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Mechanical characteristics of human skin subjected to static versus cyclic normal pressures

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Abstract--Several hypotheses exist for the etiology of decubitus ulcers, with external pressures exceeding internal capillary pressures over bony prominences claimed to be the major factor. This investigation evaluated the mechanical changes that occurred in human skin as a result of its exposure to static versus cyclic normal pressures of the magnitudes earlier recorded for the heels of human subjects on various support surfaces. The skin was characterized through uniaxial tensile testing. Static pressure alone altered the tissue's mechanical properties more than dynamic pressure cycles. Tissue subjected to pressure prior to uniaxial tensile testing always was less stiff than control tissue. Damage to the initially randomly oriented tissue collagen fiber bundles in the fibrous matrix, which may occur as a result of sustained compression, may be the cause of a decrease in stiffness of tissue subjected to prior pressure loading. This is the first report of compressive-pre-load-induced strain softening (Mullins effect) of a biological material.

Key words: *bedsore, collagen, Decubitus ulcer, elastin, Mullins effect, pressure, skin, strain.*

INTRODUCTION

This research examined the mechanical effects of static versus cyclic pressures on human skin.

Uniaxial tensile testing results of control specimens were compared with results for specimens subjected to pressures previously recorded for the heels of human subjects on various support surfaces (1).

Several alternative hypotheses exist for the etiology of decubitus ulcers, but a major factor is external pressures exceeding internal capillary blood pressure. Although the scientific and engineering literature is incomplete on this point, the external pressure required to close blood capillaries at normal human arterial pressures and flows is generally accepted as approximately 32 mmHg (2). If capillary flow is obstructed by such external pressures, it is presumed that the resulting ischemia leads to the critical tissue damage that initiates the decubitus ulcer (3). There is a reported inverse relationship between the magnitudes of applied normal-to-surface forces and the amounts of time required for irreversible tissue damage to occur (4).

In addition to nominal pressure magnitudes, factors implicated in the etiology of decubitus ulcers have included the person's state of nutrition, metabolic activities, weight, degree of general health, age, the presence of incontinence, moisture, heat, skin surface friction, mechanical shear, tissue mobility, presence of infection, neurological damage, and pressure duration (5-8). The human heel has anatomic features making it one of the most susceptible sites for ulceration due to pressure-induced damage (9,10). These features include a lack of muscle to cushion the bony prominences and avascular fat.

Published studies of support systems show that most allow some tissue sites to experience pressures considerably greater than the presumed capillary closing pressure of 32 mmHg (2), and some of the cyclic or inflating and deflating (dynamic) systems, even when deflated, sustain these high contact pressures. It is of considerable importance to learn the effects of various pressures on tissues, as well as the effects of cyclic loading and unloading.

Human epidermal tissue (skin) is compliant, and generally classified as a viscoelastic material (11-14). There are differences in the skin of the same individual depending on location on the body, and there are differences between individuals (15). There are also directional effects of skin, which are explained by Langer's cleavage lines (15-18). The stress-strain relationships in uniaxial tension show skin to be stiffer along Langer's lines than across the lines. As a result, skin incisions perpendicular to Langer's lines gap more than those parallel to the lines.

The traditional mechanical characteristics of homogeneous materials are not characteristically found for skin. Skin has no unique, single Young's modulus, nor shear modulus (11). These properties for skin are not material constants, but vary depending on the strain applied. Typically biological materials have stress-strain diagrams with an elastic part which can be linear or nonlinear and an inelastic part that is history dependent (19).

Skin tensions vary regionally and with age, health, and body weight (15). Skin that has uniaxial tension applied extends in the direction of the applied force, while it contracts in a plane perpendicular to the force. If skin has uniaxial tension applied *in vitro,* there may be a progressive decrease in the total volume of the stretched specimen when fluid is physically extruded from the specimen (15). Initially, as tension is applied to skin, a small load results in extension, but this

phase is followed by a phase in which much greater loads are required for similar extensions (15,17).

Elastin fibers are thin thread-like fibers present in close association with collagen fibers in a secondary network that is interconnected with the collagen (15,20-23). This secondary network probably acts to restore the collagen to its original alignment after small forces have been applied and removed (15). Oxlund et al. (24) found that elastin fibers are responsible for the recoiling that occurs after a deformation. The network of fibers is immersed in water, proteins, and macromolecules, which function as a lubricant during deformation (22). The restoration of the collagen fibers to their relaxed state after deformation requires that the displaced fluid be replaced. *In vivo,* this replenishment probably is readily available from the neighboring tissues into which the fluid is displaced. Detached or severely stressed skin is not able to replace the fluid and thus cannot return to its original arrangement (15). In the current study, mechanical tests were conducted with the tissue in solution to provide the tissue with fluid to replace the displaced fluid when pressure relief occurred and to partially restore the alignment of the fibers.

Jenkins et al. (20) found that, although elastin fibers are always in close association with collagen fibers, collagen fibers usually outnumber the elastin fibers; also, elastin fibers were shown to have greater extensibility than collagen fibers of the same sizes. Soong et al. (25) reported that collagen will fracture at 10 percent elongation, whereas elastin will not fracture until 100 percent extension of its original length is attained.

Based mainly on observations of these different types of filaments in response to varying tensile stresses, numerous explanations have been offered for the strain-varying properties of skin. As a tensile load is applied, tangled or mesh-work collagen fibers can straighten and orient in the direction of the load. Initially, elastin fibers are thought to stretch, while collagen fibers change geometrical configuration before they become part of load resistance. As the load further increases, it is held that collagen fibers re-oriented into the direction of the load can carry a greater proportion of the load than the unoriented fibers (20). Thus, oriented fibers may also fracture before the specimen fails (25). Stark et al. (17) summarized the features of extensibility of skin as follows: 1) dermal collagen fibers lose their convolutions, 2) more fibers become aligned and parallel to the direction of the load, 3) the parallel fibers extend only with great increases in load. As an alternative, Stark et al. (17) theorized that skin tension is mainly dependent on the network of elastic fibers in the dermis, resisting movements of the body, and variations in the bulk of the tissues covered; these skin tensions dictate the behavior at cleavage lines (Langer's lines) of the skin.

Earlier, Gibson et al. (15) had described a natural mesh-like arrangement of collagen fibers in skin that allows continual rearrangement of individual fibers to resist severe stretch under minimal stresses associated with normal activity. At rest, the fibers appear randomly oriented, but when an increasing load is applied, the fibers become parallel. After a large stress is removed, the fibers will not return to their original alignment. Gibson et al. (15) showed, histologically, that fluid had been displaced from the meshwork at this point and concluded that this loss of fluid prevents the fibers from returning to their previous random arrangement.

Pressure on tissue acts as a stress, which may contribute to the formation of decubitus ulcers. In

this study the effects of pressure on the tensile properties of human skin are investigated.

MATERIALS AND METHODS

Tissue Acquisition and Preservation

Foreskin tissue was collected from newborn circumcisions, immediately placed in GIBCO (GIBCO Laboratories, Life Technologies, Inc., Grand Island, NY) keratinocyte media with gentamycin, and refrigerated in 50 ml plastic centrifuge tubes. The tissue segments varied in length from 2 to 3 cm with an average of 2.75 cm and varied in width from 0.5 to 1 cm with an average of 0.73 cm.

Bench-Top Model Experiments

A bench-scale loading system was designed to apply either static or cyclic normal pressures in a symmetrical fashion to healthy, newborn skin (see **Figure 1**). The system was designed to simulate the loading situation at the human heel, but with simplified geometry. Agar was used to simulate the compliant tissue above the bone, itself simulated by a hard spherical section (watch glass). External pressure was applied to the tissue by an identical watch glass mounted over the agar layer.

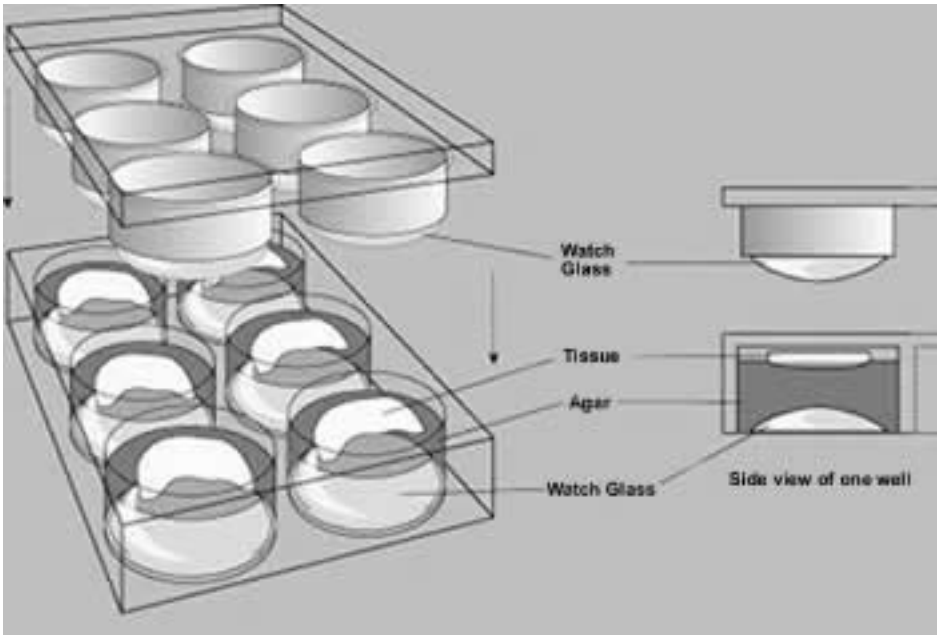

Figure 1.
Bench-top model.

Each tissue specimen was placed in its own well of a six-well polystyrene tissue-culture plate. Centered within each well bottom, under the agar, was an inverted 2.5-cm watch glass, sealed in place with paraffin. The agar was poured until it was within 5 mm of the top of the well, providing a pseudo-tissue support matrix of 15 mm over the rigid glass, convex face up. After the agar cooled to 38 °C, the neonatal skin was divided longitudinally, opened, and placed over the center of the agar with the epithelium facing up. Liquid nutritional media was added on top of the

tissue in the well and the tissue was kept moist and viable throughout the experiment. After the agar cooled further, it secured the tissue in place over the agar/watch glass combination that simulated an anatomical bony prominence.

The tissue was contacted from above by the convex face of an identical 2.5-cm watch glass. This was accomplished by use of the top of a six-well tissue culture dish having plastic mounts (50-ml centrifuge tube lids) cemented to its surface. Since the watch glasses were smaller in diameter than the wells, they fit easily.

A small motor was used to raise and lower the top of the tissue culture dish, providing cyclic pressures to the tissues. Clamps held the lids and plates in constant registry. The lid above the six-well plate was simply raised or lowered to increase or decrease the maximum pressure at the interface. Cyclic pressure was applied as 20 compressions and lifts per minute, with pressure constant for 2 s per cycle. This loading was based on the loading patterns of the dynamic systems previously used (1). Continuing static pressure was applied by adjusting the height of the lid to the desired loading, and holding the lid at that pressure until the end of the experiment. The tissue was under negligible tension during compressive loading. Pressure sensors and gauge combinations were incorporated in each experiment to monitor the pressures applied to the tissues. Sensors were placed in each well to monitor the interface pressure.

Prior to mechanical testing, each tissue specimen was subjected to one of three compressive loading conditions using the bench-top device. The choice of 4-hr experimental periods was based on results from a pilot study, first done to examine the microstructural integrity of isolated (unpressurized) epidermal tissues after 4, 8, 12, 16, 20, 24, 28, and 32 hours on agar with media at room temperature, the results of which indicated that considerably more than 4 hrs were required before noticeable microstructural changes spontaneously appeared. The three loading conditions produced pressure sensor values of 1) 50 mmHg, 2) 170 mmHg, and 3) 110-170 mmHg of dynamic (cyclic) pressure. These groups were chosen based on prior experiments using human subjects resting on pressure-relief systems. The three groups represented average interfacial pressure values recorded under the heels of human subjects laying on pressure relief systems. The 50 mmHg and 170 mmHg groups represent static system values and the 110-170 mmHg represent the average cycle values recorded on the dynamic systems. Control specimens that had not been subjected to any externally applied pressure also were tested. All tests were repeated in triplicate, using tissue specimens from different donors, and all stored in nutrient solution for 24-48 hrs to allow maximum relaxation from pre-load challenges before mechanical challenge.

Sensor and Sensor-Gauge Selection

A PSM-1 pressure gauge and numerous custom-fabricated PSP-1 pressure sensors (Gaymar Industries Inc., Orchard Park, NY), designed to be used with this gauge, were employed to measure the tissue interface pressures. The sensors are flat, flexible, polyvinylchloride plastic envelopes with copper contact strips diagonally attached to their opposite inner walls. When the cross-oriented copper strips come into contact at their centers, they close an electrical circuit, connected by wires from the copper strips through the air tube to the gauge. Air pressure is supplied from the PSM-1 gauge to one end of the otherwise-sealed envelope, inflating the sensor envelope until a pressure equal and opposite to that closing the sensor is achieved (26). When the pressure exceeds the applied pressure, the copper strips lose contact and the pump turns off,

allowing the pressure to fall until the strips touch and the air pump turns on again (26). This provides null pressure readings where the measuring devices have minimal mechanical interference in the tissue/support-surface interface.

The PSM-1 gauge incorporates a conventional mercury manometer, connected inline with the sensor and the pump, to allow the null measurement of tissue interface pressure to be read directly (26). As the sensor contact open-close events repeat, the fluttering manometer readout typically fluctuates by less than ±2 mmHg. The sensor bladder thickness at this condition is <1 mm, minimizing the mechanical influence of the measuring device.

The accuracy of interfacial pressure measurement systems has been previously evaluated in this laboratory. Both contact geometry and surface compliance can have an effect on both the gauge and sensor. Geometries closest in size to the sensor itself are most accurate. In these experiments the tissue size was close to sensor size. The compliance of the tissue and agar combination is within the range of compliancies producing the most reliable results.

Mechanical Testing

Uniaxial Tensile Testing

Control and precompressed epidermal tissue was laid flat so that a rectangle 6 mm wide could be cut from its central area, with a scalpel around a polymethylmethacrylate (PMMA) template of that dimension on a PMMA cutting surface. Tissue length was variable and ranged from 2 to 3 cm. Pilot studies showed minimal effects of length on the uniaxial test results. The tissue was kept moist and alive throughout by intermittent bathing with nutritional media during these procedures. Each rectangle of tissue was placed in a PMMA jig, 10×38 mm, with 3-mm diameter holes 3 mm from each end. PMMA tabs, 9×9 mm, were attached using cyanoacrylate cement so that they were aligned with the edges of the jig. The tissue rectangle was turned over and a second set of tabs attached, aligned with the first set and with the tissue between the two sets.

A PMMA plastic guide was placed on top of the mounted specimen while it was still in the jig and 3-mm holes were drilled through both sets of external tabs and the tissue they confined (see **Figure 2**). Thickness and width measurements were made with a caliper. Both measurements were taken in the midregion of the specimen away from the tabs at each end. The tissue was tested on a mechanical tensile tester (Columbia Labs, Buffalo, NY), employing a 1-kg loadcell with custom-made universal joint attached (27). The distance from the sample to the load cell was approximately 25 times the length of the sample itself, thus any possible misalignment angle was minimized. A chart recorded force and time, proceeding from a baseline condition in which the tissue was pre-stressed with a 6-gm weight and the gauge length measured. The machine's grips consisted of two 3-mm stainless steel bolts with stainless steel nuts. The tabs with tissue between them were placed with the bolts extending through the holes in the tissue and tabs and the nuts were used to securely fasten the grips to the sample. This technique for sample tensile testing is based on years of grip development for soft tissue testing and is the most atraumatic for thin soft tissue. The tabs are used to exert the force and by using this technique no slippage will occur between the tissue and the grips. Also, the free edge of the sample adjacent to the grip surface receives a uniform stress. The use of rectangular strips of tissue ensures a uniform stress on the sample, which allows crosshead displacement to be directly related to sample strain. During the test the sample can be readily observed and if there is any pulling from an edge the run is

discarded. For low stress/strain work, rectangular strips of tissue are used by our laboratory instead of dog bone shaped samples, which are customarily used in our laboratory for determining break stress.

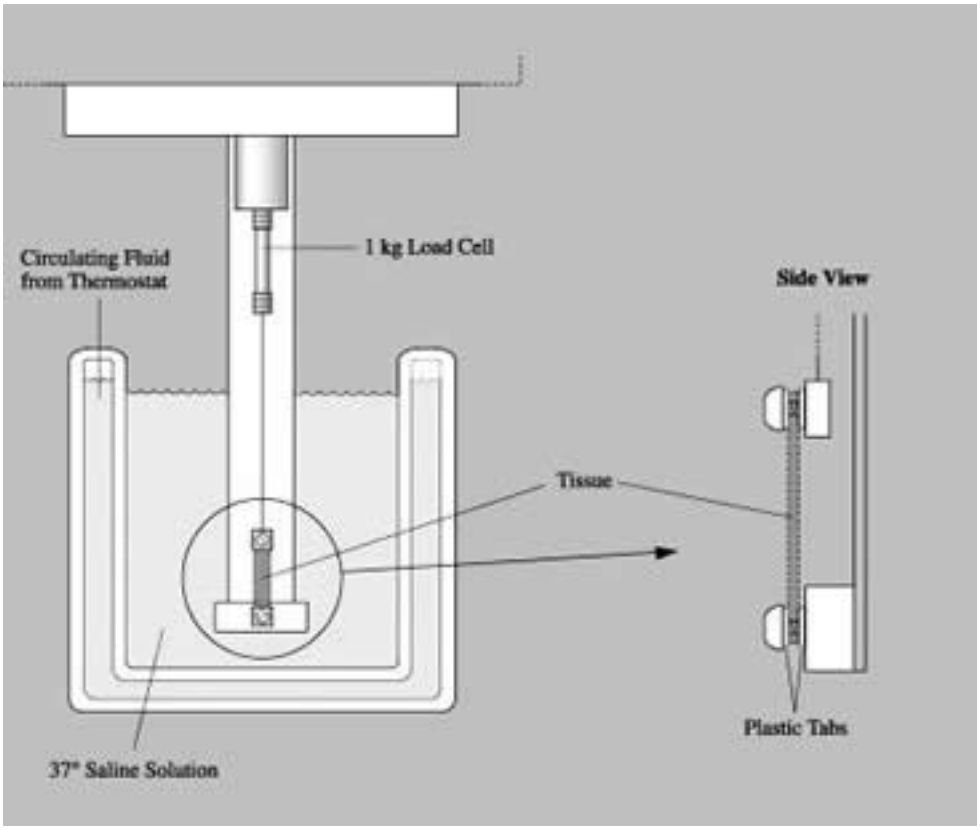

Figure 2.
Mechanochemical tensile tester.

The experiments were conducted at 37 °C in physiologic saline, conditions chosen to simulate a temperature within the range of the natural tissue environment. The temperature of the outer surface epidermal tissue varies depending on the surface the tissue is resting on, if any. Although the temperature of the surface epidermal tissue may exceed 37° in some cases, the outer surface epidermal tissue may be at a temperature less than 37° when exposed to air, the dermis and underlying tissue are at 37 °C in the tissue's natural environment.

RESULTS

Mechanical Testing

As a general finding, all these tests illustrated a diminution of the skin's elastic, compliance, and strength characteristics as a consequence of its prior exposure to compressive force.

Uniaxial Tensile Tests

Control Specimens Uniaxial tensile tests were run at various rates on control tissue specimens. The tests were run on several segments of tissue. The results were very similar when the several

rates were run on a single piece of tissue and when each rate was run on a different piece of tissue. A single segment of tissue that had not been exposed to compressive stress was tested at various rates and served as the control.

The deformation rates used for the uniaxial tensile test controls were 10.6 mm/min, 21.2 mm/min, 31.8 mm/min, and 42.3 mm/min (see **Figure 3**). As the deformation rate of the uniaxial tensile test increased, the percent strain did not significantly increase. At lower deformation rates, the tissue displayed greater apparent stiffness. This observation of strain softening, long known as the Mullins effect, has recently been noted for cardiovascular tissue as well (28).

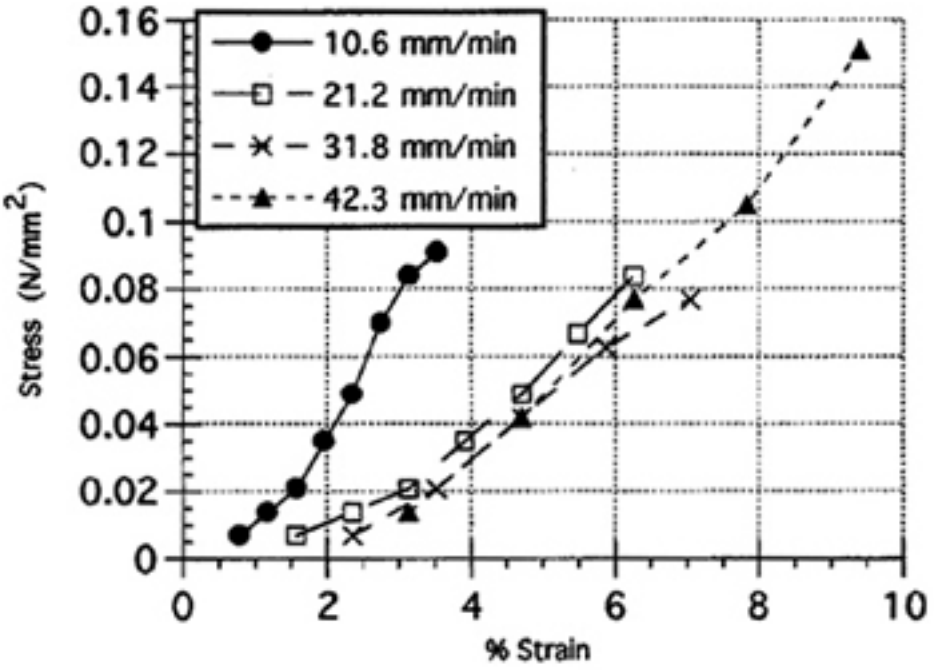

Figure 3.
Uniaxial tensile controls.

Experimental Specimens

Based on the results from the controls, 31.8 ±1 mm/min was chosen as the deformation rate at which to run the remainder of the uniaxial tensile tests. The uniaxial tensile test results for the tissue subjected to 50 mmHg and 170 mmHg static sensor pressure and 110-170 mmHg dynamic (cyclic) sensor pressure are shown in **Figure 4.** Each of the graphs shows a typical result from a series of replicate tests on single segments of tissue from different donors, where the results were similar but not identical. The tissue was much less stiff than the control after exposure to 170 mmHg static sensor pressure for 4 hrs. The tissue retained most of its control qualities after being subjected to only 50 mmHg static sensor pressure, but even these experimental tissues were less stiff than the uniaxial tensile control tissues not subjected to external pressure prior to testing. This is the first report of compressive-pre-load-induced strain softening (Mullins effect) of a biological material.

DISCUSSION

The uniaxial tensile controls and experimental groups in this study produced stress/strain curves of the same characteristic shape seen by previous investigators (16,29,30). At greater deformation rates in the uniaxial tensile control groups, the stiffnesses decreased (see **Figure 3**). Fung (31) reported the opposite finding, but he used papillary muscle from rabbits for his experiments. Vogel (32,33) also reported an increase in stiffness when strain rate was increased, but his research was done with rat skin. The decrease in stiffness seen with an increase in strain rate is explained by the Mullins effect, a strain-softening noted in complex multicompartmented synthetic and natural composites. Mullins effect or stress softening is the phenomenon shown when the load required to produce the same stretch in the second loading of a specimen loaded uniaxially, unloaded, and loaded again, is smaller than the load initially required (33). The Mullins effect has been shown in rubber, filler-reinforced vulcanized rubber, rubber-like materials, and rat left ventricular myocardium tissue (28).

The tissue exposed to 50 mmHg static sensor pressure (see **Figure 4**), was the stiffest of the experimental compressive preload groups, but less stiff than any of the controls. The tissue exposed to 170 mmHg static sensor pressure was the least stiff of any of the experimental groups and much less stiff than any of the controls. The 110-170 mmHg dynamic pressure-exposed specimens were of intermediate stiffness. Increased static pressure always decreases the stiffness of epidermal tissue.

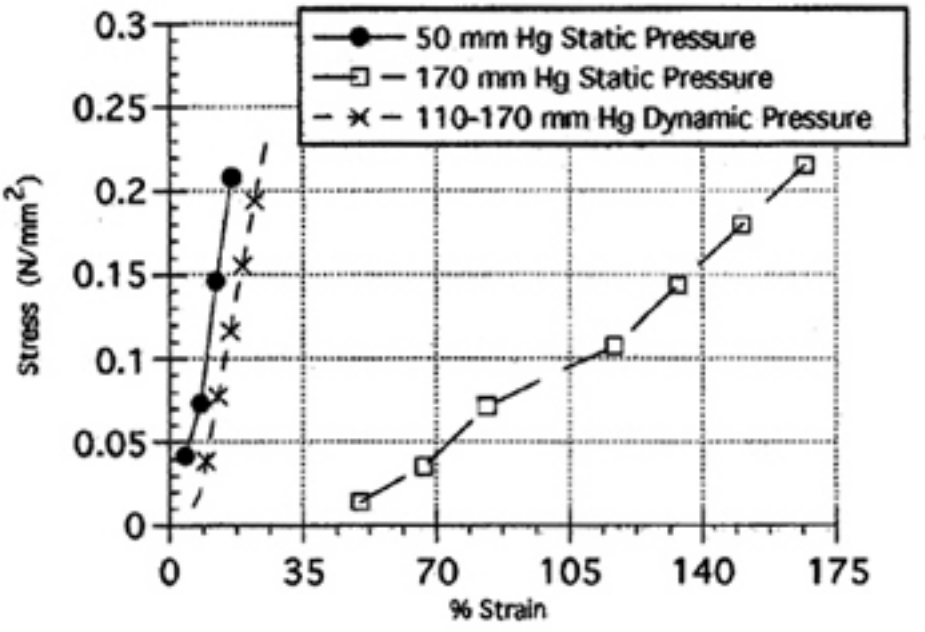

Figure 4.
Uniaxial tensile data for tissue subjected to static or dynamic pressure.

Although all the presently reported experiments were conducted on healthy neonatal tissue, these findings indicate a significant alteration of the tissue's mechanical properties and thus very serious complications in regard to the tissue's ability to sustain potentially damaging mechanical loads. Human foreskin tissue differs from the heel region in that the skin is not as callused, but both

tissues are from regions with little fat present. Also, the epidermal thickness of both tissues are similar. It is difficult to find a reference tissue that is both available and suited to experiments of this nature, but neonatal tissue provided a suitable model for this study. Currently studies are underway in our laboratory using aged skin and skin adjacent to decubitus ulcers to look for the same effects seen in the neonatal tissue model.

Implications For Prevention of Decubitus Ulcers

Compressed living skin, of course, generally becomes only locally thinner while adjacent areas swell around the regions of force application, as in the imprinting of the shape and topographic features of objects placed into forceful contact with the skin. The epidermal tissue used for these experiments was not perfused and thus lacked restorative fluidic pressures, which could potentially have aided in reducing the visible compression of the tissue after application of pressure. This might have occurred, inadvertently, in the case of the 110-170 mmHg dynamic sensor pressure because it is possible that during the lower pressure portion of the cycle, restorative fluids could have re-entered the tissue from the nutrient bathing medium. Daly (34) showed, using cadaver skin, that compression resulted in large volume changes in the skin as a result of fluid transport.

In this study the lowest pressures used in the bench-top model were 50 mmHg static sensor pressure and the controls. Since 32 mmHg is the clinical guideline used to test pressure sore prevention products, and it is accepted in the field that clinical effects will often be seen at tissue pressures greater than 32 mmHg (33-37), the value of 50 mmHg was chosen based on both this reference minimum and on the heel interface pressure data collected by Edsberg (1) for relevance to that specific anatomic site.

Dynamic pressure cycles to at least 170 mmHg do not compromise tissue mechanical properties, compared to unloaded controls, as much as does sustained static pressure. Tissue subjected to sustained pressure is less stiff than control tissue suggesting a compressive load-induced strain softening (Mullins effect).

Sangeorzan et al. (38) found that skin over bone was significantly stiffer than skin over muscle. In particular, for pressures less than 20 mmHg the skin over bone was 2.5 times stiffer, but for pressures greater than 40 mmHg it was 7 times stiffer. The findings in our current study show a decrease in tissue stiffness after exposure to static or cyclic normal pressures. Decubitus ulcers typically form in tissue over boney prominences. The decrease in tissue stiffness in an area normally associated with great stiffness indicates a serious impairment in the tissue's mechanical properties.

Krouskop (39) has proposed a hypothesis of decubitus ulcer formation directly related to the tissue's ability to distribute and support the load. When this function is compromised, the interstitial fluid is disrupted and cell-to-cell contact can occur, capillaries may burst, and lymphatic failure may occur. These changes in the tissue may lead to decubitus ulcer formation.

The specific role of pressure in the development of a decubitus ulcer is unknown, but the findings of this study suggest that pressure has a direct effect on the mechanical properties of

skin. Alterations in the skin's mechanical properties can have serious implications in regards to the tissue's ability to sustain potentially damaging mechanical loads. Changes in the mechanical properties of skin may contribute to the formation of decubitus ulcers.

Studies currently underway in our laboratory are examining the microstructural effects of pressure on human skin. Also, the microstructural and mechanical effects of pressure on aged skin or skin more characteristic of patients at risk for decubitus ulcer formation, as well as from tissue at and adjacent to decubitus ulcers is currently being examined.

REFERENCES

1. Edsberg LE. Comparative measurements of interfacial pressures between tissue sites and various support systems (thesis). Buffalo, NY: University at Buffalo; 1992.
2. Maklebust J, Mondoux L, Sieggreen M. Pressure relief characteristics of various support surfaces used in the prevention and treatment of pressure ulcers. J Enterostom Ther 1986;13:85-9.
3. Krouskop TA, Noble PC, Garber SL, Spencer WA. The effectiveness of preventative management in reducing the occurrence of pressure sores. J Rehabil Res Dev 1983;20 (1);74-83.
4. Brand PW. Pressure sores--the problem. In: Bedsore biomechanics. Baltimore, MD: University Park Press; 1975. p. 19-23.
5. Berjian RA, Douglass HO, Holyoke ED, Goodwin PM, Priore RL. Skin pressure on various mattress surfaces in cancer patients. Am J Phys Med 1983;62:217-26.
6. Elliot TM. Pressure ulcerations. Am Fam Phys 1982:25:171-80.
7. Kosiak M. Etiology and pathology of ischemic ulcers. Arch Phys Med Rehabil 1959;40:62-9.
8. Longe RL. Current concepts in clinical therapeutics: pressure sores. Clin Pharm 1986;5:669-81.
9. Crandall RC, Wagner FW. Partial and total calcanectomy. J Bone Joint Surg 1981;63-A:152-5.
10. Sugarman B, Hawes S, Musher DM, Klima M, Young EJ, Pircher F. Osteomyelitis beneath pressure sores. Arch Int Med 1983;143:683-8.
11. Cook TH. Mechanical properties of human skin with aging. In: Balin AK, Kligman AM, editors. Aging and the skin. New York: Raven Press; 1989. p. 205-25.
12. Pereira JM, Mansour JM, Davis BR. Technical note: dynamic measurement of the viscoelastic properties of skin. J Biomech 1991;24:157-62.
13. Vogel HG. Measurement of some viscoelastic properties of rat skin following repeated load. Connect Tissue Res 1976;4:163-8.
14. Fung YCB. Biomechanics mechanical properties of living tissues. New York: Springer-Verlag; 1981. p. 203\12.
15. Gibson T, Kenedi RM, Craik JE. The mobile micro-architecture of dermal collagen. Br J Surg 1965;52:764-70.
16. Ridge MD, Wright V. The directional effects of the skin. J Invest Dermatol 1966b;46:341-6.

17. Stark HL. Directional variations in the extensibility of human skin. Br J Plas Surg 1977;30:105-14.
18. Schneider DC, Davidson TM, Nahum AM. In vitro biaxial stress-strain response of human skin. Arch Otolaryngol 1984;110:329-33.
19. Veronda DR, Westmann RA. Mechanical characterization of skin-finite deformations. J Biomech 1970;3:111-24.
20. Jenkins RB, Little RW. Constitutive equation for parallel-fibered elastic tissue. J Biomech 1974;7:397-402.
21. Dunn MG, Silver FH. Viscoelastic behavior of human connective tissues: relative contribution of viscous and elastic components. Connect Tissue Res 1983;12:59-70.
22. Agache PG, Monneur C, Leveque JL, De Rigal J. Mechanical properties and Young's modulus of human skin in vivo. Arch Dermatol Res 1980;269:221-32.
23. Cotta-Pereira G, Rodrigo FG, Bittencourt-sampaio S. Oxytalan, elaunin, and elastic fibers in the human skin. J Invest Dermatol 1976;66:143-8.
24. Oxlund H, Manschot J, Viidik A. The role of elastin in the mechanical properties of skin. J Biomech 1988;21:213-8.
25. Soong TT, Huang WN. A stochastic model for biological tissue elasticity in simple elongation. J Biomech 1973;6:451-8.
26. Stewart TP. Use of the Gaymar pressure gauge and electropneumatic sensor. Orchard Park, NY: Gaymar Industries, Inc.; 1984.
27. Lauren M. Mechano-chemical testing of tissue and resorbable materials. Proceedings of the 1993 Society for Biomaterials Annual Meeting; 1993 Apr 29, Birmingham, AL; 1993. p. 320.
28. Emery JL, Omens JH, McCulloch AD. Strain softening in rat left ventricular myocardium. Adv Bioeng 1994;28:15-6.
29. Ridge MD, Wright V. Mechanical properties of skin: a bioengineering study of skin structure. J Appl Physiol 1966a;21:1602-6.
30. Bader DL, Bowker P. Mechanical characteristics of skin and underlying tissues in vivo. Biomaterials 1983;4:305-8.
31. Fung YCB. Stress-strain-history relations of soft tissues in simple elongation. In: Fung YC, Perrone N, Anliker M, editors. Biomechanics its foundations and objectives. New Jersey: Prentice-Hall, Inc.; 1972. p. 181-208.
32. Vogel HG, Hilgner W. The 'Step phenomenon' as observed in animal skin. J Biomech 1979;12:75-81.
33. Johnson MA, Beatty MF. A constitutive equation for the Mullins effect in stress controlled uniaxial extension experiments. Continuum Mech Thermodyn 1993;5:301-18.
34. Daly CH. Biomechanical properties of dermis. J Invest Dermatol 1982;79:17s-20s.
35. Krouskop TA, Garber SL. Interface pressure measurements (letter). Osteotom Wound Manage 1989a;24:12,14.
36. Krouskop TA, Garber SL. Interface pressure confusion (letter). Decubitus 1989b;2:8.
37. Stewart TP. Another opinion on interface pressure (letter). Decubitus 1989;2:8-10.
38. Sangeorzan BJ, Harrington RM, Wyss CR, Czerniecki JM, Matsen FA. Circulatory and mechanical response of skin to loading. J Orthop Res 1989;7:425-31.
39. Krouskop TA. A synthesis of the factors that contribute to pressure sore formation. Med Hypotheses 1983;11:255-67.

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