushion.

The specific wheelchair cushion is probably of secondary importance, compared to its ability to redistribute the pressure, and expert systems have shown promise as an aid for selecting an appropriate cushion (Ferguson-Pell *et al.*, 1989).

The correct support surface is that which can achieve as satisfactory a pressure distribution as is possible and produces no tissue trauma. The primary objective in providing a cushion for wheelchair users to prevent pressure sores is to ensure that the pressures over sites at high risk of developing sores, particularly the ischial tuberosities, are as low as possible and preferably less than 40mmHg. This can only be established by interface pressure measurements with the patient sitting on the chosen cushion. Failure to achieve the desired interface pressure on the cushion selected should logically lead to the evaluation of other cushions. It is, however, not always possible to reach the desired low pressure level and it may be necessary to provide the patient with cushions producing higher than the desired pressures. This should always be associated with special care to ensure that the patients carry out routine pressure relief exercises.

There is considerable variation between patients in their tolerance of pressure. This is reflected in the shaded area of Figure 2 which is in an area of uncertainty and doubt. The suitability of a wheelchair cushion for a patient can only be assessed by inspection of the tissues after the patient has used it. Localised areas of erythema or elevated temperatures are warning signs of tissue trauma and action should be taken to prevent further tissue damage.

Wheelchair cushions have limited durability and after continual use often show a degradation of performance with a danger of increased ischial pressures. As part of any wheelchair and wheelchair cushion provision service, follow-up procedures are essential. The provision of a

satisfactory wheelchair and support surface requires considerable skill, but change with time in the condition of both the devices and the patients, makes it mandatory to recognise that the provision of such assistive devices is the first stage of a programme of continuing care rather than an end in itself.

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