

A decade of pressure sore research: 1977-1987

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INTRODUCTION

This paper reviews the literature from 1977-1987 on the etiology and pathophysiology of pressure sores, factors contributing to its formation, diagnostic and pressure-measuring devices, and cushions and devices designed to prevent sores. The authors hope this review will show that many current assumptions may be invalid and should be re-examined.

A pressure sore is an ulceration of the skin and/or deeper tissues due to unrelieved pressure, shear force(s), and/or frictional force(s). Shear forces are produced when contiguous parts slide relative to each other in a direction parallel to their plane of contact. Frictional forces are due to the resistance of one body sliding, rolling, or flowing over another. Either of these forces can be produced by a person sliding on bed sheets when being placed

into a recumbent position, or by a person making frequent lateral or oblique shifts while sitting on a wheelchair. In addition to the term pressure sore, other terms used to describe the resulting ulcerations are decubitus ulcers and bed sores. However, these other terms imply that the sores or ulcers are due to lying down or occur only in beds, which is untrue.

Much has been assumed with regard to the etiology of pressure sores and used to make conclusions for the determination of risk factors, the design of devices to measure pressure, and for the design of cushions and mattresses. For example, an assumption still made in the present literature is that pressure sores occur due to pressure at the surface exceeding mean capillary pressure (32 mmHg) resulting in tissue ischemia and then necrosis.

ETIOLOGY

Recent studies have gone beyond assuming that tissue necrosis due to external pressure is a result of ischemia and have probed into how this might occur. Histologic studies by Witkowski and Parish have revealed that early signs of damage occur in the upper dermis (53). Apparently, capillaries and venules become dilated and their endothelial cells swollen and separated. Evidence of edema and perivascular round cell infiltrates then appear, followed by platelet aggregates, red blood cell engorge-

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ment, and perivascular hemorrhage (50,53). Subcutaneous fat shows signs of necrosis along with early vascular changes (53). The interesting point put forth by Witkowski and Parish is that the epidermis does not show any signs of necrosis until late in the course. They point out that this is consistent with the finding that epidermal cells are able to withstand prolonged absence of oxygen both *in vivo* and *in vitro*.

Among the theories proposed regarding tissue necrosis, Krouskop presents an excellent one in which he discusses the role of collagen synthesis, lymphatic drainage, and interstitial fluid (23). According to his hypothesis, hypoxia can result in the production of a water soluble form of collagen. When the collagen is washed out, mechanical stress is redistributed to other elements in the area causing an increased stress to these elements and a decreased transport of nutrients to this area. In addition, interstitial fluid diminishes resulting in cell-to-cell contact and possible rupture of the cells. Rupture would cause dumping of cell contents into the intercellular space. If external pressure is removed, a sufficiently low interstitial fluid pressure could be created so that capillaries would rupture and dump small proteins and their contents into surrounding tissues. If lymphatic flow is impaired by hypoxia or some chemical like heparin or glucocorticoids, the products dumped by cells and capillaries cannot be cleared, and tissue poisoning would occur (23). Krouskop discusses how glucocorticoids, released during stress, can cause the production of water soluble collagen in addition to inhibiting smooth muscle functioning in the lymphatic system. Age is also a factor because a decrease in elastic fiber production and a stiffening of the tissue occurs with

aging, which makes pressure damage more likely. Although this seems like a long chain of if's and maybe's, many of the factors described here are in agreement with other studies (24,31,42).

Other research has focused on fibrinolytic activity to explain epidermal and dermal tissue necrosis (2,50). According to Seiler and Stahelin, traumatized tissue can undergo decreased fibrinolytic activity that would result in the presence of fibrin deposits in the intercellular space and capillaries, sometimes with intra-arteriolar fibrin thrombi (50). This, in turn, could cause vessel occlusion and result in tissue necrosis. In their study, Seiler and Stahelin did find significantly decreased fibrinolytic activity within 0 to 1.5 mm of the ulcer border when compared with a zone 9 mm from the border. Even samples taken 12 mm from the border revealed significantly less activity than normal skin. Bader, *et al.* agree that decreased fibrinolytic activity is a main factor leading to tissue necrosis and specify that shear stress is the cause of this loss of activity (2). This group found that applying tangential force to the forearm of normal individuals not only caused vessel occlusion, but also collapse.

Although the above theories focus on different components of the tissue, they all agree that ischemia and hypoxia, which lead to a decreased or absent supply of oxygen to the tissue, are main factors in necrosis. Thus, other studies have been carried out on the oxygen retaining properties of tissues (35,49). Newson *et al.* compared the effect of an externally applied load on the partial pressure of oxygen in "hard" tissues (those overlying bony prominences) with that in "soft" tissues (lateral aspect of thigh) (35). The authors concluded that

both soft and hard sites could tolerate similar pressures, and that hard tissue undergoes necrosis more easily than soft tissue due to the higher pressure experienced over bony prominences. In addition, a trend was described in all tissues in which the partial pressure of oxygen decreased slowly at first, and then at an accelerating rate once a cutoff load was reached. Seiler and Stahelin disagree with these findings and describe a distinct difference between soft and hard sites (49). Hard sites showed a rapid fall in oxygen tension under increasing pressure, while soft tissue revealed a small decrease. The authors claim that this difference is the reason hard sites are preferential areas for pressure sore formation.

MICROBIOLOGY OF PRESSURE SORES

The final etiological factor presented here involves the microbiology of pressure sores. Although it is generally agreed that bacteria are not a cause of pressure sores, it is believed that they contribute to tissue breakdown and delay healing (47,50). As discussed earlier, hypoxia or anoxia results in the stoppage of lymphatic flow due to its effect on the smooth muscle of the lymph vessels. Thus, aerobic and/or anaerobic microorganisms and their waste products can accumulate in this area and cause infection and bacteremia leading to increased tissue breakdown (50). Once bacteria colonize a sore, they will undoubtedly cause a delay in healing by infecting the wound. It has been discovered that the type of bacteria found on the wound can give information on how well the wound is healing. Sapico *et al.* showed that healing wounds had

absolutely no anaerobic bacteria and that few colonies and types of aerobic bacteria were present (47). On the other hand, nonhealing wounds had the highest count of both aerobic and anaerobic bacteria. Similar results were obtained by Seiler and Stahelin (50).

FACTORS PREDISPOSING PATIENTS TO PRESSURE SORE FORMATION

Previous studies have attempted to set thresholds (with pressure and oxygen tension measurements) beyond which pressure sores will form (9,49). However, clinical studies have shown repeatedly that these thresholds are surpassed by patients without apparent damage to tissue (16,30,40). Thus, it becomes important to consider what other factors contribute to tissue resistance or to susceptibility to the formation of pressure sores.

Many of the studies in this area are correlational. Such factors as anticoagulation medication, low serum albumin level, low total lymphocyte count, fecal incontinence, fractures, increased age, increased length of paralysis, social state of the patient, and smoking have all been correlated with either the presence or worsening of pressure sores (1,17,25,41,52). One statistical study even showed an association between delayed wound healing and paraplegia and quadriplegia when compared to a control group (4). However, these studies indicate that the factors studied may be important, but do not reveal how or why.

Fortunately, some researchers have studied how factors such as age might predispose a person to pressure sores (13,23). Ek, *et al.* showed that

subjects over the age of 60 had less ability to increase skin blood flow in response to thermal stimulus when compared to subjects under the age of 60 (13). A significant difference in this ability was also found between subjects over 60 in hospitals and those not in hospitals, with the former showing less of a response. This physiological phenomenon may be quite important because temperature usually rises in the area of the body in contact with seat cushions, and a decreased response to this rise could make the tissue more susceptible to ischemia.

Another study addressed the question of how paralysis could be a major factor in making an individual more susceptible to pressure sores (9). In experiments with pigs, Daniels *et al.* showed that animals with transected spinal cords had a great reduction in thickness of soft tissue below the level of paralysis after 6 weeks had passed. This is significant because the thicker the tissue layer over which pressure is exerting its force, the more area over which the pressure can spread, thereby reducing stress on the tissue. The authors also pointed out that paralysis results in other factors which contribute to the formation of decubitus ulcers. Among these factors are an impairment of mobility resulting in unrelieved tissue ischemia, loss of sensation, which removes the "alarm system" that indicates when tissue has been loaded for too long, and incontinence, which macerates the skin and facilitates skin breakdown.

In a study by Narsete *et al.*, it was cited that normally innervated skin can withstand ischemia for approximately 3 hours longer than skin of a spinal cord injured patient without showing signs of breakdown leading to necrosis (33). Other factors such as dietary insufficiency, anemia, and hypo-

proteinemia were also discussed in regard to reducing the ability of a wound to heal.

DISTRIBUTION OF PRESSURE WITHIN TISSUE

Even though pressure is consistently discussed as the main cause of pressure sores, few papers have addressed how compressive pressure is distributed over the layers of tissue. However, study of the literature on this topic has revealed new information which is important to all those interested in formation of pressure sores. Two animal studies by Daniel *et al.* (9) and Nola and Vistnes (37) have shown that muscle (deep tissue) is more sensitive to pressure than skin (superficial tissue). This has led these researchers to believe that deeper tissues, such as muscle, will undergo necrosis under less severe conditions than the superficial tissues. Muscle might be more sensitive due to its increased metabolic activity and design (37). According to Vistnes, this should be interpreted with caution as muscle is not overlying bony prominences when the patient is in certain positions such as sitting. However, the tissues nearest to the bony prominence are thought to experience the most pressure, which then dissipates and spreads as the superficial layers are approached (Vistnes). Pressure is believed to be distributed in a triangular fashion with the point of the triangle located at the site nearest to the bony prominence. Thus, although the superficial layers experience the least amount of pressure, they are the layers with the largest area affected. Reuler *et al.* agree that pressure is concentrated at the tissue closest to the bony prominence, but believe that this

layer also has the largest area affected by pressure (upside down triangle) (45).

DEVICES USED TO MEASURE PRESSURE

Most pressure-sensing devices are designed to measure pressure at the cushion/mattress-skin interface while the subject is sitting or lying down. Among the earliest developed and most commonly used are air cell transducers. Recently, the design of these devices has been improved and electrical transducers whose measuring properties are based on differences in resistance have been developed. The main criticisms regarding such transducers are that they monitor only a small area of the skin surface which is in contact with the seat cushion or mattress, and that they interrupt the contact between the skin and support surface. In addition, researchers do not know the relationship between the interface pressure and the actual pressure in deeper tissues or if the absolute values recorded are of any value in determining a threshold (38,44). One study by Palmieri *et al.* addressed some of those issues and compared sitting pressures on 21 wheelchair cushions as measured by air cell and miniature electronic transducers (38). The researchers found that both transducers gave statistically consistent results for all 21 cushions. However, they did warn that the measurements obtained from these devices are only good for comparative purposes.

Another study by Reddy *et al.* evaluated several electrical transducers, a single air cell transducer, and a pressure evaluator pad with respect to the measurement of pressure at the buttock-cushion interface (44). Compared to calculated pressure

values, it was found that the transducers consistently overestimated the pressure at the interface. In addition, the values obtained depended on "contact geometry" and on the material from which the cushion was constructed. Thus, if measurements are needed for more than comparative purposes, the authors recommend that a correction factor be researched first. According to this research, an ideal transducer should carry all the load (requiring it to equal the area of interfacing surfaces) or a very small part of it (having a small area and air cavity). In the same study, a comparison was made between the skin-foam interface pressure measured by a Scimedics Pressure Evaluator Pad and the subcutaneous tissue pressure as measured by a wick catheter. The authors found that these measurements correlated well, showing that the interface pressure does give some valuable information on tissue load. Two other research groups have developed pads to measure the interface pressure (3,18). These pads are sometimes preferred over single transducers because they can monitor pressure in more than one area at any one time.

A unique approach to gathering this type of information has been introduced by Kadada *et al.* It uses an ultrasonic dimension gauging technique (22). With this device, the authors have been able to study the buttock-cushion interface without interfering with the skin-cushion contact. Although only two-dimensional contour data can be obtained through this machine, it has opened up a whole new area of research for measuring interface pressures.

In choosing to focus on how pressure is distributed over the buttock of a patient, some researchers have designed devices to measure many pressure points simultaneously. Drummond *et al.*

developed the Pressure Scanner, which has proven to be a useful and valuable tool (11,12). This device collects information via 64 transducers wired to a minicomputer that yields contour and three-dimensional plots. From these data, one can determine how the pressure is distributed and where the center of each pressure point is located. Another effective tool devised for this purpose is the Ischiobarograph, designed by Minns *et al.* (32). This device yields pressure patterns with different colors and provides information on the distribution and center of pressure.

Recently, a pressure-sensing device has been developed which is invasive, but which reveals previously unforeseen results regarding pressure gradients (27). Among other things, it consists of monolithic silicon pressure sensors and a sensing needle that is inserted vertically into the subject. This study was done on pigs and demonstrated that pressure increased both laterally and in depth as proximity to a bony prominence increased. Thus, deeper tissues do experience larger amounts of pressure as previously discussed (37,45). One implication of this is that, while skin surface pressures might be maintained at a low level (below a supposed threshold), deeper tissues could be experiencing a large amount of pressure leading to tissue damage and the formation of a cavity below the dermis (27). These findings disagree with Reddy *et al.*, who found similarities between interface and subcutaneous pressures (44). Although this technique has revealed much important information and will continue to do so, its use is limited because accuracy of measurements depends on tissue structure. As noted by Reddy *et al.*, if the tissue is abnormal or damaged in any way and does not

collapse around the needle as assumed, inconsistent and/or abnormal readings will be obtained.

MONITORING PATIENTS: BEHAVIOR LEADING TO PRESSURE SORE FORMATION

A different approach used to determine the factors leading to pressure sore formation is seen in the devices designed to monitor patients. One of the basic goals of these systems is to determine what type of patient behavior leads to tissue breakdown, and eventually ulcers, rather than at what pressures this might occur. One such system is the Time-Logger-Communicator (TLC), which contains a portable computer for collection of data (20,30). This system records information on how long a subject has been sitting on his/her wheelchair, how frequently pressure-relieving liftoffs have been performed, and at what times during the day liftoffs were made. In addition, the computer provides a plot showing the distribution of interlift intervals for the day and a list of the longest continuous downtimes of the day. A goal of this system is to prevent the behavior leading to tissue breakdown. As a result, the system is equipped with a device that signals the patient as to when to perform a pressure-relieving liftoff. This signal can be programmed to warn the patient after a prescribed amount of time or after surpassing a set pressure level. The TLC is very practical because it is clamped to the wheelchair frame without interfering with normal chair usage and folding.

Another monitoring system is the Wheelchair Patient Monitor (WPM), which detects patient weight shifts by monitoring seat sensors (8). The

information is gathered by a computer that can later give such data as time between moves, the duration of the move, and averages for each parameter. Graphs and histograms can be constructed showing the types of behavior that were most common. The WPM also contains a time-based alarm which is set off when weight shift behavior has not been performed for a set period of time. Practicality has been incorporated into this system as well because it can be attached to a conventional wheelchair.

DIAGNOSIS

No equipment has been specifically designed or frequently used for the diagnosis of pressure sores because most physicians rely on physical examination. However, recent findings have suggested that there is a need to look at the deeper tissues first for signs of ulcer formation (10,37,45). Radiographic studies on pressure sores have shown that such devices as radiography, computed tomography, and sinography can give information about deeper tissues and provide images showing any complications (15,21). One study using all three devices showed that such information as depth of the ulceration, presence and extent of bone erosion, presence and extent of fibrosis, and the presence or absence of an air cavity in the ulcer can be obtained from the images produced (21). The discovery of an air cavity in some patients would lend support to the theory proposed by Vistnes (27) and Reuler (45). In addition, complications such as ectopic bone located near the ulcer can be detected. This would be quite significant since such a piece of bone can act as another bony prominence near the surface leading to

increased pressure and tissue breakdown (21). Computed tomography has also been found to be useful in detecting such complications as osteomyelitis and abscesses (15).

DETECTION OF PATIENTS AT RISK FOR PRESSURE SORES

This area of research is still full of many unanswered questions and needs more direct study if prevention is ever to be achieved. A few of the devices previously discussed, however, provide some valuable information that can be used to detect patients at risk. For example, clinical studies on patients have been and should continue to be performed using the Wheelchair Patient Monitor and Timer-Logger-Communicator. The studies performed focused on behavior leading to pressure sore formation, rather than absolute pressure or time thresholds. With this type of information and patient surveys, physicians and nurses can determine which patients have been performing sitting behaviors that predispose them to pressure sore formation.

One device strictly for the purpose of detecting patients at risk is the Pressure Scanner, developed by Drummond *et al.* (11). Studying the pressure distribution of patients, the researchers have presented thresholds, expressed as percent of body weight, beyond which the respective buttock area would be at risk for developing a pressure sore. The validity of these thresholds awaits clinical trials, however, because the initial experiments were performed on only 16 patients. The Ischiobarograph could be used in a similar manner (32).

Some researchers have studied tissue behavior in normal individuals in an attempt to establish a range of values beyond which tissue could be classified as functioning abnormally and be at risk for breakdown. One such study examined the hyperemic reaction of tissue to a pressure load applied for a period of time (28). A highly diminished or lack of increase in skin temperature (due to hyperemia), when compared to normal individuals for that category, could reveal a patient whose tissue is damaged or diseased and susceptible to breakdown. As the authors noted, much more research is needed before meaningful ranges can be established. Another attempt was made using thermography that yielded only a 35 percent predictability rate but shows enough promise to warrant future research (34).

SEAT PADS AND DEVICES USED TO PREVENT SORES

Many seat cushions that distribute pressure evenly in the sitting area of persons who use wheelchairs have been developed commercially. Generally, these cushions fall into four categories: foam, viscoelastic foam, gel, and fluid flotation (7,51). Two studies performed on the four types of cushions showed that each has advantages, and that patient needs should be determined before prescribing a cushion (7,51). Foams were found to have low humidities due to their porous properties, but they also have low heat flux resulting in high skin temperatures. Gels, on the other hand, had a high heat flux, which resulted in low skin temperatures, but they developed high humidities. Water-filled

cushions tended to have the same properties as gels, except for a slightly higher heat flux and lower relative humidity (51). Cochran and Palmieri designed a device for evaluating seat cushions on such parameters as load-indentation, creep/recovery, pressure distribution, and rapid loading (7). This extensive study included clinical trials, all of which confirmed the findings discussed above, plus data on the previously mentioned parameters. The researchers even presented the design of an ideal cushion, which consisted of layers of foam, gel, and viscoelastic foam in an attempt to take advantage of the benefits of each material (7).

Experiments in constructing an ideal cushion were also undertaken by Bowker and Davidson (6). These researchers used a thixotropic substance, which is a gel in the stressed state and a solid in the unstressed state. Ideally, this material would envelope the buttock when the person first sits down, and would solidify when distribution around the buttock was complete. This type of cushion would offer the advantages of flexibility and support at the same time. However, enough pressure must be applied in order for the substance in the solid state to be converted to the gel state. This cushion includes a polythene bag full of thixotropic substance encased in foam.

An active approach to the design of a device for pressure sore prevention has been investigated by Rosenberg and Lach (46). In this device, eight basic modules make up a receptor chamber located under the person's buttock (an insensitive area in paraplegics and quadriplegics). The modules are connected to a small effector balloon which, when inflated, compresses an area of healthy skin with sensation. When areas beneath the buttock have been under

pressure for an excessive period of time, the device parallels this situation to sensitive and healthy skin, which produces an uncomfortable and eventually painful feeling. This will stimulate the person to relieve the pressure.

COMMENTS: WHERE FUTURE RESEARCH SHOULD GO

The basic goal of physicians and scientists should be to close the gap between laboratory and clinical findings. Presently, what has been discovered in the lab has shown little consistency with clinical observations and not much applicability. Among the areas that should be pursued more aggressively is histology. Results from this type of study can reveal what changes the tissue structure is undergoing during breakdown and ulcer formation. Such results would also provide more information on the crucial issues of which layer of tissue shows signs of necrosis first and what area of this layer has been affected. Histology also provides data which is consistent and less dependent on extraneous factors. Once researchers believe they understand the pathophysiology of ulcer formations, models that single out the crucial factors involved should be pursued, such as those attempted by McHanty and Roemer (29).

Recent findings have shown that pressure sore formation is a complex process and cannot be attributed to any one factor. Although it has been universally accepted that pressure begins the process, it should be noted that all the components of the tissue undergoing pressure are affected and not just the vessels. In addition, it should be emphasized

that this is not a surface phenomenon. Deeper tissues are not only affected but may be the first to undergo necrosis. One area of ulcer formation that has been given little attention is frictional and/or shear forces. It has been pointed out that paraplegics can create these forces by actively moving within and in and out of wheelchairs (16). This is significant since shear forces are believed to be able to reduce tissue resistance to pressure by a significant factor (26).

Acknowledging that ulcer formation is complex, scientists and clinicians should be more aware of other factors contributing to the process. Factors other than the pressure under the "hard" sites must be considered and investigated when examining a patient. Such information as smoking and drinking habits, past health, present treatment, and general health of the patient affect tissue health and resistance to pressure. With respect to laboratory experiments, thresholds should be approached cautiously for the same reasons. Recently given thresholds have used absolute values that ignore individual differences in age and state of health. Thresholds can be useful and should be approached in terms of ranges and percentages, such as the ones discussed by Drummond *et al.* (11). In trying to detect patients who are at risk for pressure sores, hospitals should use behavioral information given by the Wheelchair Patient Monitor and Timer-Logger-Communicator in addition to pressure distribution measurements. Pressure distribution measurements should be made on such useful devices as the Pressure Scanner and Ischiobarograph. Hospitals and physicians should also be using diagnostic machines like computer tomograms and sinograms to obtain crucial information on the extent of an ulcer and any complica-

tions. Since pressure sore formation might begin in deeper tissues first, this might also lead to earlier detection and prevention.

With respect to measuring interface pressure, devices such as air cell and electrical transducers, which have a very small surface area and air cavity, should be pursued. Although pads might be useful, it is the opinion of these authors that total disruption of skin-cushion contact affects the pressure distribution. Ultrasound mapping of the buttock-cushion interface should be further researched for this very reason (22). Other approaches to measuring pressure, such as the Monolithic Silicon Pressure Sensor device (27), should be further researched and refined to form a more complete understanding of pressure distribution.

Few studies have resulted in a valuable, stationary sore-prevention system because these devices are limited in their pressure-relieving properties. Even the "ideal" thixotropic cushion did not show enough improvement over other cushions to warrant widespread use. Weight-shifting behavior monitors and active devices such as the one described by Rosenberg and Loch are the means that will deliver the best results.

As a final point, clinical trials should attempt to increase the number of patients studied in order to make the results more statistically significant. In addition, the placebo effect, which is not addressed in depth here, should be avoided at all costs in

validating results and conclusions. The placebo effect refers to a phenomenon where a pill or device, which is devoid of any pharmacological or curative effect, results in an improvement of a patient's disease or condition simply due to that person's belief that it works. This improvement is usually temporary, due purely to psychological factors, and can be instigated or accelerated by an enthusiastic staff. It is illustrated well in a study by Fernie and Dornan, in which a device was constructed that looked like a camera on a tripod but did absolutely nothing (14). The authors introduced the device into two large teaching hospitals and claimed that it emitted a healing electromagnetic radiation. The patients' sores healed rapidly after supposed treatment and the healing was attributed to the staff's and patients' enthusiasm since neither group knew the device was being used as a placebo. Thus, one can obtain results showing an improvement in, or healing of, pressure sores that may actually be due to psychological factors and not the drug or device being studied. To avoid this, a "double blind" study should be performed in which one group of staff and patients is given the experimental medication or system and the other (control group) is given a placebo without the groups knowing which one they received. In this way, any increased improvement observed in the experimental group over the control can be positively attributed to the experimental drug or device.

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