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Decubitus ulcer : a manifestation of pressure : with comments on prevention and therapy

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THE DECUBITUS ULCER--A MANIFESTATION OF PRESSURE: WITH COMMENTS ON PREVENTION AND THERAPY

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Introduction: The Decubitus ulcer is in many ways a product of hospitalization for it is in this setting that this entity has its origin. It is a complication and never a primary disease; a complication which theoretically can be 100% prevented by an absolute of patient care and attention to details. If on the other hand this care is deficient and the decubitus ulcer is left to run a natural course, it can and will consume the patient and may be the final stage in a patients death--in fact it is a major cause of death in the hospitalized patient who fulfills the requirement of a decubitus diathesis.(43) The paraplegic represents one part of the hospital population who is readily prone to develop a decubitus ulcer, and there are multiple reports which show this lesion to be at the lowest in civilian paraplegics at 28%,(48), and its highest in the paraplegics of World War II with figures ranging from 57%-75%-85%-95%.(18, 36, 41, 49, 50). What this represents can best be shown by a recent report from a rehabilitation center with 30 patients with decubitus ulcers. These patients lost 4,874 days or 13.3 patient years of active rehabilitation because of decubitus ulcers.(24) It would be unfortunate indeed to think our rehabilitation centers, with their vital needs, would have to disrupt their active process of retraining to treat indolent and often unnecessary lesions because of a lack of understanding by the initial attending physicians. Another report demonstrates that patients who have been ulcer free for long periods often rapidly develop this lesion on readmission to the hospital.(31) What this

means, is that the physician and attending hospital staff are as responsible for this lesion as they are for resistant infections, post-operative pulmonary embolism and the host of nosocomic diseases resulting from modern hospitals.

The Lesion: The word decubitus is derived from the Latin word for lying. This is no longer the only method in which this ulcer forms; for in the paraplegic the ischial lesion from prolonged periods in a wheel chair is more common. For this reason many have preferred to name the lesion by its mechanism-pressure, hence the pressure sore. I have no argument with either, and they will be used interchangeably in this paper.

The decubitus ulcer or pressure sore is unique in many ways from other ulcers, but specifically, it is a localized area of cellular necrosis. As the ulcer is caused by pressure which is transmitted evenly and equally between the external surface and bone below, all layers are involved in the cellular damage.(19) This means that a fully developed decubitus ulcer will result in an area of necrosis and eventual slough of the skin, subcutaneous tissues, fascia and muscle.(7) A simple ulcer is generally considered to be any loss of continuity of an internal or external skin surface--a much less complex problem.

The location of decubitus lesions depends in many ways on the type of hospital population that is being dealt with. Every author

presents his statistics on the location and frequency of pressure sores, while in truth, individual statistics represent individual cases and standards of care. The location of pressure sores depends on the bony conformity of the skeleton and the position of the patient. One can easily see how in a ward with paraplegics in wheelchairs the ischial ulcer would predominate, while in a ward of aged and debilitated at constant bed rest on their back with respiratory difficulty when on their sides and abdomen, the sacral lesion would predominate. A general outline for location by position is presented below:(49)

Supine:	heels, sacrum, posterior iliac crests, scapula, occiput
Prone:	knees, anterior iliac crests, elbows (when used for support)
Lateral:	lateral aspect distal and proximal fibula greater trochanter
Sitting:	(at 45°) sacrum
Sitting:	(upright) ischial tuberosities, elbows
Adductor Spasm:	medial aspect of knees

This readily demonstrates that position and skeletal conformity dictate the location of pressure sores. The type of skeletal projection creating pressure will influence the type of ulcer which is formed. The sacral ulcer caused by a large flat bony plate, produces a wide mouthed shallow lesion; while the ischial tuberosities or trochanter, which produce pressure through a small point, produce small mouthed widely undermined lesions. The heels and knees produce lesions somewhere between these extremes. Also, the ischial and trochanteric lesions can produce adventitial bursae which make them extremely refractory to therapy.(49)

These lesions by their nature have large amounts of necrotic tissue--this means excellent culture media for bacteria. Any open wound is necessarily contaminated, decubitus ulcers are no exception. The contaminants are always mixed groups of organisms, unless there is extensive infection by any single organism.(39)

The organism most frequently associated with pressure sores are S. aureus (coagulation^{+}, hemolytic^{+}), Proteus vulgaris, E. coli, Aerobactor aerogenes, Pseudomonas areugenosa, B. hemolytic Streptococcus.(2,9,20,39) The organisms characteristically are those found in feces or any urinary tract infection.(8) As most of these lesions extend to the bone, osteomyelitis may be a frequent accompaniment in the neglected lesion. In one study of 334 patients with decubitus ulcers some form of bone involvement including simple degeneration fibrosis due to bacterial infection was found in 51.8% frank ostéitis or osteomyelitis in 24.1%, and no bone involvement in only 24.1%.(7)

A very important aspect of the pressure sore arises from the fact it does represent a loss of continuity of skin surface and thereby a loss of the protective mechanism of skin. Besides being a portal for the entry of infection, it becomes a portal for the exit of vital nutrients and fluids. Reports in the literature demonstrate measured losses of from 5-50 grams of protein per day from these lesions.(2,8,54) It is a generally agreed fact that these wounds do not heal in the face of negative nitrogen balance and hypoproteinemia; this readily points out the significance of

these protein losses. The meaning of this type of protein loss in an already malnourished and inanated individual is devastating.

There are three basic stages in the formation of a decubitus ulcer, and they have great clinical importance. (after Guttman-2) The differentiation between stage I and II is the most vital in the early stages.

I. Stage of transient circulatory disturbance: At this stage the pressure was sufficient only to cause transient erythema. This condition is reversible and promptly disappears if pressure is relieved. One can determine if the lesion remains in this reversible phase by pressing on the erythematous area, for if this blanches, the vessels are patent and necrosis has not yet occurred.(2,8,49) Capillary thrombosis in the presence of necrosis manifests itself by lack of blanching with pressure.

II. Stage of permanent Damage: This stage can present itself in four distinct patterns; all marked by inability to blanch and the inevitability of ulcer formation.

(1) Erythema and congestion of the skin which does not disappear after decompression but leads to induration and discoloration of the skin.

(2) Superficial layers of skin excoriated exposing the exudating cutis vera. This type resembles an ordinary abrasion.

(3) Dead epithelium remains intact; it is raised by exudation from the cells of the cutis underneath and a blister develops which may be dark in color if hemorrhage has occurred.

(4) Deep layer of skin involved resulting in necrosis and formation of an ulcer. The border of such an ulcer becomes pigmented.

III. Stage of deep Penetrating Necrosis: This involves the subcutaneous tissue, fascia, muscle and bone. This leads to gangrene (dry), and once the slough is removed, a deeply formed ulcer.

There is one other broad classification which can be added to the previous set of lesions: that is the infected lesion. There is a great difference between the contaminated lesion and the infected lesion, only the organisms are similar. With the exception of urinary infection, this is the most common source of sepsis and death in the paraplegic.(37) The infection is mixed and will extend to the bone causing periostitis, osteitis, and

osteomyelitis. As was previously noted, ischial ulcers have small external openings and widely undermined areas. In sepsis of these ulcers there are three distinct pathways these infections take.

1. Along the ischial rami, lateral to the hip.
2. Down along the posterior fascial compartment. These may go as far as the knee in an ambulatory patient.
3. Forward into the groin(37)

Pathologically these lesions demonstrate just what one would expect; much necrotic tissue, venous thrombosis, engorged venules and thickened arterioles.(37)

There are some cases where malignant lesions are found in the ulcers, but there is little question that they are casual and not causal in relation. The decubitus ulcer can be an extremely chronic lesion and there is one report of a lesion spanning a 31 year period.(17) The incidence of amyloidosis in these lesions is unquestionably related, as amyloidosis is found in chronic debilitating diseases. This has not been a frequent association.

The developed, older lesion (which will be discussed later) represents a highly vascular bed of granulation tissue, and therefore there is a marked tendency for this lesion to bleed. (12) This is so marked, some consider it a contra-indication to surgical debridement. (55)

Pressure sores have an interesting characteristic in that they are painless. Even with extensive necrosis, they produce

practically no sensation. This is due to pressure atrophy of nerves and removes one of the most important sensations man has: pain. With this behavior, once the ulcer forms, it silently grows.(32)

Etiology: In considering the etiology of the decubitus ulcer, it is important to realize there is only one primary cause and that is pressure. The pressure must only be of supracapillary proportions sufficient to cause local circulatory arrest and maintained over adequate time for the deleterious effects of ischemia to occur. I do not believe this statement is an oversimplification in any way, and this is an important premise with which to begin. However, it remains true that the pressure sore occurs in distinct hospital populations, and it is this variation in susceptibility which is the overriding consideration in a closer look into this problem. The importance of secondary or contributing factors in the formation of these lesions must be the initial warning if the pressure sore is to be prevented.

If there is a single primary cause; this must necessarily be able to produce a pressure sore in the absence of any other pathology or contributing factors. Also, supracapillary pressures must be demonstrated to exist in the situations which have led to the formation of the pressure sore. Finally, the demonstration that ischemia rather than direct cellular damage must be demonstrated as the cause.

The pressure sore in the healthy individual is no stranger to the orthopedic surgeon who takes all precautions in the application of a cast for correction or immobilization to prevent pressure areas which he sees all too often cause an ischemic lesion--whether

it be due to something as gross as an overall too snug application, swelling beneath the cast, or as simple as the impression in the cast of his assistant's thumb--all have caused ulceration.(13) Ulceration has been noted in newborns at the site of forceps from difficult deliveries, and in one amusing situation, the occiput of a woman following a post-operative course for retinal detachment who obeyed the surgeon with "noteworthy obedience and resignation" when he ordered her to remain very still.(55) Also the pressure sore has been reproduced experimentally in animals; although these were the basis for other problems, they reproduced identical pathology with only pressure.(41,46) In truth this is making the problem more difficult than it is, for the necrotic result of mere arterial ligation is no different than the necrosis of tissue from pressure ischemia.

Normal pressures of cutaneous circulation have been demonstrated to be 32mm Hg at the arteriolar limb, 20mm Hg at the midcapillary area, and 12mm Hg at the venous limb; this rises to approximately 60mm Hg in the mid-capillary area during reactive hyperemia.(42) Other figures are 20-40mm Hg in the capillary, rising to 35-45 mm Hg with reactive hyperemia and 70-80mm Hg in response to the local application of heat. In numerous experiments the pressure necessary to cause circulation arrest is considered to be 50-60mm Hg.(30,33,37) What are the pressures the hospitalized patient is the subjected to? We assume the time duration will be sufficient; although this

can obviously be prevented. On studies of various surfaces with the patient recumbant on a hard contoured surface, pressures were consistently over 300mm Hg and on a hard flat surface, pressures up to 700mm Hg were recorded.(42) With the patient sitting, as in wheel chair, pressure over each ischial tuberosity was recorded as 300mm Hg.(46) The recorded ischial pressures were reduced to 160mm Hg with the addition of 2" of foam rubber padding.(46) This would be a comparable pressure reduction in the patient in bed; however, with factors such as folded creases in sheets and lack of absolute care, the pressures can soar to much greater levels. This obviously fulfills the requirement of supracapillary pressures in the everyday routine of the hospitalized patient. "Normal cellular metabolism is dependent on the receipt of nutrients and elimination of metabolites; any condition which interferes with this exchange will effect the function of the cell. The circulation of peripheral blood fulfills the metabolic needs of the cells, so that any alteration in the circulation is reflected in cellular change. Severe or prolonged circulatory interference will ultimately lead to death of the cell."(43) This erudite description of cellular needs clearly points to the effect of supracapillary pressures.

Does this pressure act secondarily by local circulation arrest, or does it cause cell death by direct damage to the cell? Experiments have shown the cell itself is extremely resistent to the effects of great pressure, and measurement recorded at 250,000

pounds per square inch have not caused cell death.(43) This pressure when reduced to the amount acting on each cell is certainly much less, but nevertheless helps demonstrate that the effect of pressure truly is the obliteration of vascular channels.

We have shown that pressure alone can and does cause ischemic necrosis in the face of a cell resistant itself to pressure, and that the pressures necessary to accomplish this are present in the hospitalized patient.

Perhaps the greatest variable and the most controllable is the time over which the supravital pressures are allowed to exert their influence. This is probably best demonstrated by the foot in walking which although subjected to great pressures, certainly does not develop necrosis on the planter aspect; the reason being that the pressure acts over extremely short periods of time. Two hours is a rather generally agreed upon time period for producing enough cellular damage to result in a necrotic slough of tissue. In one experiment, the aspect of time is sharply demonstrated for pressures of 240mm Hg alternating over 2 hours never produced tissue change while pressures of 70mm Hg acting over 2 hours consistantly produced tissue damage.(43) Once supracapillary pressures are reached, they will cause tissue necrosis if allowed to act over sufficient time. In the rabbit ear the lowest pressure which would eventually cause necrosis would do so at 13 hours.(46) The time factor is further evidence that ischemia rather than cellular damage is the cause of pressure sores.

There is another important aspect of pressure over which general agreement does not exist, and this is the residual effect of great pressures--in the range of 600+mm Hg. The arterial walls are perhaps the least resistant to pressure and cellular infiltration with the presence of edema appearing within 24 hours of pressure not involving general tissue necrosis demonstrates this. The point of disagreement involves the importance of venous thrombosis after the application of pressure. In some experiments this was not noted and was not considered a factor.(13,46) The more commonly subscribed to theory states that intense pressure for a short time is responsible not only for the interference and complete cessation of capillary circulation, but also changes in the large vessels with the result that venous thrombosis may be found. The presence of venous thrombosis then interferes with the reactive hyperemia mechanism (see below) after the pressure is removed with the result that the tissue continues to remain ischemic and will eventually lead to the inevitable formation of a pressure ulcer. (41,43) The experimental findings that 100mm Hg acting over 2 hours equaled 600mm Hg acting over 1 hour is important in the concept of vascular change due to thrombosis.(30) If mere circulatory arrest were the only factor here, once supracapillary pressures were reached, there would be little difference in ultimate time to produce necrosis. On the other hand, if a short period of high pressure produced necrosis, we must assume vascular obstruction remained

which maintained ischemia and resulted in eventual cell death. This may be an extremely important fact in patients subjected to great pressures as on the orthopedic table or the thoughtless turning of a patient to change the sheets.

An extremely important component and perhaps one of the most frequently overlooked is the shearing force. If a nurse cranks up a patient's bed (i.e. so that a paraplegic may see about him) greater compressive force is placed on the posterior sacral tissue. As added weight is referred through the vertibral column, there is in addition to increased compressive force a shearing factor or force exerted upon posterior sacral tissue. This shearing force becomes a factor when the head of the bed is raised. When the torso slides down, it "transmits force to the sacrum and firmly attached deep fascia. Posterior sacral skin stays in the same place because of friction of the bed and skin. The interface between skin and superficial fascia is interlocking and unyielding. The deeper portion of superficial fascia is rather loose and mobile in the sacral region therefore, the effect of a shearing force is concentrated in the deeper portions of the superficial fascia."(52) The actual obstruction and severing of vessels may be another problem due to this force, for the blood supply to the posterior sacral area is from the posterior branches of the lateral sacral arteries and superficial branches of the lateral sacral arteries and superficial branches of the superior gluteal arteries--the former pass through the posterior sacral foramina to supply the local muscles and then pierce the deep

fascia to supply the superficial fascia and skin. A shearing force in the deeper portion of the superficial fascia places the blood supply in this area on a stretch, and perhaps angulation, as they are more or less anchored at their point of perforation through the deep fascia. It can easily be seen how this can cause ischemia.(52) If the patient is on a circle bed or if the bed is lifted (not merely angled up) this force can be prevented by use of foot boards--but it must be remembered the feet are subjected to ischemia.

The mechanism of reactive hyperemia which has been alluded to previously is the result of retained metabolites due to vascular stasis and is a purely local phenomenon and therefore present even in the paraplegic. Studies have shown that it can nearly double capillary pressure and therefore is one of the most important mechanisms of body defense.(42,48) Therefore, it is not physiologic capillary pressure, but the pressure of reactive hyperemia which must be overcome. This is why pressures of 20-40mm Hg do not cause necrosis.

If we have pressure, we must have a pressure point or prominatory where the pressure becomes magnified and can reach supercapillary pressure. It is the skeletal structure of man that provides these points; without them weight would be distributed evenly enough to prevent the pressure sore. The skeletal promintories dictate the site of pressure sores. Natural protections against this are subcutaneous fat, localized fat pads, and muscle tissue. These work

by redistributing weight; however fat is a two-edged sword, for if a person becomes inanated with a severe weight loss, he loses this distributing factor; on the other hand, if he gains excessive weight his surface area lags behind weight gain and pressure on any individual area is increased.(46) Therefore any variation in fat content can be considered an etiologic factor in the formation of decubitus ulcers.

Another mechanical factor is the malleability of the surface on which the patient lies. If the surface is hard and unyielding, small skeletal points will support great weights and produces pressure sores more easily. If the surface is yielding, it will conform to the body, distribute the weight and produce smaller absolute pressure areas.

Let us take a closer look at the hospital population and clinical variants that demonstrate this lesion with increased frequency. The literature is replete with articles contributing their formation to such a host of causes (anemia, dehydration, shock, paraplegia, incontinance, diabetes, arteriosclerosis, vasculitis, infection, debility, neglect, sensory loss, alcoholism, dulled sensibility, malnutrition, hypoproteinemia, hypovitaminosis) that one is forced to speak rather in generalities than with regards to most of these as specific entities. It is important to remember that pressure remains the primary cause and these other factors are contributing in nature.

Vascular Insufficiency: The pressure sore is a manifestation of ischemia; therefore, if a pre-existing vascular insufficiency

is present, pressure can more easily create a degree of ischemia which will result in tissue necrosis. This has been demonstrated experimentally with animals who were subjected to pressure before and after vessel ligation. In the animals with vascular insufficiency, 50mm Hg acting over 1 hour paralleled 100mm Hg for 2 hours in the normal animal. This helps explain the more common clinical occurance of pressure sores in the arteriosclerotic or other general conditions of vessel change as the arteriosclerosis of diabetes and the vasculitis of the collagen diseases as rheumatoid arthritis, L. E., polyarteritis and scleroderma.(43)

Tissue Edema: Tissue edema which may be a common finding in the patient with incipient congestive failure or hypoproteinemia, and which is generally more pronounced in the sacral area may aid in the development of pressure sores. The rate of diffusion of oxygen and metabolites from the capillary to the cell decreases in direct proportion to the distance that remains between each other. As tissue edema increases this distance, its profound effect on cellular metabolism can clearly be seen.

Debility and Coma: Perhaps the greatest protection against the formation of decubitus ulcers is the discomfort attending prolonged pressure. This discomfort results in position change and relief from the prolonged effects of pressure. In the very old, debilitated, alcoholic or comatose patient, there is either apathy toward this discomfort or lack of sufficient stimulus or strength to cause re-

adjustment of weight. This leads to prolonged single posturing and the resultant prolonged ischemia in vulnerable areas leading to the inevitable pressure sore. Spontaneous movement during sleep is one of the best protective mechanisms. In studies measuring this electronically, patients with considerable amounts of spontaneous nocturnal movements did not develop pressure sores, while those who moved very little were subjected to pressure sores.(30)

Paraplegia: Perhaps the most complex and far reaching problem in long term hospitalization is the paraplegic (or other cord lesions). In the paraplegic one may be dealing with a younger hospital population which is mentally alert and faces a potentially long and productive life after his hospitalization and rehabilitation. Almost every causative factor in the production of the pressure sore will eventually act on the paraplegic patient, and I will therefore use this example to expand other causative factors.

Much has been written of a neurotrophic factor in the formation of decubitus ulcers. This concept was originated by Charcot in 1879 who postulated a metabolic factor and gross alteration in physiology below the level of the cord lesion.(19,54) In truth, there is little basic evidence to support this theory of a neurotrophic factor and a multiplicity of rational explanations exist for the frequency with which this lesion is demonstrated in cord injuries.(12,43,46,54) Total anesthesia below the lesion is unquestionably the most important factor; for there is no discomfort attendant continuous pressure.

One author goes as far as to describe a reflex initiated by the discomfort of pressure and leading to weight shifting and protection. (57) Perhaps I would not go as far as to state it is the disruption of so complex a reflex, but this certainly does describe the protective mechanism of spontaneous, unconscious movement due to the discomfort of pressure. Anesthesia is perhaps the major component. Further evidence that anesthesia is the main component is the fact decubitus lesions are rarely found in the polio patient with severe paralysis, but who retains normal sensory function.(57)

Perhaps the best evidence to support the refutation of Charcot's neurotrophic factor is found in the experiments performed by Kosiak who applied continuous pressure to animals with and without spinal lesion. He found that there was no variation in the amount of pressure, duration of time, or healing rate between the two groups of animals.

There are some factors in paraplegia that are changed with the cord lesion. Some authors believe that the vascular changes are relatively unimportant.(9) Others maintain capillary pressure is supported by active vasomotion which is lost in cord lesions.(43,50) The mechanism of reactive hyperemia however does remain. Also the fact that vascularity is improved with sympathectomy, and pressure ulcers are never seen with sympathectomy, leads one to believe vascular alteration is minimal.(57) The fact that there is diminished muscular activity below the lesion means overall vascular flow is diminished and pressures are reduced.(33)

Loss of the protective functions of muscle tone is involved. The decubitus ulcers are more common in flaccid than spastic paralysis which seems to offer a protective function.(13)

Moistures effect on the skin is detrimental in that it causes maceration and makes it more susceptible to trauma. This becomes extremely important in the paraplegic who is incontinant of both urine and feces--naturally this applies to incontinance in any hospital population. There is some question as to how important this actually is. It is at best of minor importance for it is rare to see ulcerations on scrotum and thigh in the most neglected patients; and if they form, they clear rapidly.(31) The mechanism of reflex sweating in the paraplegic can theoretically produce enough moisture to cause some tissue maceration.(12,50) I do not believe this can be of great importance.

Skin maceration and abrasion due to any cause will aid in the destruction of tissue, permit bacterial invasion and make ulceration more likely. The paraplegic with muscle weakness may rely on nursing care to lift him up, and if he is pulled instead of lifted, abrasion against sheets will aid in ulcer formation. Again this pertains to any hospital population.

The initial phase of paraplegia is accompanied by various metabolic changes; changes which when noted alone have been associated with an increased incidence of pressure sores. Following closely the onset of paraplegia there is a marked loss of appetite

and weight. Malnutrition with tissue wasting and a loss of subcutaneous fat and muscle bulk causes a loss of mechanical padding in a state of negative nitrogen balance. These all lead to an increased incidence of pressure sores.(39) We have one group reported where the average weight loss for a newly admitted paraplegic was 50 pounds until stabilization occurred.(20) In considering the magnitude of cord lesions, in one report there were an estimated 83,000 - 100,000 paraplegics in the United States with an increase of 1,000 per year.(36)

Miscellaneous Factors: These include a group of rather nonspecific elements in the formation of the decubitus ulcer. Age is one, and in general the ease of decubitus formation increases with age. The difference between age 75 and 85 is an incidence of twice as many ulcers.(30) A racial factor has been introduced with one author claiming a decreased susceptibility in the Negro, and others deny any racial variation.(18,32) Hypoproteinemia and hypovitaminosis are more readily associated with delayed wound healing, but an increased incidence of ulcers has been noted by many in these conditions. Fever's contribution to ulcer formation is related to the concomitant increase in metabolism. This of course means a decreased time of ischemia necessary to cause cellular necrosis. Anemia is an oft noted accompaniment of the decubitus sore, and it has been stated they will not develop in the average patient whose hemoglobin is above 10-12 gms.(49) Probably the greatest

factor is neglect in patient care, but this will be discussed more fully under prevention.

Prevention: With a knowledge of the etiology, and hospital population in which pressure sores form, a logical and effective preventive program can be instituted. On the other hand if these factors are forgotten, disaster can result. It may be interesting to consider a case of forgotten basics. I have composed a simple example where nursing care has gone awry. The well meaning (but uniformed) nurse who tucks a patient in, pulls him up in the bed and then cranks the bed up so he can look around, is this case. She has made him warm, which increases the humidity of his clothes; she has abraded his skin by pulling him in bed; she has added a shearing force, and eliminated much of his ability to move and roll about; as with the back of the bed up, he must lay on his back to be comfortable. This simple case demonstrates the fact that it is more than common sense which must be used to prevent these lesions; it is attention to details and compulsive coherence to every preventive measure.

Pressure, which is the primary cause of decubitus ulcers, must be dealt with first. There are three things that can be done with pressure:

1. eliminated by displacement
2. dispersed over a greater area
3. reduced in time

Decreasing the duration of time over which pressure acts is perhaps the easiest to control. As was previously noted, this

time should never exceed 2 hours. Therefore, simple direction of turning the patient from side to side and back to front can almost completely prevent the formation of pressure sores; if adhered to compulsively. This can be accomplished with a Stryker or Foster frame in paraplegics or the severely debilitated. Another method, the air mattress, developed by Gardner, has proved to be extremely beneficial in preventive programs.(5,33,34) This consists of an inflatable air mattress with alternating compartments 3 cm. in width. These are alternately filled with air every 5 minutes, thereby successfully accomplishing a reduction in the time over which pressure acts. The original mattresses had horizontal compartments which caused some difficulty with stasis of blood; however, the newer models have vertical compartments and eliminate this problem.(57) These have greatest value in patients forced to remain in fixed posturing due to orthopedic tractions arrangements, or very old patients who suffer respiratory embarrassment when placed in lateral or prone positions. These, however, are not their only use.

In the use of the air mattress or any such automatic device; it must be remembered that these do not obviate other basic needs in a preventive program. As the heel fits exactly between these compartments and may be subjected to constant pressure, thinking the mattress takes care of everything may be disasterous.

The paraplegic with an alert mind and useable upper extremities must aid in control of time over which pressure acts if he is ever

to function and avoid pressure sores. He must be educated also, in every facet of prevention, for the pressure sore is a constant threat. In the wheelchair, he is taught to support himself strongly on his arms at constant intervals and decompress his ischial region. As he no longer has the warning of pain and discomfort to signal him, he must substitute conscious awareness to assure interval decompression and weight shifting. If he fails, a decubitus ulcer and many months delay in rehabilitation will be a lesson to reinforce his conscious awareness.

The total elimination of pressure by displacement may be necessary to avoid even minimal pressure over rehealed lesions which are fibrous and lack vascularity making an area of increased susceptibility. This can be accomplished by cut-out pillows and seat boards for wheelchairs known to all. This keeps pressure from ulcer-prone ischial areas. The common use of air rings and doughnuts are other methods of eliminating pressure by displacement. An important consideration with this method is the realization that pressure has been displaced, not eliminated, and may create problems elsewhere. This does remain a useful technique in wheelchair patients. The doughnuts which must be used with extreme caution, for they may produce larger rings of ischemia, are useful in relatively active, elderly individuals who, if in bed for short term confinement, develop malleolar lesions. These can protect this form of lesion and allow it to go on and heal without causing a great deal of incapacity.

The dispersal of pressure is the most general and useful means of pressure prevention and a factor which applies to the entire hospital population. This principle allows for conformity of the surface to the contours of the skeleton and allowing weight to be distributed. The foam rubber mattress accomplishes this; and foam rubber padding, where applicable. In the hospital population where pressure sores are likely to occur, the head of the bed should never be raised, as this produces a shearing force. This has been discussed in the etiology. A second factor in keeping a patient at an angle is the constant tendency to slide down in bed which is often countered by a hospital aid pulling the patient up and abrading the patient's skin against the sheets.

Trauma to skin is an extremely important predisposing factor. The number one cause is in the thoughtless moving of the patient. When a patient is to be moved, he should be rolled or lifted and never pulled. By this simple method, unnecessary skin abrasion is avoided. When skin is soiled or moistened, it is much more prone to trauma as the effects of maceration due to moisture make it much less resistant. This means a patient must be constantly attended to if there is a question of incontinence of bowel or bladder. He must never be allowed to remain in a soiled bed. Also, the humidity of bed clothes should be reduced by avoiding excessive coverings and temperature control of rooms. Some have recommended talcs and powders to prevent moisture of sweating, but too often these act also as abrasives.

The use of anabolic testosterone derivitives have been advocated for periods of negative nitrogen balance during hospitalization, and there is general agreement that the measureable effects of negative nitrogen balance are eliminated by these agents.(39,59) There are, however, few controlled studies to indicate the real usefulness of these agents. In one extensive and controlled study of 400 patients, specifically for the prevention of decubitus ulcers, there was no variation between control and experimental groups. The study did demonstrate occurrence of incontinence in 69% of those getting ulcers from both groups.(28)

Massage in the prevention of pressure sores is based on the circulatory stimulation of manipulation, but this effect is relatively transient and if done with vigor and use of a lotion may be harmful. Therefore, if massage is used it must be done lightly and without lotions or oils.

Exposure to ultraviolet light is another frequently mentioned method of prevention, which is also based on circulatory stimulation, which it certainly does. It also accomplishes maintenance of skin bacteria at lower levels. The pigmentation which ultraviolet light produces, is believed to toughen the skin.(39)

The best prevention is constant observation. The paraplegic, if able, can perform self-examination with a long handled mirror, looking for the ominous erythema that heralds excess pressure and periods of ischemia. In the old and debilitated this is attended to by the hospital staff, who examines and tests erythema by the blanching method as previously noted.

The general aspects of patient care such as diet, secondary infection, treating of anemia, are all important and will be discussed more completely in the section on therapy where they are so intimately related with wound healing. But in prevention, it should not be forgotten we are dealing with a total patient. One aspect of general care which should be mentioned, is the immediate attention which should be rendered to incipient congestive heart failure for if this is left for several days the effects of sacral edema will aid in ulcer formation.

No discussion of prevention with regard to decubitus ulcers would be complete without the inclusion of the sheep-skin; one of the oldest and truest means. In one report they have been used for over 35 years.(11) The patient in whom the development of a pressure sore is likely, is placed nude upon the sheepskin. The skin acts by dispersing weight--the hairs acting as numerous coils, and eliminating moisture--they can absorb up to 33% of their dry weight without feeling wet. The thick skin also prevents wrinkling on itself. Therefore, pressure, friction, and moisture are reduced with little effort by the attending staff. These are somewhat difficult to use in the incontinent, but can be washed frequently. In laundering the skins, a bacteriocide must be used to prevent the harboring of bacteria.(11,29) This represents one of the least expensive and most efficacious methods of prevention in hospitals with limited personnel.

Therapy: When we speak of therapy for a decubitus ulcer, we are speaking simply of aiding the healing of a wound by secondary intention through the series of granulation tissue formation, migration of epidermis, collagen formation, fibroblast proliferation and wound contraction. In truth, other than conforming to the basic rules of wound treatment, there is little we can do to aid and hurry healing however, there is much that we can do or leave undone to delay wound healing. Therefore, before I discuss therapy, there are some essentials of wound healing and delayed wound healing which must be used for a basis. Most of the following information has been extracted from more recent literature and comments from older literature omitted as it would not be contributory. The discussion of wound healing was extracted from Edwards & Dunphy's articles and Williamson's symposium.(26,27,58)

Healing depends in a broad sense upon the two basic biologic mechanisms of tissue migration--contraction and epitheliazation, and regeneration--including repair which is replacement with a dissimilar cell, as scar tissue. These mechanisms depend on a host of interdependent biochemical mechanisms.

Epitheliazation occurs by an active mitotic process from the edge of the wound. This mitotic activity is limited to within 1mm from the wound edge and later over the newly formed epithelium. This is of importance for in debridement by surgical means, care should be taken to avoid this important zone and only necrotic tissue

must be taken. As this advances, it undermines the fibrin crust which has formed and a proteolytic enzyme, a fibrinolysin, is assumed to be produced by this epidermis. In large wounds and tissue not fixed by underlying structures, contraction is the major mechanism by which closure takes place. There is also the deposition of collagen which provide tensile strength, and all of these are laid over the substrate of granulation tissue which is formed early after tissue disruption. An important fact in the healing of cutaneous wounds is the fact that in contrast to muscle and visceral wounds, skin is reunited with scar tissue which is weaker and more poorly vascilarized than the original tissue. This must be remembered in dealing with healed decubitus ulcers, for just because the wound has been epitheliazed, does not mean it can receive the same daily use other tissue can. Also, this weaker skin is in an area of subjection to more stress by the nature of its origin.

The metabolic factors in wound healing are the most complex and difficult to evaluate, and are in the greatest state of flux at this time. However, there are some important aspects of this problem that are well documented. The negative nitrogen balance of malnutrition and specific amino acid defects can be corrected nutritionally; however, the catabolic response of severe trauma--as may be seen in the paraplegic--cannot be so corrected. Despite this fact, healing does begin in negative nitrogen balance demonstrating that anabolic processes are at least locally operative.

The expected protein sparing effect of a high carbohydrate diet is not observed and will not decrease nitrogen excretion or change the rate of healing of wounds; however, the feeding of a high protein diet increased the rate of healing over controlled low protein diets and resulted in a closer approach to nitrogen balance. Wound healing does occur in fasting animals and in order to arrest wound healing, long periods of starvation are necessary with a loss of a large percentage of body weight. Although anabolic hormones do decrease negative nitrogen balance, there is no evidence to indicate they aid or increase wound healing. There does remain good evidence that the material for wound healing comes primarily if not exclusively from tissue protein. Therefore, the frequently used rationalization for cessation of healing and spontaneous ulcer formation ducto nutrition and nitrogen balance has little basis in fact. As an aside, the single amino acid considered to be deficient when true wound healing takes place is cystine.

The problem of scorbutic healing is frequently mentioned, and is intimately related to the problem of wound healing. In scorbutic healing the basic defect is related to collagen synthesis with the result that contraction and scarring fails and tensile strength is greatly decreased. As vitamin C is the one vitamin which cannot be stored in the body the body economy requires constant replenishment. However, there is no evidence to indicate that excess vitamin C intake is in any way beneficial to wound healing.

It is interesting to note that cortisone, which in general is related to delayed wound healing, relieves some of the effect of scorbutic wound healing. As the intimate relation of both to the collagen diseases exist, this may present in the future some interesting developments.

The problem of steroids and wound healing is extremely complex, for although they are generally considered to retard wound healing, they do aid scorbutic healing, and some authors feel steroids have no effect on granulation tissue and contraction. This refers to the antiphlogistic steroids. There is the same effect reported for topically applied as well as systemically used steroids.

Other factors involve patients with poor cardiac output, or local circulatory problems as diabetics and patients with venous insufficiency, all of which retard wound healing by depriving of oxygen and nutrients.

The search for methods to speed the rate of healing is as old as medicine, and the agents alleged to accomplish this are too numerous to even list--they range from radiation to urine. However, it remains generally accepted that there is no practical method to obtain accelerated wound healing. It is wise to remember a wound or lesion has high priority in the body economy: its granulation tissue protects it from bacteria--the number one cause of delayed healing; and if properly cleansed and left to its own resources, it will eventually heal. Most of the agents proported in the past and

present to increase wound healing, did so only because the wound was given attention and kept clean--perhaps the agent slowed the actual healing process. Many agents did not accelerate the rate of healing but did restore it to a normal rate which is effective. In considering therapy, this paper will be limited to the conservative management of pressure sores, and the surgical development which had its origin in 1945 will be omitted. It is well to note conservative therapy in the development of a good base of granulation tissue is the first step in a surgical approach. However, the technical procedures involved in such procedures as free and flap grafts and ischiectomies and trochanterectomies are beyond the scope of this paper.

The first thing which must be attended to is the general health of the patient. The diet should be high protein (the protein sources should be varied to assure the presence of all essential amino acids at one time) (49) with an adequate vitamin intake. Any secondary infection must be vigorously treated. Anemia should be corrected with transfusions if necessary to maintain hemoglobin at over 14 grams per cent.(18,20,23,49,59) Hypoproteinemia can be corrected early by plasma(18) or concentrated human albumin.(49) The patient's fluid intake must be closely supervised and supplemented with intravenous fluids if oral intake lags. A program of general support should always be maintained.

In treatment of a formal decubitus ulcer, the ulcer must be absolutely removed from pressure, as it will not be able to tolerate

pressure and heal. This means that air mattresses and sheepskins, etc. have no place in treatment, but remain important in prevention of other sores forming. It is important to remember not to forget prevention while treating an ulcer. Patients can be kept off of ulcerated areas with air rings and Foster or Stryker frames.

An essential beginning in treatment is thorough and continuing debridement of necrotic tissue, for it is in this tissue contaminating bacteria live and propogate. By thorough debridement the chain of bacterial proliferation is halted. The older method of Munro of leaving the slough in place is extremely dangerous.(37,48) There are three methods of debridement available: hydrodebridement, surgical debridement, and topical debridement with enzymes. In the initial stage of formation surgical debridement is probably the most efficacious method, but after this with a granulation bed and a narrow, vital strip of mitotic epidermis subject to excessive bleeding or healing arrest, this loses its efficiency. The bleeding tendency as was mentioned previously has been considered by one author to be a contraindication to surgical debridement. Hydrodebridement is the least traumatic and most cleansing method available, accomplished in a Hubbard or other tank with or without the brine solutions recommended by some(49), it can be frequently repeated and involves nothing which would delay healing. There is one problem associated with this form of therapy, and that is in the patient requiring a catheter who would be subjected to urinary infection.

Still this can be performed on days when the catheter is changed.

The agents to produce enzymatic debridement are legion and there is really little to recommend them. It is certainly true that a forcep and scissors, or one episode in a Hubbard tank, can accomplish more in a few minutes than all these enzymes can accomplish in any amount of time. A simple wet dressing can produce sufficient maceration of necrotic tissue to allow irrigation to rid the lesion of more tissue than the enzymes will digest and prepare for removal. The enzymes fall into three catagories:

I. Bacterial Enzymes

Streptokinase: This functions by converting a plasma factor, plasmenogen, to plasmin (fibrinolysin). This works best at a pH of 6 and puss is more alkaline than this. Also in areas of vascular insufficiency the plasma element is insufficient.

Streptodornase: This acts by depolymerizing DNA which is the viscus product of puss cell breakdown. This also works best at a pH lower than puss. Together with fibrinolysin, which breaks up fibrin this aids in removal of coagulum and purulent exudate but does not cause the dissolution of necrotic tissue.

Collagenase: As A and B are derived from Streptococcus, this enzyme is derived from Clostridium histolytica. This is a powerful digestant of necrotic tissue, but it also may be toxic when absorbed from large areas.

II. Animal Enzymes:

Fibrinolysin--Deoxyribonuclease: These two enzymes are from bovine pancrease and act similar to Streptokinase--Streptodornase mixtures but do not require plasma precursor. They will not affect large amounts of necrotic tissue.

Trypsin: This is a proteolytic enzyme from animal pancreas. Its action is very short and it is readily destroyed by bacteria and for this reason it is employed with a topically antibiotic and must be changed frequently to be useful.

Pepsin: This is another derivative of animal pancreas. It requires an extremely acid pH which is seldom found in a wound.

III. Vegetable Enzymes

Papain: This is a proteolytic enzyme.

Exposure of the lesion to luminous heat is a frequent recommendation.(45,49,55) This includes ultraviolet and simple luminous heat which stimulates circulation through an increase in capillary pressure. This also acts to decrease the contamination which is

inevitable. There are also a few reports in the literature where ultraviolet has been used with an increase in wound healing.(47,51) Ultrasound would seem to produce this effect by heat production which increases circulation. However, one report used a 10cm head and water and mineral oil coupling at 1.5 watts per cm^2 for 2-4 minutes and stated the heat would be negligible at this power range.(57) They do not propose a reason for its effect.

There are so many topical solutions used for therapy of pressure sores it is difficult to know where to begin. However, so few of them are of apparent use, I will omit many. It is well to remember the basic fact that "wound healing is a function of living tissues and is not hastened or stimulated but often retarded by any chemical agent applied to the surface." (58)

The antibacterial solution may produce sensitization and no chemical will eliminate bacteria from a chronically open wound but it may cause the rapid replacement of a resistance for a vulnerable organism. They should never be used in a normally healing wound. If contamination becomes a concern, proper debridement of tissue on which bacteria live and proliferate is the avenue with which this problem should be approached. A time honored method is the packing of granulated sugar into these wounds.(49,53,56) These seem to work by osmotically destroying bacteria. Zinc peroxide ointment has been recommended for it produces O_2 which is detrimental to growth of potentially dangerous microaerophilic streptococcus.(1,18,48,56) Most antiseptic solutions are too strong and irritate and retard healing or are too dilute to be of use.

The introduction of dried blood plasma to be placed in the decubitus ulcer, perhaps represents the only true physiologic millieu which has been developed.(16) Also the loss of large amounts of protein from wounds can be stopped by this method as the osmotic gradient is reversed. Some consider a protease to exist in this blend and a slow debridement to take place also, but the true benefit is the providing of a physiologic millieu and the reversal of the osmotic gradient to prevent protein loss.

I do not believe such things as gentian violet, urea (a protein solvent), chlorophyll, balsam of peru, merthiolate and the host of other topical agents really have any use in the treatment of decubitus ulcers.

The proper dressing to cover the lesion is another component which remains in dispute--for there are the wet schools, the dry schools, and the leave-it-open schools. It would seem as we are dealing with a granulating subsurface wound which will not be exposed to pressure or trauma of any form, a moist saline or similar dressing should be used. This should be covered with numerous soft packs and held in place with stockinet cut on a bias to avoid tapes which abrade skin. If moist dressings are used silicone sprays around a wound may aid in avoiding the maceration of peripheral tissue. Howevever, if all pressure is kept off this area this maceration will be of little importance.

In summary, therapy should consist of attention to the general needs of the patient; including nutritional support, absolute removal of pressure from wound area, thorough debridement--at the most, once with surgery and then exclusively with hydrodebridement, daily exposure to some form of luminous heat (diathermy should never be used), moist dressing with dried blood plasma and avoidance of any other topical agents, frequent change of dressing, and psychological support for the patient.

Conclusion: The decubitus ulcer or pressure sore has presented a trying problem to both patient care and skill in therapy. In the past, due to Charcot's theory, they were looked on as inevitable and almost incurable. Medicines' search for a method of dealing with them and to stimulate their healing led it astray from basic principles of wound care which it had considered sacred for many years, and as is evidenced in some relatively recent literature, in many camps, return to these principles has not yet taken place. The sina qua non of decubiti is pressure--without it they do not occur; with it they do not heal. The most sure method, therefore, to prevent this lesion is constant attention to the patient with intermittant decompression of body pressure by the most expedient means available. No lesion should be looked upon as incurable--for we have in the literature one case of a decubitus ulcer of 31 years duration which was cured with intensive care.(51) The treatment depends on sane principles of basic wound care as was previously outlined. There is no place in therapy for toxic topical agents, suspending patients from pins (15), sawdust beds, and mysticism. With attention to details of patient care, sound principles of wound healing and general medicine the decutitus ulcer can successfully be prevented and treated if it becomes necessary.

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