THE ETIOLOGY, PREVENTION AND TREATMENT
OF PRESSURE SORES

Louise Vizzini

An essay presented in accordance with the regulations of hospital pharmacy pre-registration for membership to the Pharmaceutical Society.

GREATER GLASGOW HEALTH BOARD
391 SAUCHIEHALL STREET
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Greater Glasgow Health Board (Northern District) 1976.
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<table>
<thead>
<tr>
<th>Contents List</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction</td>
<td>1</td>
</tr>
<tr>
<td>1. The Etiology of Pressure Sores</td>
<td>2</td>
</tr>
<tr>
<td>1.1 The Skin</td>
<td></td>
</tr>
<tr>
<td>1.11 The Structure of the Skin</td>
<td></td>
</tr>
<tr>
<td>1.1a Skin in Temperature Regulation</td>
<td></td>
</tr>
<tr>
<td>1.2 The Circulation</td>
<td></td>
</tr>
<tr>
<td>1.21 The Capillary Vascular Bed</td>
<td></td>
</tr>
<tr>
<td>1.3 Self-regulating Protective Mechanisms</td>
<td></td>
</tr>
<tr>
<td>1.4 Summary</td>
<td>8</td>
</tr>
<tr>
<td>2. Prevention of Pressure Sores</td>
<td>10</td>
</tr>
<tr>
<td>2.1 Patient Assessment</td>
<td></td>
</tr>
<tr>
<td>2.2 Patient Care</td>
<td></td>
</tr>
<tr>
<td>2.3 Mechanical Aids</td>
<td></td>
</tr>
<tr>
<td>2.31 The Ripple Mattress</td>
<td></td>
</tr>
<tr>
<td>2.32 Fluid Beds</td>
<td></td>
</tr>
<tr>
<td>2.33 Fluidised Air Beds</td>
<td></td>
</tr>
<tr>
<td>2.34 Cushions and Pads</td>
<td></td>
</tr>
<tr>
<td>2.341 Gels</td>
<td></td>
</tr>
<tr>
<td>2.342 Viscous Particulates</td>
<td></td>
</tr>
<tr>
<td>2.343 Foams</td>
<td></td>
</tr>
<tr>
<td>2.344 Air Pads and Cushions</td>
<td></td>
</tr>
<tr>
<td>2.345 Water Pads and Cushions</td>
<td></td>
</tr>
<tr>
<td>2.346 Fluid/Foam</td>
<td></td>
</tr>
<tr>
<td>2.347 Elastic Foam Cushions</td>
<td></td>
</tr>
<tr>
<td>2.348 Visco-Elastic Foam Cushions</td>
<td></td>
</tr>
<tr>
<td>2.349 Bean Bags</td>
<td></td>
</tr>
<tr>
<td>2.350 Solid-body Shaped Seats</td>
<td></td>
</tr>
<tr>
<td>2.351 Gels</td>
<td></td>
</tr>
<tr>
<td>3. Treatment of Pressure Sores</td>
<td>14</td>
</tr>
<tr>
<td>3.1 Medical Treatment</td>
<td></td>
</tr>
<tr>
<td>3.11 Diet</td>
<td></td>
</tr>
<tr>
<td>3.2 Supportive Treatment</td>
<td></td>
</tr>
<tr>
<td>3.3 Surgical Treatment</td>
<td></td>
</tr>
<tr>
<td>Conclusion</td>
<td>17</td>
</tr>
</tbody>
</table>
Figures

<table>
<thead>
<tr>
<th>Fig.</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fig. 1</td>
<td>The Structure of the Skin</td>
<td>2</td>
</tr>
<tr>
<td>Fig. 2</td>
<td>Skin in Temperature Regulation</td>
<td>3</td>
</tr>
<tr>
<td>Fig. 3</td>
<td>Effect of forces on Capillary Interchange Flow</td>
<td>5</td>
</tr>
</tbody>
</table>
Introduction

At the beginning of this century the patients who were most prone to develop pressure sores were young people suffering from certain wasting diseases such as tuberculosis, osteomyelitis, typhoid fever and renal disease. Today, by far the largest susceptible group consists of the elderly, who in increasing numbers suffer from long-term debilitating illnesses.

Although the world's population will double, within the next 35 years those aged 60 and over will be twice as numerous within only 30 years; thus the number of elderly is increasing even more rapidly than the world population. (1) In France, by the year 2,000, as compared with 1970, those aged 85 and over will increase by 122 per cent.

The young chronic sick e.g. with multiple sclerosis now live into the upper age ranges and thus the elderly of tomorrow will include many more frail and disabled people than today. Although the incidence of pressure sores rises steadily with age (2) considerable attention must be devoted to the potential damaging effects of sustained pressure on the tissues, in all age-groups of hospitalised patients.

When patients develop pressure sores, treatment occupies three-fifths of the time the patient spends in hospital. A tremendous strain is imposed on the nursing resources, as one-third of a nurse's working time can be occupied attending to pressure sores.

About 25,000 people in the U.K. suffer from pressure sores and the cost of the subsequent hospital treatment incurred is about £60 million per annum (3)

Thus it is observed that the problem of pressure sores is large, and expensive, and will be even more so in future years.

The aim of this essay is to present the basic principals of the etiology, prevention and treatment of the complex issue of pressure sores.
Since pressure sores do not occur in the ordinary healthy individual, consideration will first be given to the relevant normal protective mechanisms of the body. In the context of this essay, the term 'pressure sore' will be taken to mean: "An ischemic necrosis and ulceration of tissues overlying a bony prominence which has been subjected to prolonged pressure against an external object" (4) Synonomous terms include: Bedsore, decubitus ulcer, TROPHIC ULCER. As will be outlined, other factors can be additive in producing a pressure sore.

1. 1. The Skin:
   The function of the skin is to protect the internal environment of the body, to provide contact with the external environment and to allow the body to maintain internal temperature.

1. 1.1. The Structure of the Skin:

Figure 1: The Structure of the Skin

Figure 1 illustrates the main layers and cells of the skin. All skin has 3 basis layers; the epidermis, the dermis and the telasubcutanea. The thickness of each varies around the body.

The Dermis is a fibrous connective tissue. A high proportion of collagen fibres provides tough tensile strength to the skin. Elastin, present as interlacing fibres provides tensile strength and plasticity in all directions.

The Basal cell layer represents the growing point for all of the upper epidermal cells. This basal layer undergoes continuing mitosis and eventually as the cells change and go up the layer they form the dead keratinised flat surface of the
Stratum Corneum. The rate of replacement of skin cells seems to be determined by a negative feedback mechanism on the amount of chalone present in the prickly cell layer. Chalone depresses the mitotic activity of the basal cells. However, if cells are lost, for example on injury, there is less chalone, hence inhibition of mitosis is less giving increased production of basal cells and resulting in cell replacement. The activity of basal cells is also affected by general hormones, for example cortical steroids, sex hormones and thyroid. It is thought these agents may influence the rate of production of the chalone, or act directly on the basal cells themselves.

Shearing forces applied to the skin can be a factor in bed-sore formation. For example, when a patient lies down a bed the superficial layers of skin move across the deeper fixed layers causing blood vessels to be subjected to pressure, kinking and rupture (5).

It has been reported (6) that the mechanical properties of human skin may alter with age and sex. Using graphs, it was found that there are variations in the shape and average slope of the stress-strain curves.

It has been found that the average slope (stiffness) decreases through maturation, reaches a minimum between ages of 15 and 25 and then appears to increase with advancing age. The data points begin to diverge after approximately 30 years of age. It was hypothesized that this diverging phenomenon may be a result of the cumulative effect of ultraviolet radiation on collagen and elastin networks. An examination of the shape of the curves appears to indicate that stiffness of collagen in the dermis increases with increasing age. However it appeared that the lower portions of the curves, possibly associated with the elastin and ground substance components undergo changes not common to every individual. It was also hypothesized that 'scatter' in the data obtained from females and the sudden change noted in the shape of the curves at puberty, may be due to hormonal changes.

Another important function of the skin is temperature regulation.

12. Skin in temperature regulation

The main blood artery runs in the tela subcutanea layer of the skin. It is well insulated from heat gain or loss. These arteries send loops (arterioles) up to the surface, these very small arterioles are characterized by delicate smooth muscle control (i.e. their calibre is controlled) situated at the basal cell layer, are the blood capillaries which drain into tiny venules and veins.

There is a direct junction between arteriole and venule called an Arteriovenous Anastomosis.

If skin is cold, a vasoconstrictor reflex mediated by α-adreno-receptors at the afferent arterioles is elicited i.e. blood can only flow to B (see Fig 2)
If skin is warm, the vessels are relaxed and blood flows up A (see Fig 2) and supplies dermis and basal layers of the skin. This does not apply to the upper layers of the skin. It is probably deficiency of nutrients which enhances the degeneration process in upper layers.

Thus by shunting blood near surface, the temperature can be regulated. Heat is transferred by conduction through the epidermis and at the surface by convection and radiation.

It is known that clothes inhibit the conduction and convection of heat. This fact is of importance to the bed-fast or chair fast patient. A local rise of temperature in skin can result in an increase in the property of penetration in that area. Therefore any mild irritation which produces erythema can result in penetration by another organism and lead to infection of the skin with possible tissue degeneration. Wheelchair bound persons are often obliged to sit continuously for many hours at a time in the same position.

It has been reported (7) that high temperature and humidity in the sitting area have a bearing on the initiation of "chair sores" and resultant infections.

Traditional wheelchair seats and covers have high temperature and humidity in the sitting area, with skin temperature reaching the equivalent of body temperature.

Terry towelling or sheepskin covers did not influence the temperature significantly although they did improve the humidity conditions compared to conventional wheelchair covers.

Plastic materials gave high humidity condition, while "Net-work" woven plastic bands gave best results as far as temperature and humidity were concerned.

The environmental room temperature appear to affect the sitting area humidity. High humidity values appear to be produced by room temperatures of 25°C or higher. Thus it is necessary when assessing material covers to consider general environmental factors.

The sweat gland of the skin further aids temperature regulation and is an important excretion site for nitrogenous substances.

When hot, the cells lining the coils of the sweat gland by means of cholinergic sympathetic innervation stimulate the sweat gland to produce a thin aqueous secretion. This secretion is transported to the surface of the skin where it evaporates and extracts latent heat of evaporation from the surface of the epidermis. Temperature regulation by blood shunting is also in play.

The constituents of sweat are: little protein, minute traces of glucose, $A^-$, $SO_4^{=}$, Na($+$), K($+$), Ca($++$), and the nitrogenous materials, urea and uric acid.

Thus sweating is a secondary means of excreting urea and uric acid.

The glands are constantly active and effect a reasonable loss of nitrogenous substances.

If loss of heat by evaporation of the secretions of the sweat gland is hindered by clothing or non porous seat covering, this will obviously increase humidity locally.
It has been reported (8) that moisture of skin due to sweating and incontinence of urine are extrinsic factors in producing infection and early decomposition of tissue once ischaemia due to pressure has developed.

The integrity of the skin depends on receiving sufficient materials such as nutrients and oxygen to enable the continuation of the dynamic process of regeneration. These nutrients are transported around the whole body to organs, tissues and the skin. Consideration is now given to circulation.

1. 2 The Circulation:

The heart is a pump which drives the blood (a complex fluid containing food materials, respiratory gases, waste products, protective and regulating chemical substances) around the blood vessels in a closed system of tubes.

Blood is transported from the pump (heart) by arteries to the tissues of the body. The arteries branch into capillaries where the interchange of gases and waste substances occurs. The capillaries reunite to form veins which convey blood from the tissues of the body back to the 'pump' (heart).

It is in the capillary bed that interchange of gases and nutrients occurs, this process is described below.

1. 2.1 The Capillary Vascular Bed

The bed consists of capillary vessels of one cell thickness. The cells lining the bed are endothelial 'pavement' cells. The erythrocyte is squeezed on passing through the capillary and optimum conditions for two way transfer is achieved.

The blood flow through the capillary bed is controlled by the pre-capillary sphincters which are innervated by sympathetic nerves. Regulation of flow is achieved by vasomotion, whereby open sphincters rhythmically close while closed ones open.

In a state of high metabolic activity most sphincters are dilated, the blood then courses through the capillaries and results in nourishment. In low metabolic activity less sphincters are opened and thus less nourishment of tissues occurs.

Nutrient flow is determined by the algebraic sum of hydrostatic and osmotic pressure existing across the membrane.

The hydrostatic pressure is approximately 32 mm Hg at the arterial end of the capillary bed and drops to to 15 mm Hg at the venous end of the bed.

Interstitial fluid pressure (≈ 7 mm Hg) exists outside the capillary wall and aids in the driving force so allowing filtration to occur.

In the capillary are large molecules, for example albumin, which cannot diffuse across the membrane. Such molecules provide a colloid osmotic pressure of 25 mm Hg, on the membrane which opposes the driving force described above. Thus the net driving force across the capillary membrane is 14 mm Hg.
The high hydrostatic pressure forces water and molecules of less than 60,000 such as \( \text{O}_2 \) and glucose out of the capillary.

As these molecules leave the capillary space, the protein concentration rises causing osmotic pressure to rise. Thus, as hydrostatic pressure decreases osmotic pressure increases. As the blood moves along the capillary the lower hydrostatic pressure decreases the loss of substances from the capillary while the increasing osmotic pressure causes substances to enter the capillary. In this manner such as waste products, \( \text{CO}_2 \), urea are brought into the capillary.

Not all fluid returns to the capillary, since some enters the tissue spaces and is drained by lymph vessels.

A very important function is therefore carried out by the delicate capillary. If the flow of blood is blocked anywhere in the circulatory vessels, for example by platelet aggregation, resultant damage to the tissues served by the damaged vessel can lead to tissue starvation and necrosis.

Skin necrosis is basically due to interference with the skin's blood supply. Any continual pressure which is sufficient to disrupt the circulation for more than a few hours will lead to tissue death and an ulcer or sore (9). The relationship between externally applied pressure and skin blood flow is most significant in the occurrence of decubitus ulcers.

It has been reported that the flow of blood in human skin is most significantly reduced by pressures of up to 15 mm Hg, although further increases in pressure up to 50 mm Hg (10) continues to reduce blood flow, but less dramatically.

The wounding of the skin from pressure has been reported to be usually the result of 2 concurring factors which cause anoxic necrosis: (11) (a) Exclusion of blood from the skin by pressures in excess of the mean capillary pressure (i.e. \( P_a \)).
(b) Disruptive damage to the skin commonly caused by intermittent blows or shearing forces, which damage blood vessels and cause platelet thrombosis of microcirculation. Force is distributed over a wide area of the skin but tends to be concentrated within a smaller area where muscle or subcutaneous tissue overlies bony prominences. Since it is here that the pressure is greatest (Peak Pressure), the more serious type of pressure sore starts in the deep tissue and spreads outwards to involve the skin. Thus, at a time when the skin only shows erythema, extensive and irreparable damage may have occurred in the deep tissues.

With the exception of the soles of the feet, the skin of man is not adapted to sustain pressures of more than 40 mm Hg for periods of a few hours.

An important aspect of the mechanical behaviour of human tissue is that its deformation under load is time dependent. In other words, if a force is applied to human tissue, the tissue will deform by a certain amount as soon as the force is applied. However, if the force is maintained on the tissue, the tissue will continue to deform for some hours but at a continually decreasing rate. After release of prolonged pressure, interstitial oedema develops, the lymphatic and venous channels may become choked with tissue damage following. There is also a slowing of blood flow to that particular tissue part resulting in ischaemia. Pressure effects on tissues can induce pathological changes in the muscle which in turn impair its functional capacity. Histological evidence of muscle damage can be demonstrated in the tissue deep to human bed sores. This appears to be the result of prolonged pressure rather than infection and almost certainly precedes the surface manifestation of bed sores.

It has been reported (12) that in rats, low pressures maintained for long periods are more damaging to the tissues than high pressures for short periods; that is the duration of the pressure is more important than its intensity. For example, in leg muscle, a threshold of 100 mm Hg must be reached to produce definite microscopical changes and effects are similar to those produced by a pressure of 600 mm Hg for one hour.
It is therefore noted that there are two quite different time factors involved in tissue damage. One is the time required for impaired metabolic activity to produce cell death, the other is the time - dependence of tissue deformation with load.

Pressures relating to bony prominences may exceed 100mm Hg while the mean capillary pressure is approximately 25mm Hg. It is therefore to be expected that the distribution of pressures is determined in part by the distribution of bony prominences.

1. 3 Self-regulating protective mechanisms

The pain arising as a result of pressure applied to the surface of the body and the natural reaction of man to attempt to relieve pain by movements; are responsible for the lack of pressure sores in the normal person, which might otherwise occur during a night's sleep.

The following is a brief account of the coordination of perception of sensation (e.g. pressure) and the initiated movement to relieve this sensation.

The detector of cutaneous pressure is the pacinian corpuscle which is situated deep in the dermis layer of the skin. The Pacinian corpuscles are also situated in the neighbourhood of tendons and joints where they function as proprioceptors. The Pacinian corpuscles respond to deformation caused by firm pressure and are quickly adapting; that is they become used to the pressure if it is sustained for long periods and are no longer stimulated by the pressure.

Proprioceptors are the sense organs which are stimulated by movement of the body. They are important as ingoing afferent pathways in reflexes for adjusting posture and tone. General proprioceptors are found in skeletal muscles, tendons, and joints.

Proprioceptors, when stimulated by stretch or pressure, send impulses which travel from the trunk and limbs in the spinal nerves and are linked by a chain of 3 neurones with centres in the cerebellum or Parietal Lobe of the Cerebral Cortex. In the cerebral cortex conscious awareness of muscle and joint sense is appreciated; while in the cerebellum, maintenance of posture and sense of balance or equilibrium is non-consciously appreciated.

In addition, afferent impulses from Pacinian corpuscles relay to conscious levels signalling deep pressure. Impulses in thin myelinated and unmyelinated fibres give rise to conscious sensations of muscle pain.

Impulses received by the cerebellum may be sent to the cortex which then sends impulses down spinal nerves to the effector muscle. Alternatively the cerebellum may relay impulses down the spinal cord to the effector.

It is noted from the above that the nervous pathways are very important in reflex movements and voluntary movements.

If many skin receptors fail to establish central connections, skin sensitivity is reduced and areas of anaesthesia may be produced.

If the skin is anaesthetic without the patient being aware of it, a pressure sore may easily develop.

It has been reported that patients with insensitive feet, frequently present fresh pressure ulcers without undergoing any experience that would take him for outside his normal range of forces of walking. It has been postulated that this condition was 'tissue necrosis resulting from repetative moderate mechanical stress'; since the patient had in all cases taken more steps than usual in the
days preceding the ulcer. The authors suggest that the changes in the tissue are neither mechanical in nature nor are they primarily ischaemic. It is claimed that the necrosis may be due to enzymes from inflammatory cells, which are called in by one bout of mechanical stress, and then stimulated to spilt their enzymes by a repetition of the stress. It is suggested that a major difference between normal and insensitive feet may be that the latter are not able to signal the lowering of threshold to damage that accompanies inflammation, and to avoid breakdown patients could be tested for increased local temperature which accompanies inflammation.

In severe illnesses, particularly where powerful analgesics are being used, or in local diseases (especially in those causing anaesthesia for example paraplegia or neuropathies) the patient will accept disruptive forces due to pressure which a normal person will find intolerable.

It has been reported (9) that muscle spasm and repetitive movements causing repeated internal abrasion of fascia and subcutaneous tissue, ultimately lead to skin necrosis.

Diseases reducing monility such as paralysis, sensory disturbances and mental apathy; heavy sedation, anaesthesia, and coma, lead to lack of response to painful stimuli arising in the skin. (13)

Spinal cord injuries interrupt the protective mechanisms initiated by afferent and efferent nervous impulses that run in pathways in the spinal cord. In severe spinal cord lesions, sensibility is lost in the body below the spinal cord lesion. Therefore, such a patient is unable to appreciate those symptoms of blockage of circulation such as numbness, which normal subjects experience when part of the body is exposed to pressure for some time. The sensation of numbness represents the early signs of impending ischaemia and in the normal individual initiates change of posture. If the spinal injured patient is not moved regularly, ischaemia eventually results. (8)

1.4 Summary:

There appears to be two types of lesion.

(a) The Superficial Lesion, which is caused by extraneous irritants e.g. urine, sweat, chemicals. These irritants have a macerating effect on the skin and decrease its strength to resist the resist damage. In such cases the lesion is painful and shallow.

(b) The Pressure Sore which originates in the deeper tissues overlying a bony prominence and later extends upwards to the surface and sometimes down to underlying bone. It is caused by prolonged pressure; extensive and severe necrosis of deep tissues can be present while the skin shows only a dusky redness.

Pressure sores can be caused by either local or systemic factors.

Systemic factors include damage to the nervous system, protein deficiency hormonal levels, and anaemia.

Local factors include force and irritants. Systemic factors alone do not produce pressure sores. However, any alternation in tissue metabolism may contribute to pressure sore formation, by reducing the resistance of the tissue to damage by local factors. The major contributing factor to pressure sore is the immobility which allows local loads to act until the patient is deliberately moved.

The most probable cause of pressure sores is external pressure acting on a region
of the body for a period of some hours, which causes local tissue ischaemia in that region of the body.

Having considered the causes of pressure sores the following chapter examines means of preventing such sores.
Prevention of Pressure Sores

Three important factors in prevention of pressure sores are reduction of the time over which pressure acts, reduction of the magnitude of the external pressure and recognising "patients at risk".

2. 1 Patient assessment:
The impairment of the protective mechanism which prevents the damaging effect of sustained pressure on the tissues, is normally dependent on the adequacy of restless movements. Thus a reduction in the number of spontaneous movements is directly related to an increase in the incidence of pressure sores. In a study (14) it was reported that nine out of ten patients who moved less than twenty times during a seven hour period developed pressure sores.

It is clear that the prime factor leading to sustained pressure and the development of pressure sores, is impairment of mobility. It is important to determine how this is related to the patients clinical condition. This can be achieved by having a system for assessing the patients clinical condition.

Such a system exists (15) whereby points were scored in relation to the patients condition. The factors considered included, physical and mental condition, activity, mobility and incontinence. Points were awarded from 1 to 4 for each of these factors. This rating system appears to show a good correlation with a study of patients, who developed pressure sores. The study provides nurses with a reliable guide to detect patients at risk at an early stage, when preventive measures are most effective. The system was however misleading in cases with oedema of the sacral area and legs.

2. 2. Patient care:
The general nursing routine for preventing pressure sores in "risk" patients has been described by Jellis (1976) (16). It was suggested that; mattresses should be firm; patients should, wear cotton nightclothes to absorb sweat; patients position should be altered every two hours; the skin over pressure sites should be gently washed and the materials used should be non-irritant after which the skin should be dried thoroughly. It was also stated that diet is important in maintaining skin health; that walks should be undertaken for sedentary patients; and that Zinc and Castor Oil Cream should be applied after the washing of incontinent patients, as a water proofing agent.

2. 3. Mechanical Aids:
All the mechanical aids, that will be discussed below act to a greater or lesser extent by reducing the time over which pressure acts on any region of the body; and by reducing the magnitude of the external pressure which acts on local regions of the body such as bony prominencies.

When the body presses on a support, the support in turn presses on the body. This body-support interface pressure is not uniform over the area of contact. High peakpressures exist at the bony prominences such as the heels, the sacrum, spinous processes, scapulae and occiput. These are the regions where pressure sores occur most commonly.

The body-support interface pressure depends on the weight distribution of the body and the area of contact. The area of contact in turn depends on the size and shape of the relatively incompressable skeleton; the thickness and compressability of the overlying soft tissues and on the properties of the support material.
2. 31 The Ripple Mattress.
The Ripple mattress can supplement the two-hourly turning routine procedure. It is designed to decrease the time over which pressure acts on any given region of the body by alternately inflating and deflating regions of the mattress, at approximately four minute intervals.

2. 32 Fluid Beds (17, 18)
Fluid beds increase the area of contact between the body and the support media, thus lowering the average pressure on the body. More importantly they produce e.g. a more uniform distribution of pressure over the contact surface, thus decreasing the magnitude of peak pressures.

Theoretically, in the fluid bed the pressure over any horizontal plane is constant and peak pressures are avoided.

In practice this is not fully achieved because of the need to separate the body and the fluid by some form of sheeting.

The disadvantages of fluid beds are that they are heavy, expensive, require temperature regulation; create nursing problems due to the difficulty of manoeuvering patients; and create problems such as blood pooling due to posture assumed by the patient.

2. 33 Fluidised Air Beds (19, 20, 21)
The above principles of the Ripple Mattress and Fluid Bed are also applicable to the Fluidised Air Bed.

The bed consists of 21 moccroous sacs which are filled with heated air. The pressure in the groups of sacs can be adjusted to meet the requirements of the patients. The moccroous nature of the sacs allows perspiration to pass into the sacs, hence the patient is nursed directly on the nylon sections which are covered with a flannelette and are washable with sapon. It has been stated that provided the beds are operated correctly, pressure areas do not need attention and that pressure sores do not develop on the bed fast patient.

Two other special support beds are Sand Beds (22) and Lud Beds (23).

2. 34. Cushions and Pads
Ideally, pads should "wrap around" and support the anatomy from all sides, giving as much hydrostatic loading as possible. Hydrostatic loading avoids tissue shear strains, which tend to collapse capillaries and promote ulceration.

The outer coverings (membrane materials) of the pads include various plastic films, fabrics and fleece materials. These materials influence the stress state inside the body tissues in two ways. Firstly, the membrane contacting the body produces friction or interface shear on the skin. Secondly, the membrane produces a "hammock effect" which changes the pressure distribution property of the cushion. In a fluid cushion (liquid or gas contained within a membrane) both friction and hammock effects are dominating factors in the cushion's behaviour. In the case of semi-solid or viscoelastic cushions, the importance of the membrane materials in the whole system performance decreases.

The two properties of a membrane which determine it's influence on the stress and strain state of body tissue are stiffness and the coefficient of friction. The lower the membrane stiffness the less is the hammock effect. It is this effect that tends to produce a taut of the padding material to "wrap round". The lower the coefficient of friction, the lower the distortion-producing frictional shear stresses at the skin surface.

Further the presence of moisture on the membrane results in a sharp rise in the coefficient of friction. The following types of pads and cushions can be used in the prevention of pressure sores.
2. 341 **Gels**: Synthetic gel cushions are designed to imitate fatty tissue and redistribute the pressure over its area.

2. 342: **Viscous Particulates**: Such as feather mattresses and pillows are effective supports since their filling consists of randomly arranged anisometric particles. Thus when uniaxial pressure is applied some feathers slip sideways while others will be locked together, thus accommodating the load imposed. However frequent "puffing" is required.

2. 343: **Foams**
Foams do not distribute pressure as well as a homogenous gel

Resilient polyether foams have a slightly delayed elastic recovery and a low 'compression set' but are good materials for cushioning and are specified for hospital mattresses. They however must be replaced after a few years use due to deformity.

Debilitated patients tend to slip forward when upright for long periods; in doing so they concentrate much of their weight in shearing stresses particularly over the sacrum. Bolsters were once used under the thighs to prevent the 'forward slide'. They were however inclined to anchor the patient in one position and cause pressure distortion on a relatively small part of the thighs. The bolster has appeared to cause venous thrombosis and has been consequently prohibited.

A 'gutted' bed bends the mattress to fit the shape of a patient. Hips and legs are flexed, thus distributing pressure evenly along the thighs and relieving concentrations of sacral stress.

The draw sheet - mackintosh combination, used on beds of incontinent patients, easily crumples under the forces produced by the forward slide effect while creased sheets produce ridges of concentrated pressure

The Marathon Dupic incontinence pad has a gel centre and a towelling cover allowing the surface to dry quickly. The time when friction coefficient is high (i.e. when wet) is therefore less than with other under pads.

2. 344 **Air Pads and Cushions**
Air enclosed inside an imperious envelope forms a cushion which is inexpensive and easily handled, but may be unstable with some patients. There is also the possibility of a leak and/or under inflation.

2. 345 **Water Pads and Cushions**
Such cushions are heavier than air, and tend to be unstable and may also leak.

Some pressure equilisiation occurs but the support is by hammocking of the cover which gives little control of pressure or shear.

2. 346 **Fluid/Foam**
Support from such pads and cushions is caused by the hammocking due to hydrostatic pressure in the sides of the cushions. It is not caused by the foam insert, which may have little resistance to indentation. The cushions are heavy and difficult to handle.

In the "liquid filled polymer microcell sponge" that is fluid enclosed in a closed-cell construction, the pressure distribution is reasonably effective
and shear is reduced. However they too are heavy and their ‘floopiness’ makes them difficult to handle.

2. 347 Elastic Foam Cushions:
These cushions employ rubber, synthetic rubber or plastic foams that have minimal viscous characteristics. The density of the foams can be altered to produce a different feel. For example how density foams have a “soft feel” property and are preferred by patients with intact sensation.

These foams however loose their elasticity with age and become deformed. Thick foam cushions may often be improved by carefully designed cut-outs to relieve high pressure areas which can tolerate extended time – pressure.

2. 348: Visco - Elastic Foam Cushions:
This type of cushion provides more stability than a purely elastic cushion.

Once a viscoelastic cushion has conformed to a patient it will tend to retain its shape during small movements of the patient. Hence, pressure relief occurs momentarily if the patient pulls away from a point of support, thus permitting adjustment of position without shear and with momentary increase in local circulation. This effect is absent in a purely elastic cushion which maintains pressure against the skin during small movements.

2. 349 Bean - Bags:
These are cloth envelopes filled with dry macrospheres. In principle this cushion will assume a conforming configuration during use which relieves high pressure areas and distributes pressure to other soft tissue areas. However in practice it appears to be unsatisfactory.

2. 350 Solid Body - Shared Seats:
A vacuum is employed to set macrospheres in a bag to produce an impression of the patient’s thighs buttocks and back. A positive mould is made of this impression, which is then used as a form for a fibre glass or vacuum - moulded plastic seat. This seat may be lined with a relatively thin layer of plastic foam. Good results have been reported and the chair is light and portable although the cost is high.

2. 351 Gels:
Silicone and vinyl gels are enclosed in latex rubber or fabric covers. The gel is elastic non - friable, non - porous and semi – solid having a hydrostatic property. It is therefore easily capable of flowing laterally under pressure and returning to its original shape when external pressure is removed. Gel pads are rather heavy and are more expensive that plastic foam cushions.

Sustained pressures on ischii should be kept to below 30mm Hg, on posterior trochanters to below 60mm Hg, while thighs can readily accommodate pressures of up to 100mm Hg.

The patient should, if possible, be made aware of the problems associated with tissue breakdown and should be encouraged to do all he can to avoid such tissue break down.

Once a pressure sore has developed, it requires considerable time to heal and may involve several techniques described in the following chapter.
There are a number of signs which indicate the irritation of a pressure sore. The affected area is initially swollen, hard, usually anaesthetic and situated over a bony prominence. Pressure has usually been applied for four to six hours.

Within approximately 24 hours the patient's temperature often rises to 103°F and redness appears over the swelling. This redness turns to blackness of the skin when frank necrosis is indicated. The necrosis and slough then slowly separate down to the bony prominence.

Following segregation of the sloughs the constitutional disturbances subside.

Treatment will be started when the first signs occur, and includes general medical and supportive treatment and local dressings.

3. 1 Medical Treatment:

3. 11 Diet:
The diet will usually be altered to a high calorie and high protein foods, to provide the higher levels of nutrients and metabolic activity necessary for tissue regeneration.

Ascorbic acid may be necessary in the wound healing process for the regeneration of collagen fibres and the development of new capillaries. Thus ascorbic acid intake may supplement the patient's diet. Any state of anaemia must be corrected, since the presence of anaemia decreases healing potential and so lengthen healing time. Consequently

3. 2 Supportive Treatment:

If the skin is red, the area should be carefully washed, dried and exposed to the air. It is important that no more mechanical damage occurs during the process of washing and drying. Since the tissues are already very fragile, further breakdown could occur from this process.

A very thin coating of a barrier cream, such as vasogen silcone, may be applied to protect the area from water, irritants and body exudates.

The use of topical antibiotics is rarely indicated in treatment since they may be ineffective, cause damage to the skin, retard ulcer healing and increase risk of resistant organisms developing.

If pus formation is evident, a swab of the affected area should be taken and the causative organisms identified. Systemic anti infective therapy should then be initiated according to the organisms sensitivity to an anti infective.

If the skin is broken, the sore may be washed out with an antiseptic solution such as hydrogen peroxide, a strength eusol or chlorhexidine 0.05%. The ulcer may then be packed with a dressing, impregnated with one of these solutions. Eusol helps to separate sloughs, while hypochlorite solutions (milton) stimulate granulating tissue. Strong antiseptics must not be used as this will lead to increase tissue destruction. The ulcer may then be covered with a dressing.

In the past, the established dressings have been wool, Gamgee tissue and crepe. However, newer types of dressings include the semi-permeable membrane dressings such as Micropore (26) Stomahesive(27), and Opsite. These semi permeable
dressings provide a good environment for development of granulating tissue by decluding body exudates and macro organisms whilst allowing air into the wound.

There are three factors that most drastically affect the pattern, speed and quality of healing; first the dehydration of exposed tissue; second, the status of the blood supply bringing oxygen and nutrients to the area; and third, sepsis.

Wounds exposed to the air lose water vapour and so the upper dermis dries and healing takes place beneath a dry scab. Covering a wound with an occlusive dressing prevents scab formation and radically alters the pattern of epidermal wound healing. Blowing on wounds creates a scab within three hours instead of the normal twenty-four, but more tissue is sacrificed in the process. Such treatment can only be justified if it is considered rapid scab formation would significantly reduce the incidence of severe infection. However, chemically dried air has been successful in the treatment of pressure sore (26)

Wound surfaces are relatively anoxic because of damage to the blood supply and stasis in the extracellular fluid compartment. Also, the inflammatory cells present in very large numbers in a chronic infected wound, rapidly utilise oxygen and reduce the amount of oxygen available for regenerative activities. Treatment is directed towards re-establishing a suitable environment for epithelialisation and connectivetissue regeneration. When dealing with indolent wounds bordered by a zone of infected necrotic tissue the minimum requirements for such regeneration are: a moist, protein rich medium into which the cells can move, and a supply of oxygen and glucose molecules. Although leucocytes can injest bacteria in conditions of hypoxia, they cannot kill them. Macrophages, required to clear away the debris of necrotic tissue, are avid users of oxygen. Fibroblasts cannot function efficiently at low oxygen tensions. These specialised cells are required in the healing process. Through their action, in the initial period of healing, the necrotic tissue becomes macerated and the wound appears extremely unsightly. However, on approximately the sixth day, the wound clears dramatically. This is because the microporous dressings maintain a moist atmosphere at the tissue surface and allows diffusion of oxygen while preventing re-infection; thus providing favourable conditions for the leucocytes and macrophages to cleanse the wound. The cleaning process is aided by the frequent removal of dressings and adhering tissue breakdown products. Covering wounds with films of low oxygen permeability delays epithelisation. Covering wounds with ointments is equivalent to using an occlusive dressing. Some ointments may become incorporated into the wound and cause undesirable tissue reactions.

Reston, a polyurethane foam dressing has also been successfully used both in the prevention and treatment of pressure sore (29, 30, 31).

Treatment of pressure sores by ultra sound has also been reported (32). The mode of action of healing is thought to be due to a micro massage obtained within the tissues together with vasoconstriction and vasodilation and an increase in oxygen absorption. Although insufficient patients have been treated with this method to allow serious conclusions to be drawn, the results have been encouraging.

Ultraviolet and infra red rays have also been employed in treating pressure sores. Both act by causing an erythematous condition by vasodilation though ultraviolet irradiation produces a more lasting erythemia.
3. **Surgical Treatment:**
In some cases of chronic ulceration, surgical treatment may be necessary; for example, amputation may be necessary for ulceration of the foot. In less serious cases, surgical treatment may aim to remove the hard bony-prominence deep in the sore; it may also aim to bring into the ulcerated area, good skin and fat to act as a viable cushion.
Conclusion

Over the years, decubitus ulcers have been a severe problem to those entrusted with the care of patients.

Decubitus ulcers are difficult to heal and necessitate prolonged treatment, thus delaying vital rehabilitative measures.

Various methods for the prevention and treatment of pressure sores have been discussed. Research and clinical trials continue to develop newer methods. Each new method, whether mechanical or chemical, results initially in accelerated healing. However, many are eventually discarded because of an inability to sustain performance. Any clinical trial of a new system must take into account the increased enthusiasm and stimulus to the nursing routine which may play a very great 'placebo' effect.

Practical attempts to reduce the pressure sore problem centre mainly on the design of equipment to reduce pressures or to distribute pressure more evenly over the whole body. Such mechanical solutions include, water beds, sand beds, and mud beds. However effective they may be, they have not been adopted except in minimal number of clinical situations. The impracticality of the weight of such support beds has not, as yet, been overcome. Incidental costs are likely to arise from the need for such beds to be located either at ground level or on specially strengthened floors. Consequently, there is as yet, no possibility of equipping a 60 bed geriatric unit with such support beds. Thus complex and expensive solutions are viable only in highly specialised clinical contexts. There is no simple mechanical solution. Nurses must concentrate, as much as ever, on the need for close and careful personal nursing attention. It is also vital to pursue the early recognition of patients at risk to pressure sores.

Perhaps there is a lesson to be learned in the Chinese saying that the incidence of pressure sores acquired in hospital is twice that acquired at home.

Examination should be given to the regimen of pulling sheets taut over the supporting mattress, followed by the tucking of sheets into the side of the bed so that the patients cannot move. It is the author's opinion that such a policy should be changed for the reasons mentioned in this essay. It should be realised that all patients do not require the same level of nursing care. Patients in the home feel more inclined to get up and walk to the toilet without fear of being reprimanded for upsetting a newly made bed.

One example of this regimented routine concerns elderly ladies detained in hospital for the pinning of a fractured hip. It was found, with little variation that all had been taken from their beds and put into an armchair beside their beds for breakfast. The patients remained in their chairs until 6.00 a.m. when they were returned to bed. One patient said, "The afternoon, sitting in this chair is interminable and I get to the point where I could scream and throw myself on the floor." (33).

It appears that the encouragement for movement and activity, both physical and mental is likely to decrease the pressure sore problem, particularly amongst patients who are confined to a chair or to their beds.
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