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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  
351 SAUCHIEHALL STREET  
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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.

The Etiology of Pressure Sores

## Chapter 1

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) Synonymous 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.

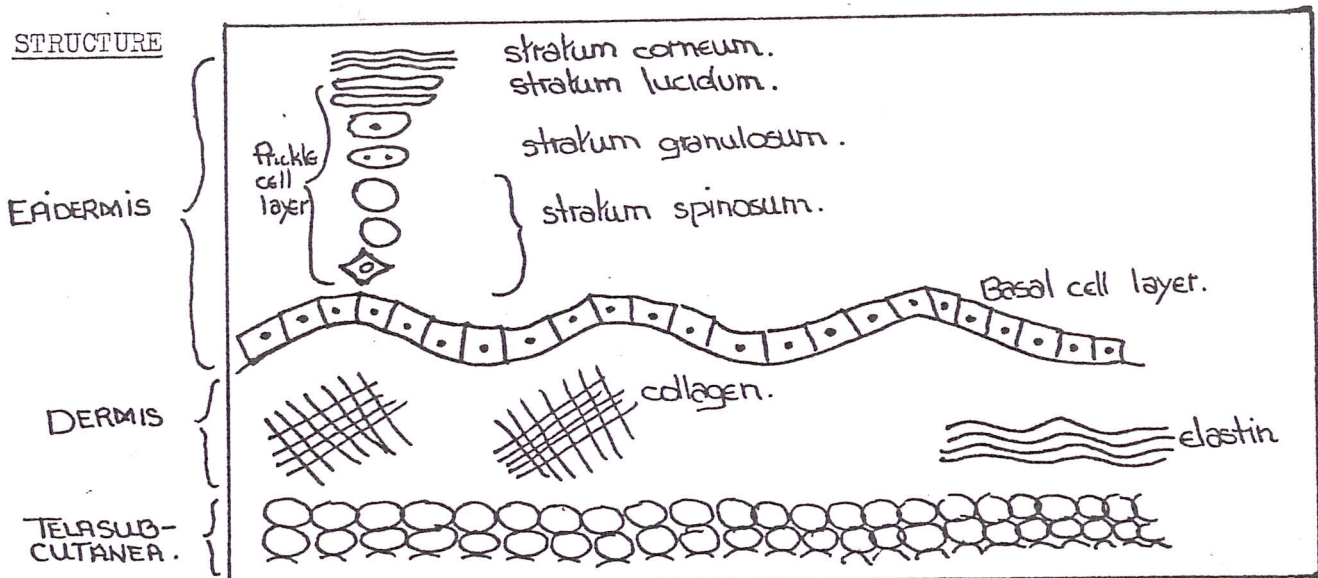
1. 1.1. The Structure of the Skin:

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 slacticity 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 feed back 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.

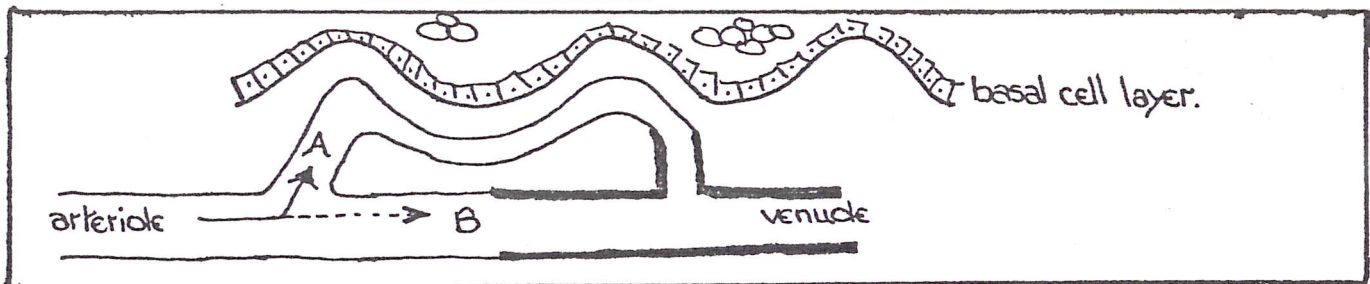
Shearing forces applied to the skin can be a factor in bed-sore formation. For example, when a patient slips down a bed the superficial layers of skin move across the deeper fixed layers causing blood vessels to be subjected to pressure, kinking and rupture (5)

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 ( $\Rightarrow$  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.

#### 1. 12. Skin in temperature regulation



The arrow indicates direction of blood flow.

Figure 2 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~~ arteriole and venule called an Arteriovenous Anastomosis.

If skin is cold, a vasoconstrictor reflex mediated by  $\alpha$ -adreno-receptors at the afferent arterioles is illicited 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 erytherma 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 its 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<sub>4</sub>(=), 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 + 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 food 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. 21 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 32mm Hg at the arterial end of the capillary bed and drops to 15mm Hg at the venous end of the bed.

Interstitial fluid pressure ( $\approx -7$  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 25mm Hg, on the membrane which opposes the driving force described above. Thus the net driving force across the capillary membrane is 14mm Hg.

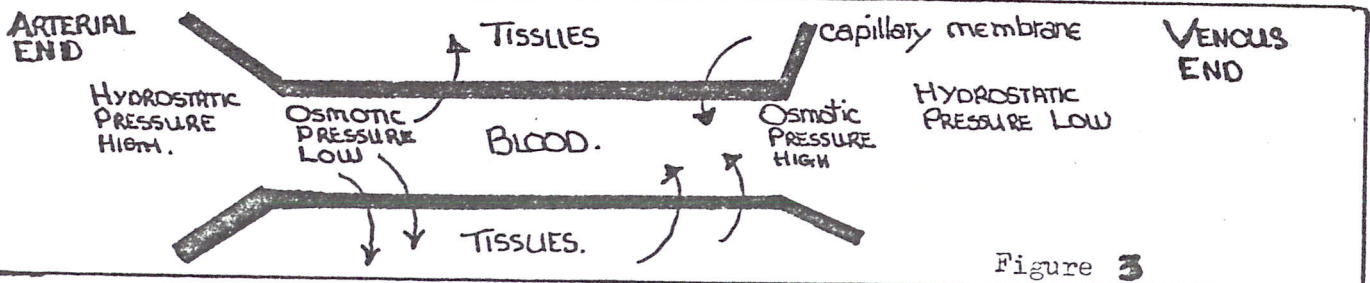


Figure 3

EFFECT OF FORCES ON CAPILLARY (TISSUE SURFACE) FLOW

The arrows indicate direction of flow