A Reprint from Surgical Technology International XXVI

Oxy-Mat™ Mattress System Development Utilizing Simultaneous Measurement of Interface Pressure and Deep Tissue Oxygen Saturation

Glenn J. Butler, CHT, CEO
Founder Partner and Chief Engineer

David J. Kenyon, BME
Principal Engineer

Edward Golembe, MD, UHM, FACCWS
Director, Surgical Affairs Consultant

Scott Gorenstein, MD, UHM, FECEP
Clinical Director, Medical Affairs Consultant

Bok Lee, MD
Professor, Clinical Research Chief

Thomas Davenport, MD, FACS
Director

Jacques Vieweg
Test Program Engineer

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GLENN J. BUTLER, CHT, CEO
LIFE SUPPORT TECHNOLOGIES GROUP
FOUNDER, PARTNER, AND CHIEF ENGINEER
OFF-LOADING TECHNOLOGIES, INC.
TARRYTOWN, NY

DAVID KENYON, BME
PRINCIPAL ENGINEER
HAMILTON RESEARCH, LTD.
TARRYTOWN, NY

EDWARD GOLEMBE, MD, UHM, FACCWS
DIRECTOR
SKIN INTEGRITY SERVICE
WESTCHESTER MEDICAL CENTER
VALHALLA, NY
SURGICAL AFFAIRS CONSULTANT
LIFE SUPPORT TECHNOLOGIES GROUP
TARRYTOWN, NY

SCOTT GORENSTEIN, MD, UHM, FACEP
CLINICAL DIRECTOR
DIVISION OF REGENERATIVE MEDICINE
WINTHROP UNIVERSITY HOSPITAL
MINEOLA, NY
MEDICAL AFFAIRS CONSULTANT
LIFE SUPPORT TECHNOLOGIES GROUP
TARRYTOWN, NY

BOK LEE, MD
PROFESSOR
DEPARTMENT OF SURGERY
NEW YORK MEDICAL COLLEGE
VALHALLA, NY
CLINICAL RESEARCH CHIEF
LIFE SUPPORT TECHNOLOGIES GROUP
TARRYTOWN, NY

THOMAS DAVENPORT, MD, FACS
DIRECTOR
SURGICAL WOUND CARE
WINTHROP UNIVERSITY HOSPITAL
MINEOLA, NY

JACQUES VIEWEG
TEST PROGRAM ENGINEER
LIFE SUPPORT TECHNOLOGIES GROUP
TARRYTOWN, NY

ABSTRACT

The development and management of pressure ulcers (PUs) among hospital and nursing home patients is one of the greatest preventable challenges to healthcare worldwide. For over 50 years, pressure mapping and subjective comfort have been the primary indicators for mattress selection. Our research demonstrates that mattress/patient interface pressure and relative blood/oxygen perfusion do not inversely correlate and pressure is not a meaningful, real-time indicator of tissue ischemia and risk of pressure ulcer development. Developed in our research is a real-time sensor system to simultaneously measure and record these parameters over the

anatomical sites at risk of PUs. Measurements focused on the heel, sacrum, trochanter, ischium, scapula, and occipital. A modified pressure mapping system is used for interface pressure measurements and integrated with multiple, near-infrared sensors to measure specific deep-tissue hemoglobin saturated oxygen or rSO2. Testing and mattress design development was done during the period of 2007 to present. Over 200 human tests using 16 commercially available mattresses were conducted in supine, 30-degree, and 70-degree positions, ranging in times of up to four hours. During this time period, we utilized 20 test subjects–8 female and 12 male–with ages ranging from 18 to 65 years. The result of this proprietary off-loading device evaluation and design system shows that the Oxy-Mat™ (Off-Loading Technologies, Tarrytown, NY) Non-Powered Mattress System consistently provides optimized tissue perfusion as measured by natural deep-tissue oxygen saturation levels. In extensive laboratory and clinical evaluations, the Oxy-Mat™ was shown to be functionally superior to CMS Group 2 powered mattresses. Another outcome of our research was that a powered mattress system may not be appropriate for most sensate and semi-ambulatory patients. Further research is underway.

INTRODUCTION

Pressure Ulcers—A Pivotal Clinical Problem

The development of pressure ulcers among compromised hospital, nursing home, and extended care patients remains one of the greatest clinical challenges and ongoing costs to healthcare services worldwide. Increased morbidity and mortality resulting from pressure (decubitus) ulcers, deep-tissue injury, sepsis, increased length of hospital stay, and hospital readmissions are common complications.[1]

For over 50 years, patient/mattress interface pressure mapping has been the primary indicator for mattress selection. Future clinical mattress selection criteria must include the application criteria of such products based on documented scientific principles and research.[2-4] Compromised patients restricted to wheelchairs or total bed confinement are at greatest risk of pressure-induced ischemia, resulting in hypo-cellular function and pressure ulcer development from prolonged pressure-loading on bony prominences from skin friction and shear.[5,6] Tissue ischemia, hypoxia, and resultant reperfusion injury are known to be primary contributors to the formation of pressure ulcers.[7,8] Such injury to tissues is less likely when patient/mattress interface pressure exerted on the body is evenly distributed over time, and when deep-tissue oxygenation tensions (saturation values) remain above 40 mmHg.[9] Insensate, diabetic, obese, and paraplegic patients with significant co-morbidities are, therefore , at greatest risk for pressure ulcer development.[10]

Hospitalization for ulcers are on the rise. "...Between 1993 to 2006, the total number of hospitalizations related to pressure ulcers increased by nearly 80 percent."[11] "Adult hospital stays noting a diagnosis of pressure ulcers totaled $11.0 billion in 2006"[2,11] and an estimated 60,000 patients die each year as a result of pressure ulcers.[1,12]

In October 2008, the Centers for Medicare/Medicaid Services (CMS) made a decision not to provide DRG in-patient payments for hospital/nursing home acquired pressure ulcers (stage III/or greater) or related infections.[13] This places potentially overwhelming costs directly back to Medicare/Medicaid provider institutions.

The lifetime cost of pressure ulcers, patient suffering, related co-morbidities, and finally mortality are among the greatest preventable challenges to CMS and the commercial healthcare community. Any new risk assessment criteria superior to the Norton and Braden scales, clinical procedure, or mattress system that can measurably reduce the incidence of pressure ulcers at a lower cost should be a top priority for the international healthcare community.[14]

In this article, we will describe our research to couple the deep-tissue oxygenation saturation measurements, critical for healthy skin and organs, with mattress/skin pressure measurements; pressure measurements alone will not guide mattress development to an effective solution.

PATHOPHYSIOLOGY OF PRESSURE ULCER DEVELOPMENT

It is widely accepted that the prime causal factor for the development of pressure ulcers consists of excessive tissue pressure-loading sustained for time periods sufficient to induce pressure-prone tissue to become ischemic, then hypoxic, leading to reperfusion injury and necrosis.[15,16]

Since nearly all patients are in bed for eight hours or more, the mattress system selected for clinical use becomes a significant variable in the reduction and/or relief of pressure on the patient's body, particularly over bony prominences. Any increase in mechanical stress (pressure and shear) further affects the availability of nutrients, such as oxygen, to susceptible tissues.

Ischemia leading to hypoxia is the result of decreased blood flow to cutaneous tissue after prolonged periods of elevated tissue interface pressure. Any resulting reperfusion injury causes

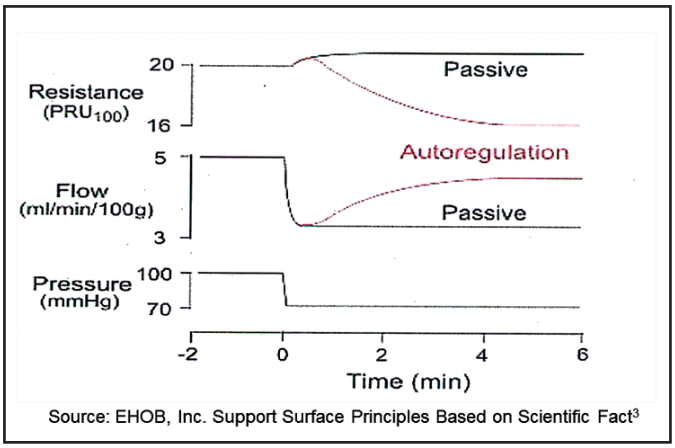


Figure 1. The effects of suddenly reducing perfusion pressure.

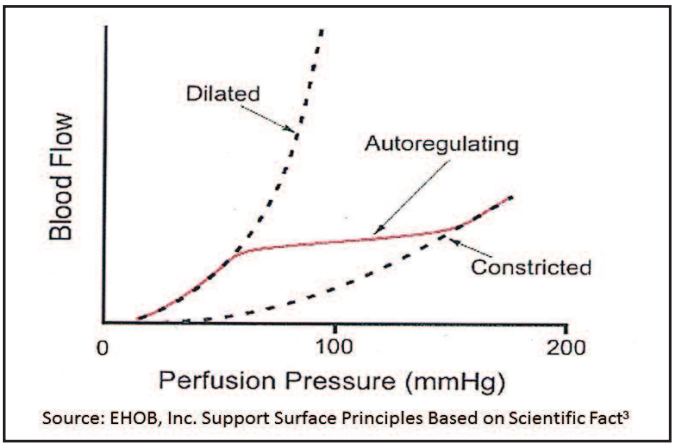


Figure 2. Auto-regulatory responses.[3]

neutrophil capillary endothelium adherence, cell rolling, sludging, and clotting that inhibits/occludes blood nutrient and oxygen supply.[16] This vicious ischemia/reperfusion cycle (Fig. 4) decreases blood flow and increased hypoxia forces tissue cells to use anaerobic pathways to produce ATP energy which causes more lactic acid to accumulate, resulting in greater acidosis, as well as increased quantities of hydrogen ions and more potassium around the cell.[17,18]

In normal individuals, this biochemical cascade of metabolites/oxygen radicals should lead to nitric oxide (NO) release and other vessel vasodilatation up-regulators that promote more blood with oxygen and nutrients to the tissues. This helps to mitigate pressure-prone tissue ischemia and hypoxia.[19]

A review of these physiological mechanisms is in order to fully appreciate why deep-tissue oxygen tensions are the only meaningful real-time indicator for pressure ulcer prevention and in evaluating mattress system efficacy.

AUTO-REGULATION OF BLOOD FLOW[3]

Auto-regulation is a manifestation of local blood flow regulation. It is defined as the intrinsic ability of an organ to maintain a constant blood flow despite changes in perfusion pressure. For example, if perfusion pressure is decreased to an organ (e.g., by partially occluding the arterial supply to the organ), blood flow initially falls, then returns toward normal levels over the next few minutes.

This auto-regulatory response occurs in the absence of neural and hormonal influences and therefore is intrinsic to the organ. When perfusion pressure (arterial minus venous pressure) initially decreases, blood flow falls because of the relationship between pressure, flow, and resistance. When blood flow falls, arterial resistance falls as the resistance vessels (small arteries and arterioles) dilate. Many studies suggest that metabolic, myogenic, and endothelial mechanisms are responsible for this vasodilation. As resistance decreases, blood flow increases despite the presence of reduced perfusion pressure.

Figure 1 shows the effects of suddenly reducing perfusion pressure from 100mmHg to 70mmHg in a passive vascular bed; one that does not show auto-regulation. This will result in a rapid and sustained fall in blood flow. In fact, the flow will fall more than the perfusion pressure because of passive constriction. As the intravascular pressure falls, it is represented by a slight increase in resistance in the passive vascular bed.

If a vascular bed is capable of

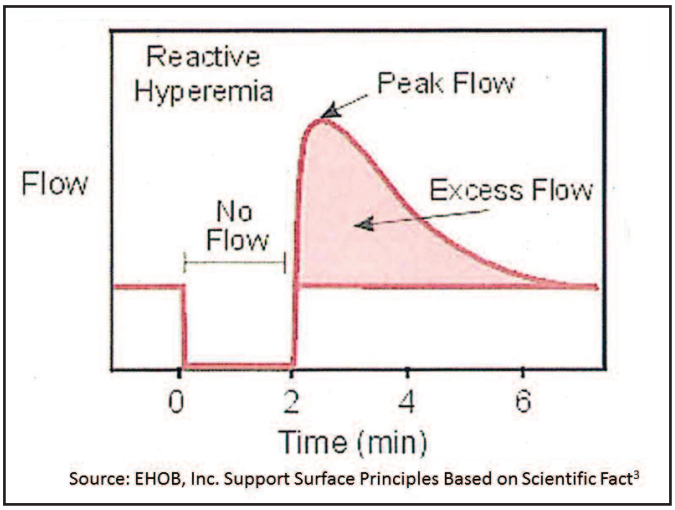


Figure 3. The effects of a two minute arterial occlusion.

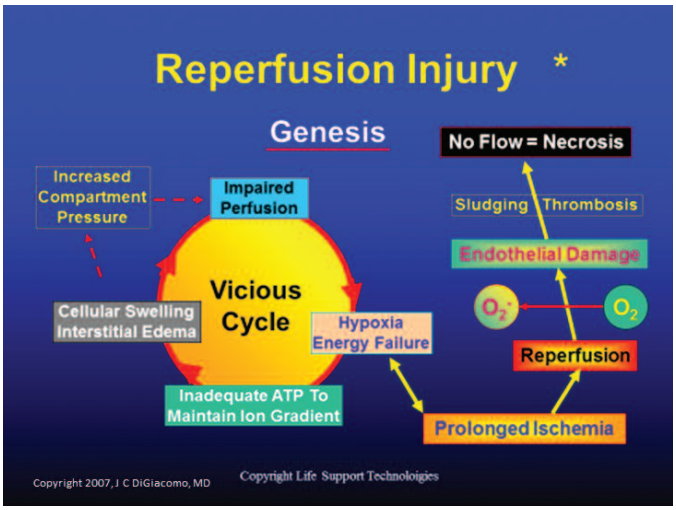


Figure 4. When repetitive hyperemia leads to ischemia.

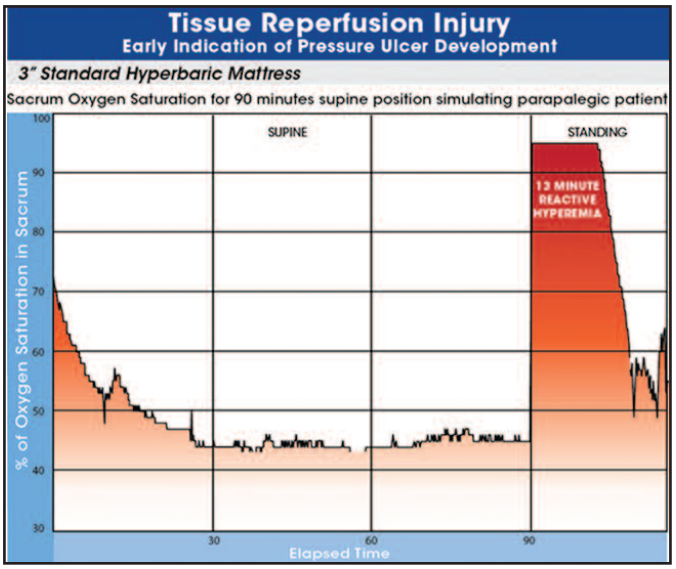


Figure 5. Graphical representation of an ischemic\reperfusion injury. Copyright 2015 Life Support Technologies Group.

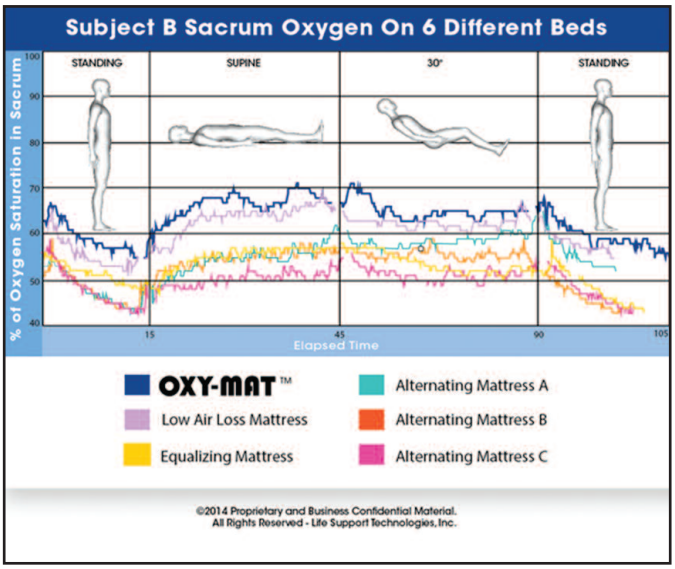


Figure 6. Normal active hyperemia oxygen signature.

undergoing auto-regulation, then after the initial fall in perfusion pressure and flow, the flow will gradually increase (red line) over the next few minutes as the vasculature dilates (resistance decreases—red line).

After a few minutes, the flow will achieve a new steady-state level. If a vascular bed has a high degree of auto-regulation (e.g., brain and coronary circulations), then the new steady-state flow may be very close to normal despite the reduced perfusion pressure.

If a tissue is subjected to an experimental study[3] in which perfusion pressure is both increased and decreased over a wide range of pressures, and the steady-state auto-regulatory flow response measured, then the relationship between steady-state flow and perfusion pressure can be plotted as shown in Figure 2. The red line represents the auto-regulatory responses in which flow changes relatively little despite a large change in perfusion pressure.

If a vasodilator drug is infused into an organ so that it is maximally dilated and incapable of auto-regulatory behavior, the curve labeled "Dilated" is generated as perfusion pressure is changed. It is non-linear because blood vessels passively dilate with increasing pressures, thereby reducing resistance to flow.

When the vasculature is not maximally dilated, many organs will display auto-regulation as the perfusion pressure is reduced. When this occurs, there will be a range of perfusion pressures (i.e., auto-regulatory range) where flow may not decrease appreciably as perfusion pressure is reduced.

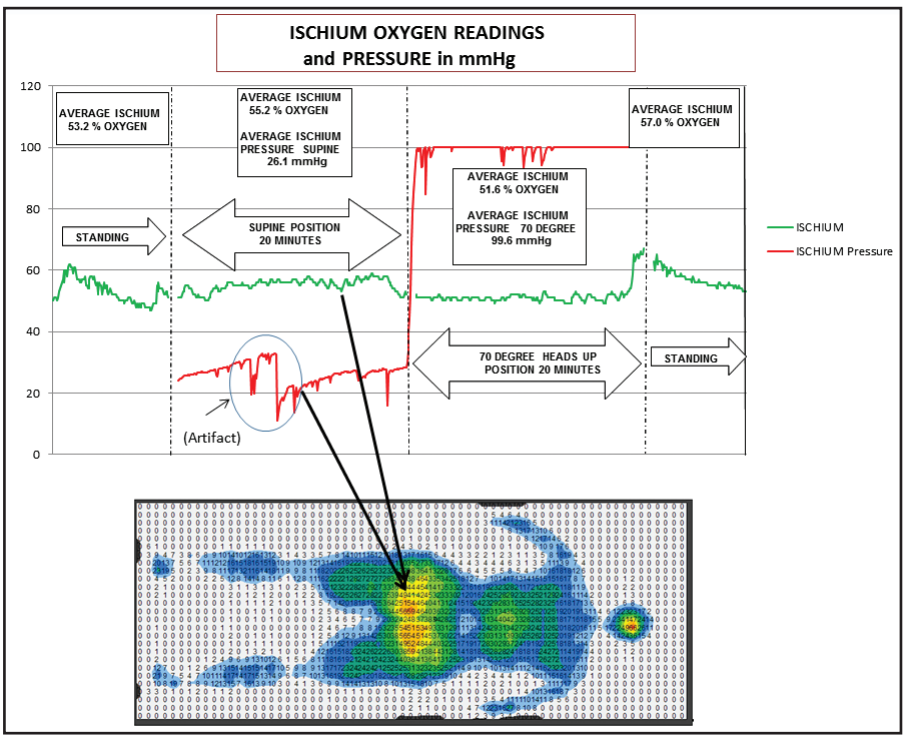


Figure 7. Pressure and the relationship to deep tissue oxygen saturation. Copyright 2015 Life Support Technologies Group.

ACTIVE HYPEREMIA[3]

Active hyperemia is a normal physiological process that automatically compensates for reductions in blood flow due to transient vessel occlusions and increased tissue-interface pressures, such as prolonged sitting in healthy persons.

As persons become older, develop co-morbidities such as diabetes, associated neuropathy, paraplegias, or compromised mentation, they become less able to initiate normal autonomic active hyperemia and become more susceptible to pressure-related hypoxia, leading to ischemia.

Active hyperemia is the increase in organ blood flow *(hyperemia)* that is associated with increased metabolic

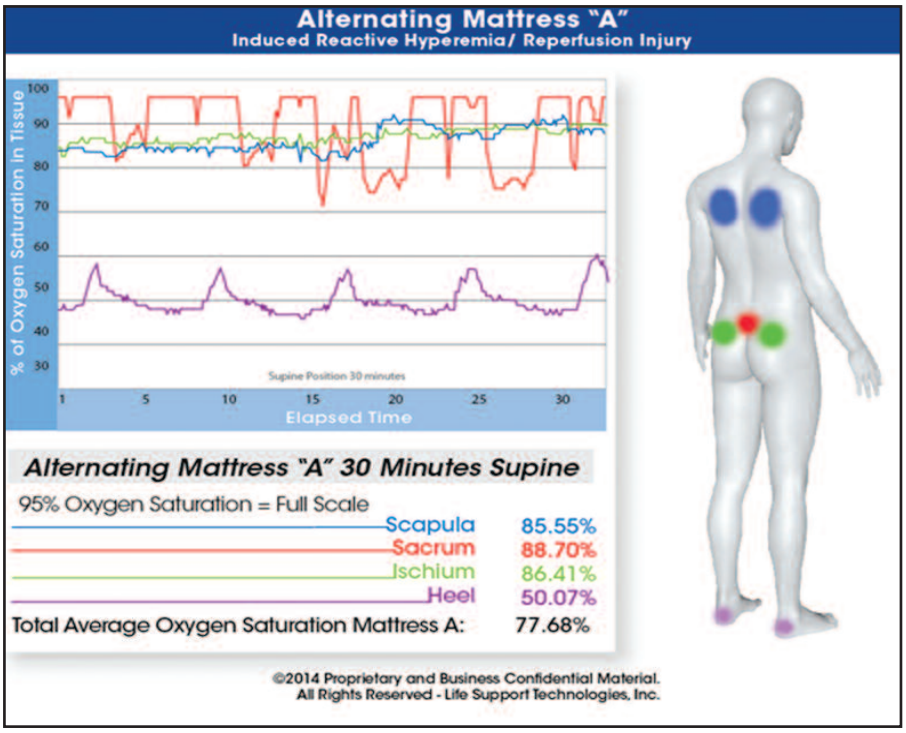


Figure 8. Alternating mattress "A" inducing reactive hyperemia and eventual I/R injury.

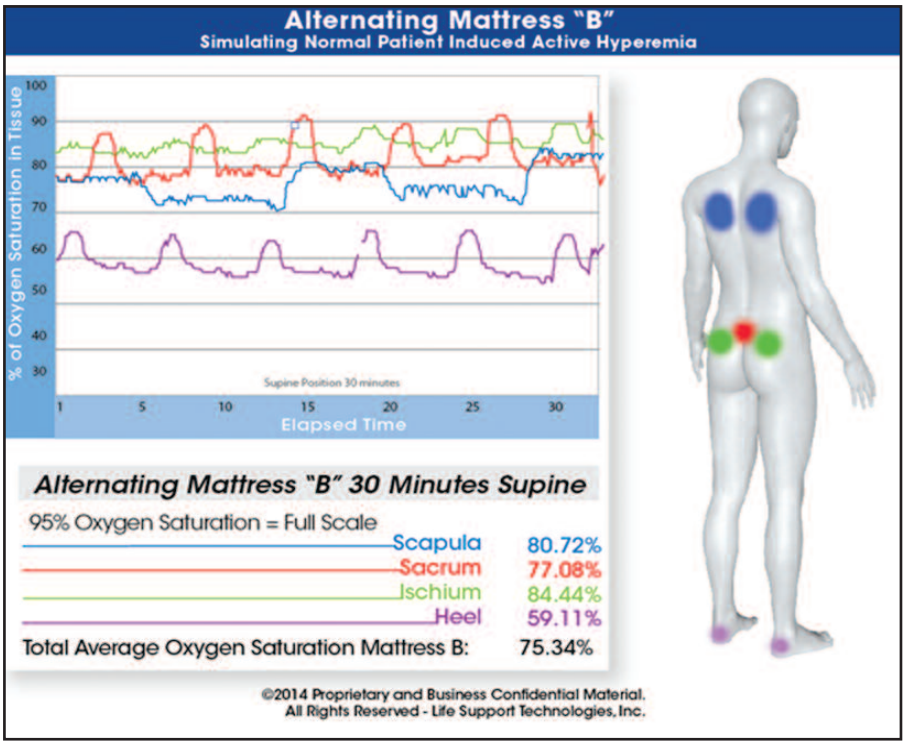


Figure 9. Alternating mattress "B" inducing normal active hyperemia.

activity of an organ or tissue. An example of active hyperemia is the increase in blood flow that accompanies muscle contraction, which is also called exercise or functional hyperemia in skeletal muscle.

During active hyperemia, blood flow increases because the increased oxygen consumption during muscle contraction stimulates the production of vasoactive substances that dilate the resistance vessels in the skeletal muscle. Other examples include the increase in gastrointestinal blood flow during digestion of food, the increase in coronary blood flow when heart rate is increased, and the increase in cerebral blood flow associated with increased neuronal activity in the brain.

Active hyperemia may be due to a combination of transient hypoxia and the generation of cell metabolite vasodilators such as potassium ion, carbon dioxide, adenosine, and nitric oxide.

REACTIVE HYPEREMIA[3]

Reactive hyperemia occurs after the normal physiological mechanisms of active hyperemia are exhausted. It is the next step in the biochemical cascade toward reperfusion injury. The only meaningful variables are patient co-morbidities that down-regulate vasodilation and tissue-oxygen tension-times required to become ischemic.

Reactive hyperemia is the transient increase in organ blood flow that occurs following some period of ischemia (e.g., arterial occlusion). Reactive hyperemia can follow the removal of a tourniquet, unclamping of an artery during surgery, or restoring flow to a coronary artery after reopening a closed artery using an angioplasty balloon or clot-dissolving drug. It also can occur after a prolonged period of tissue pressure that reduces blood flow in prone anatomical locations such as the sacrum, ischium, trochanter, scapula, thoracic spine, and heels.

Figure 3 shows the effects of a two-minute arterial occlusion on blood flow. In this example, blood flow goes to zero during arterial occlusion. When the occlusion is released, blood flow rapidly increases (i.e., hyperemia occurs) and lasts for several minutes. The hyperemia occurs because during the period of occlusion, tissue *hypoxia* and a build-up of vasodilator metabolites (e.g., adenosine, nitric oxide) dilate arterioles and decrease vascular resistance.[3]

When perfusion pressure is restored (i.e., occlusion released), flow becomes elevated because of the reduced vascular resistance. During

the hyperemia, the tissue becomes re-oxygenated and vasodilator metabolites are washed out of the tissue. This causes the resistance vessels to regain their normal vascular tone, thereby returning flow to control levels.

WHEN REPETITIVE HYPEREMIA LEADS TO ISCHEMIA

If the patient's position is changed often enough (classical two-hour nursing turn) after a mild ischemic incident, some focal tissue pressure will be released and there will be some moderate active hyperemia and blood vessel dilation. This increases blood flow and flushes out metabolites/free radicals and then a normal blood flow auto-regulation will resume. This is a normal process.

Excessive and repetitive ischemia, hypoxia, and then excessive reactive hyperemia will lead to a true ischemic/reperfusion injury and neutrophil adherence to the capillary endothelium that results in cell rolling, sludging, and progressive blood flow reduction/occlusion.

This ischemic/reperfusion injured tissue becomes increasingly compromised and susceptible to ulcer development upon repeating this vicious cycle.

For patients with co-morbidities and for those that are more prone to pressure ulcer development, the anatomical areas most susceptible to pressure and shear are the scapula, the sacrum, the ischium, trochanter, and heel. For seated patients, the areas most impacted are the buttocks and the ischium.

The role of shear forces developed in sitting with respect to tissue trauma in the region of the ischial tuberosities may be significant in pressure ulcer causation. Prior study results have shown that cutaneous pulsatile flow measured at the buttocks of the geriatric hospitalized patient and seated paraplegics is considerably reduced compared with that of healthy subjects.

Average shear values developed by the geriatric hospitalized group were three times that of the young, healthy group. It also has been shown that the sitting shear force developed by paraplegics is considerably greater than corresponding measurements of normal subjects.[1-3,12]

REACTIVE HYPEREMIA CLINICALLY DEMONSTRATED

Using our simultaneous Near-Infrared Spectrographic Tissue Oximetry/Interface Pressure system we have noted that about 80% of normal test subjects demonstrated some reactive hyperemia (RH) of the sacrum; in particular, upon standing after being supine on a mattress surface for a 90-minute test period. This is the first time this has been demonstrated.

As an extreme example of this phenomenon, the Life Support Technologies (LST) lab group tested a standard, firstgeneration, three-inch, monolithic memory foam mattress designed for use in monoplace hyperbaric chambers. This testing was initiated because of LST's clinical concerns regarding this mattress' ability to adequately off-load compromised patients receiving hyperbaric oxygen therapy over a two-hour supine period.

The degree of RH was sometimes significant; for example, Subject #4 of 8 subjects tested went from a pretest sacral area oxygen saturation value of 76%, then down to a value of 45% over a 90-minute period while supine and as immobile as possible to simulate a paraplegic/insensate patient. Most subjects experienced ever-increasing to eventual extreme pain in the sacral area during this 90-minute supine/immobile test period.

Upon standing, the subject's sacrum oxygen levels went immediately up to over 95% oxygen saturation (instrument full scale) and remained in that fulminant reactive hyperemic level for a 13-minute period before normalizing (auto-regulation) back to 65–68% (Note: below test baseline). On standing, the subject noted an extreme sensation of heat and interruption of pain.

The authors consider the extent of this reactive hyperemia as a hallmark of "reperfusion injury" and an early indicator of pressure ulcer development.

SOMATIC (WHOLE BODY) ACTIVE HYPEREMIA DEMONSTRATED

Another test program was designed to compare sacral oxygen tensions in subjects while lying on six different mattress systems. Each test subject demonstrated a unique pattern of somatic active hyperemia (SAH) when going from a standing to a supine position, then sitting in a 70-degree position, and back to standing. This pattern was unique to each subject, and the auto-regulation wave pattern results were both unique and constant, regardless of the bed surface. Only the oxygen tension averaged percent shifted based on the mattress type being tested, not the unique "signature" graph shape, regardless of the mattress.

The authors have designated this phenomenon "Active Hyperemia Oxygen Signature" because of its unique and repeatable pattern for each subject, regardless of the surface being tested and even when tests were conducted more than one year apart.

***Example*:** Subject B's sacral oxygen saturation has the same pattern signature on each of six different types/manufacturer mattress surfaces. Figure 6 shows standing time before the 20-minute supine test measurement, then test measurements over a 20minute period while in the 30-degree position, and then, again, when standing.

Note: The top line of the graph on Figure 6 with the highest natural Sacral Oxygen Tension is taken from Subject "B" on the Oxy-Mat™. The lowest oxygen value is taken from Subject "B" on an alternating mattress.

EVALUATION OF MATTRESS SYSTEMS BY INTERFACE PRESSURE MEASUREMENT ALONE

Over the last 50-plus years, dozens of mattress designs have been produced to help better distribute, or periodically reduce, pressure on anatomical areas of the body at higher risk for the development of pressure ulcers. All of the scientific data that has been developed to support mattress manufacturers' off-loading claims have been based on interface

pressure (mmHg) measurements. By itself, this is not a meaningful measurement.

These pressures are then extrapolated over time using an empirical algorithm[9,10] to estimate tissue ischemia in an effort to predict pressure ulcer development. To measure internal tissue pressure changes, invasive or indirect methods are used. The use of pressure-sensing catheters, which is invasive, is unsuitable for use in human volunteers. Indirect methods included magnetic resonance chemical tagging, in which the displacement of tag lines allows one to calculate internal tissue strain and deformation.

LST provides emergency, In- and Out-patient wound care and hyperbaric medicine services around the New York Tri-State area and conducts about 80% of the hyperbaric emergencies in the region. During hyperbaric therapy, such patients must lie on a hyperbaric chamber mattress system that is often not as pressure-relieving as their "special" alternating or low air-loss, hospital-floor provided bed system.

In an effort to provide hyperbaric oxygen therapy to a larger population of sicker Inpatients and post-surgical patients with compromised flaps and graphs and to those requiring "special" off-loading mattress systems, LST had to develop the capability of fully evaluating pressure-relief mattress systems, beginning with hyperbaric mattresses.

TRADITIONAL PRESSURE MAPPING

LST first turned to the industry standard of mattress "pressure mapping" systems available on the market. Several systems are commercially available and this basic technology is used to design and promote commercially available clinical and retail mattress systems on the market today.

TISSUE OXYGEN VALUES

It was soon realized that measuring pressure alone was not a meaningful predictor of tissue ischemia/reperfusion (I/R) injury leading to pressure ulcers. In fact, tissue-oxygen values are the only reliable "real-time" indicator of relative tissue ischemia leading to reperfusion injury and pressure ulcer development. Colin, Loyant, *et al*.[20] measured transcutaneous oxygen tensions on the sacrum of 20 healthy individuals positioned on five different mattress types. That study demonstrated lower oxygen tensions as compared to a control as subjects were exposed first to a standard mattress, then improved foam, and, finally, a water-equalizing mattress.[20]

Subsequent studies (Cullum, Deeks, 2001[17]) were not able to demonstrate significant differences in the prevention of pressure ulcers across categories of support surfaces within each design type. What is still unknown is the amount of pressure/time required to cause pressure ulcer development in an individual patient and especially those elder patients with co-morbidities.[21]

Clearly, pressure is only one of the contributors to the pathophysiology and relative risk of pressure ulcer development. Factors such as blood perfusion, BMI, nutrition, and co-morbidities affecting Nitric Oxide (NO) vasodilatation and the management of reactive oxygen species (ROS) all play an important part in relative pressure ulcer risk and actual clinical pressure ulcer development.

SIMULTANEOUS PRESSURE AND OXYGEN MEASUREMENTS—A COMPLETE PICTURE

Simultaneous measurement of both pressure and deep-tissue oxygen tensions should provide a more complete picture of the effects of pressure and any blood flow reduction resulting in lower oxygen tensions leading to ischemia. To solve this problem, we modified and integrated near infrared spectrographic oximetry used to measure brain oxygen during anesthesia (tissue oxygen saturation) into our existing pressure-mapping system, as a further indicator of blood perfusion and a direct indicator of tissue oxygen tension. The combination of simultaneous pressure and oxygen tension has permitted us to truly evaluate off-loading system designs and to better evaluate patients well beyond the accepted evaluation methods of relative risk of pressure ulcer development alone.

An example of the simultaneous interface pressure/tissue oxygen graph is depicted in Figure 7. In this graph, and in the 2-D map of mattress/body interface pressure, for clarity, we are looking at only ischium pressure/oxygen values. Standard studies included scapula, ischium, sacrum, trochanter, and heels. The subject goes from a standing position to supine for 20-minutes, then elevated to a 70-degree recline for 20-minutes and then, finally, returns to a standing position.

Note that in both standing and supine positions, ischium tissue oxygen averaged 55% while ischium pressure averages 26mmHg in the supine position. In the 70-degree position, the subject's weight transfers to the ischium and the average interface pressure rises to 99mmHg while the ischium oxygen tension only decreases to 51%. The net pressure increase from the supine to sitting is over 280%, but the oxygen only decreases by 6.5% from the supine position. This is an effect of the human body's ability to auto-regulate blood perfusion as measured by oxygen saturation.

This is a typical example of how pressure and blood perfusion are not inversely proportional in healthy subjects able to carry out normal auto-regulation/active hyperemia.

Conversely, it helps us better understand how age and co-morbidities compromise blood perfusion auto-regulation, hyperemia, I/R, and pressure ulcer development.

PRESSURE/OXYGEN RELATIONSHIP

The generally accepted hypothesis is that there is a close correlation between an increase in tissue pressure and a reduction in blood flow, with approximately 30mmHg pressure resulting in capillary vessel collapse. In fact, our testing demonstrated that an increase in pressure at a specific anatomical location and that tissue blood flow as measured by oxygen saturation often did not inversely correlate; a high interface pressure often did not mean a lower tissue-oxygen saturation leading to ischemia and that

a lower tissue interface pressure did not always result in better blood flow and higher oxygen saturation. Our testing also demonstrated that an increased tissue-oxygen phenomena is only demonstrated when subjects were lying on equalized weight re-distribution surfaces that provide for maximum immersion, enveloping all bony prominences in a three-dimensional format (length, width, depth) that conforms to the anthropometric characteristics of the human body.

This oxygen phenomenon is not demonstrated in un-equalized weight re-distribution surfaces (alternating pressure), which accommodate the human body in a two-dimensional format that allows the body to ride on top of the inflated cells without means to envelop bony prominences. This method of periodically delivering high/low pressure over a very small (three-inch) area of the body (the width of some air cells in alternating pressure mattresses) can lead to a reduction in subcutaneous tissue oxygen saturation.

AGE AND CO-MORBIDITIES

Our test data trending seems to demonstrate that young and healthy individuals are able to sustain higher tissue pressures longer and still demonstrate normal hyperemia response and oxygen tensions above 40mmHg. Conversely, older patients with comorbidities that compromise blood auto-regulation appear to lose tissue oxygen tensions faster under moderate pressure (50mmHg) and do not demonstrate a normal hyperemic response and will demonstrate lower pressure tissue oxygen levels than starting tensions. More testing is underway to evaluate this trend.

ARE POWERED MATTRESSES APPROPRIATE FOR ALL PATIENTS?

Our research and the literature indicate that patients who are alert/oriented and who are able to get out of bed to walk or who benefit from formal physical therapy may not be ideal candidates for Group 2 powered systems.[1,6,20]

Full or partially ambulatory, lucid, and sensate patients induce their own off-loading active hyperemia by periodically shifting weight, although nurse monitoring (to help ensure movement) and fall-risk compliance are always required.

In part, due to the 2008 decision by CMS not to pay for nosocomial pressure ulcers, many medical institutions have purchased large quantities of Group 2 powered systems believing that this "one size fits all" concept was the best way to avoid any mismatching of patients to beds. They erroneously thought all powered, off-loading mattress systems provide the greatest reduction in pressure ulcer risk and, therefore, must be better for all patients. In fact, they may not be clinically appropriate for a significant number of patients.

Many alert and ambulating patients report that powered mattress systems move too much, generate noise, and will not allow them to sit on the edge of the bed without the air cells collapsing. Nurses are concerned that rolling patients for posterior dressing changes can induce a similar cell collapse. Further, these air-cell systems are designed for flat-supine use. Patients who sit up in articulated beds often collapse the cell(s) under their sacrum/ischium and "bottom-out" on the steel bedframe; this defeats the bed's design intent. The use of powered surfaces by ambulating and sensate patients may also contribute to increased length-of-stay through sleep deprivation and unconscious muscle tensioning movement. When examining the etiologies of sleep disturbance, studies have focused on environmental stimuli such as 24/7 increased noise and diagnostic testing.[23] However, increased ICU noise level as a sole cause of sleep deprivation has been questioned.[24] One overlooked stimuli is the proprioceptive subconscious counter-effect demonstrated by some sensate patients placed on a regularly moving and audible powered mattress system.[25]

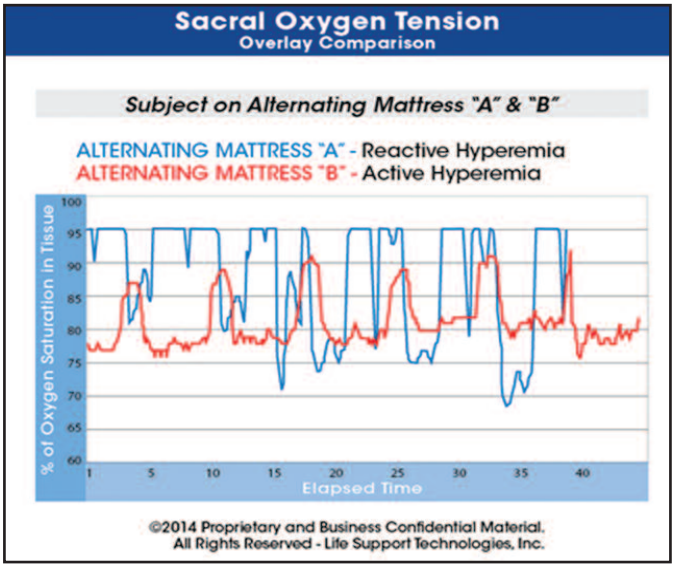


Figure 10. Sacral oxygen tension—overlay comparison graph.

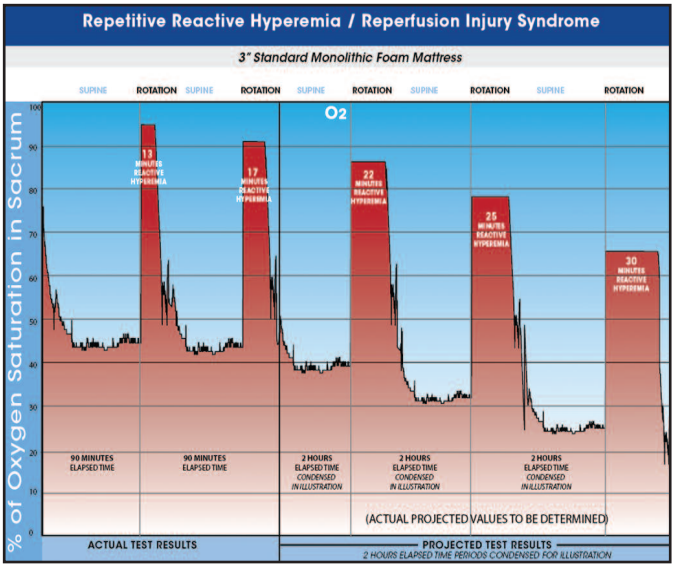


Figure 11. Hypothetical progression of repetitive ischemic/reperfusion injury. Copyright 2015 Life Support Technologies Group.

SOMATOSENSORY SYSTEM INDUCED MUSCLE FATIGUE

Proprioception is a term used to describe the unconscious awareness of the human body position in space as well as the situational position of arms and legs.[25] Somatosensory awareness includes many environmental factors such as movement, vibration, texture, and temperature.

The term "low frequency isometric muscle fatigue" (LFIMF) has been developed by the authors to help describe a non-specific myalgia experienced by some ambulatory and sensate patients after sleeping on powered bed surfaces; in particular, alternating designs. Isometric muscle fatigue is well understood and experienced in high vibration and gravity accelerations by astronauts,[26] high-performance aircraft pilots, and race car drivers when they are required to do hand-eye coordinated physical work while under rapid motion (one-plus gravity) conditions.

This muscle fatigue seems to occur as the subject's autonomic balance center tenses muscles in an attempt to maintain a fixed physical orientation despite special movement, vibration, and G-forces acting on the body. This constant state of isometric muscle tensioning can induce fatigue and generalized muscle pain.

The authors hypothesize that the same proprioceptive isometric muscle fatigue seen in aerospace subjects[26] may also occur to a lesser degree in some patients on powered mattress systems. Even at the very low frequencies and amplitudes of movement experienced while lying on a powered mattress, some ambulatory and sensate patients report movement discomfort. It is thought that this discomfort is due to their somatosensory systems' involuntary attempts to maintain position on a moving surface. LFIMF may be more prominent in sleeping patients. This phenomenon may be linked to insomnia, fatigue, and non-specific myalgia experienced by patients after sleeping on powered systems. LFIMF can manifest as a profound non-specific myalgia that may require the use of pain medications and may result in decreased ambulation and performance in formalized physical therapy.[6]

CAN POWERED MATTRESS SYSTEMS INDUCE AN ISCHEMIC/REPERFUSION INJURY?

During several tests, it was noted that when some powered, alternating mattresses are not adjusted correctly for patient weight, they can become too hard, then too soft. This may induce a significant repetitive reactive hyperemia with 30% plus changes in sacrum, heel, and ischium deep-tissue oxygen tensions as demonstrated by using near-infrared spectroscopy oxygen saturation measurements.

In the graph (Fig. 8), alternating mattress "A" is an example of an alternating mattress induced reactive hyperemia where the sacral tissue oxygen saturation tensions (red-line) alternate every seven minutes between 95-70% (95% = instrument full-scale) when the alternating mattress air cell is decompressed and the sacrum is off-loaded, and when the mattress air cell is compressed.

All clinicians should be aware that alternating mattress systems should be adjusted to simulate normal patient-induced bed off-loading movements (active hyperemia). They should not induce the reactive hyperemia as seen in Figures 5 and 8.

Such extreme alternating changes in oxygen tensions may actually be inducing a repetitive I/R injury syndrome leading to hypoxia and pressure ulcer development.

The graph (Fig. 9) is an example of an alternating mattress, simulating normal patient-induced active hyperemia. The sacral tissue oxygen levels (red line) alternate every seven minutes between an average of 86-73% when the alternating mattress air cell is decompressed and tissues are off-loaded and when the mattress air cell is compressed.

Figure 10 is an overlay comparison of the previous alternating mattress "A" and the alternating mattress "B" graphs (Figs. 8 and 9, respectively). This comparison is intended to clearly demonstrate the differences in sacral oxygen tension fluctuations between an alternating mattress "B" (red line) properly adjusted to simulate normal patient off-loading active hyperemia and the alternating mattress "A" (blue line) that is misadjusted for patient weight.

(It should be noted that such significant changes in oxygen tensions may be inducing a reactive hyperemia that will likely — over time — result in a repetitive ischemic/reperfusion injury syndrome leading to anaerobic cell respiration pathways and pressure ulcer development.)

Figure 11 represents a theoretical progression of I/R over patient turning cycles. The three peaks on the right side of the graph are extrapolated from actual testing depicted in the two graph peaks at the left side of the graph. These two events (the first is Fig. 5) were 90-minutes apart with a 13-minute and then a 17-minute uncontrolled reactive hyperemia of the subject's sacrum.

Based on our test results to date, we believe that this extrapolated data is representative of the actual progression of repetitive ischemia and hypoxia leading to significant I/R injury and neutrophil adherence to the capillary endothelium that results in cell rolling, sludging, and a progressive blood flow reduction/occlusion.

The authors are presently developing a murine animal model in order to further demonstrate and research this phenomenon.

A NEW STANDARD FOR EVALUATION AND DEVELOPMENT OF OFF-LOADING MATTRESSES

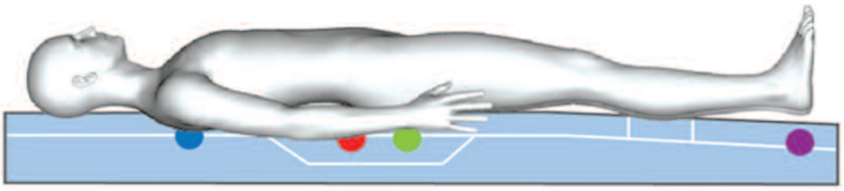
Over the last several years of development, including hundreds of laboratory tests and clinical evaluations, LST has developed a proprietary, non-invasive method to evaluate and design off-loading bed surfaces and to assess a patient's relative risk for pressure ulcer development.

This patent-pending system of oxygen saturation technology and deep-immersion engineering combines established measurements of whole-body interface pressure with near-infrared spectroscopy to simultaneously analyze and correlate both interface pressure and deep-tissue (to 5cm deep) blood-oxygen saturation levels at multiple anatomical locations.

This is the first mattress evaluation system to utilize noninvasive, near-infrared spectroscopy to measure deep-tissue, blood-oxygen saturation

Table I
Average oxygen saturation for a 30 minute supine position.

Oxy-Mat™ vs Other Leading Designs - 30 Minutes Supine



Comparative Oxygen Saturation - 3 Mattresses - 30 minutes Supine				
	Scapula	Sacrum	Ischium	Heel
OXY-MAT™	86.81%	95.00%	94.59%	53.39%
ALTERNATING	85.55%	88.70%	86.41%	50.07%
LOW AIR LOSS	84.98%	95.00%	89.78%	44.79%

levels as the prime risk indicator for compromised blood flow leading to ischemia, hypoxia, necrosis, and pressure ulcers. The LST system provides up to eight simultaneous measurements on a supine/sitting human subject's heels, sacrum, ischium, scapula, and occipital.

The LST system is designed for rapid and convenient determination of oxygen utilization in deep-tissue, measuring arterial-venous oxygen saturation differences. Measurements are made using near-infrared (NIR) spectroscopy at selected pressure-sensitive and weight-bearing anatomical posterior sites while the subject is lying on either a commercial therapeutic support surface or on the Oxy-Mat™.

The optical properties of blood are wavelength-dependent and vary with the oxygenation of the hemoglobin and, to a lesser extent, with other tissue components, such as myoglobin. The data provides a means for demonstrating the comparative effectiveness of off-loading surfaces in maintaining and improving oxygen utilization.

The use of optical spectroscopy generates two or more photon wavelengths (660- and 880nm) using two light-emitting diodes (LED) which are alternately illuminated. At 760nm, hemoglobin occurs primarily in the de-oxygenated state (deoxyhemoglobin), whereas at 850nm it occurs in the oxygenated state (oxyhemoglobin).

By monitoring the difference in absorbency between these two wavelengths, one can monitor the degree of tissue de-oxygenation. This change reflects the balance between the oxygen supply at the level of the arterioles, capillaries, and venules[22] and the amount of oxygen extracted by the tissues.

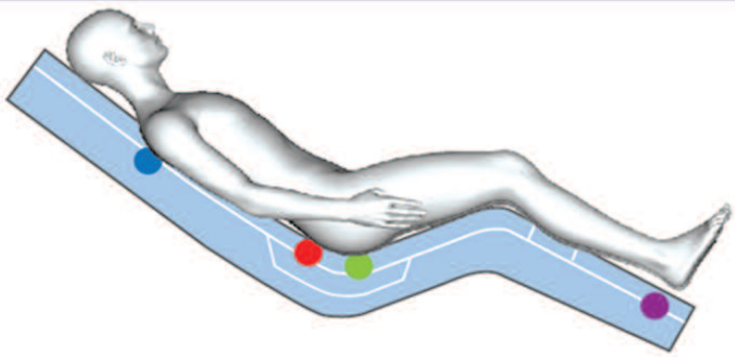
Patients having higher deep-tissue, oxygen-saturation levels while resting on their mattresses are less likely to develop pressure ulcers, and are more likely to heal faster if they already have pressure ulcers. This assumes compliance with the current VHA guidelines (e.g. adequate nutritional intake, regular skin assessments, etc.).

LST has conducted extensive testing on healthy volunteers. The evaluation of existing mattress systems using simultaneous patient/mattress interface pressure and deep-tissue oxygen levels began in 2007 and continues to date. During this period, 20 subjects have been involved in the program — 8 women and 12 men — with ages ranging from 18 to 65 years. Subjects' health ranged from no prior history to significant co-morbidities, including renal failure/dialysis, Type 1 and Type 2 diabetes, COPD, and para- and high-functioning quadriplegic.

Over 200 pressure/oxygen studies

Table II
Average oxygen saturation for a 30 minute, 30 degree incline position.

Oxy-Mat™ vs Other Leading Designs - 30 Minutes 30° Incline



Comparative Oxygen Saturation - 3 Mattresses - 30° Incline				
	Scapula	Sacrum	Ischium	Heel
OXY-MAT™	82.48%	95.00%	94.84%	60.30%
ALTERNATING	87.34%	89.07%	89.50%	53.17%
LOW AIR LOSS	83.97%	95.00%	91.79%	47.61%

have been completed using 16 commercially available alternating, low air loss and non-powered, equalizing mattress systems. The development of the oxygen saturation and deep-immersion engineering technologies and design of the clinical and home-care Oxy-Mat™ mattress systems and other Off-Loading Technologies, Inc. products are based on this continuing research program. Overall, results of the average deep-tissue, oxygen-saturation levels among volunteer test subjects, as measured at the scapula, sacrum, ischium, and heel while resting on low air-loss mattresses, alternating mattresses, and the Oxy-Mat™ are shown in Tables I and II; 95% oxygen is full scale.

SUMMARY OF OXYGEN STUDIES FOR ALL MATTRESS TYPES

The Oxy-Mat™ showed better deep-tissue, oxygen-saturation levels in every category tested (Table I).

LST's proprietary research and product development technology simultaneously measures the patient/mattress interface pressure as well as provides real-time, deep-tissue oxygen tension measurements using near-infrared spectroscopy. Results demonstrate that mattress/patient interface pressure alone is a poor indicator of blood/oxygen perfusion and pressure ulcer prevention. This testing has made it apparent that low tissue oxygen tensions lending to ischemia is the primary and only real-time predictor of pressure ulcer risk.

CONCLUSIONS

From our literature reviews, laboratory testing, Oxy-Mat™ development, and clinical evaluations, we conclude:

1. For over 50 years, healthcare mattress systems have been designed solely on patient/mattress interface pressure. Our testing supports the predominant literature that pressure alone has demonstrated not to be a reliable real-time indicator of mattress design superiority or to measurably reduce pressure ulcer risk or incidence.

2, The development of pressure ulcers and their related morbidity and mortality represents an international healthcare crisis. Any methods or systems that can measurably reduce the incidence of pressure ulcers at lower lifetime patient costs should be pursued.

3.Effective October 2008, the Centers for Medicare/Medicaid Services (CMS) no longer provides DRG In-patient payments for hospital/nursing home acquired pressure ulcers or related infections and patient readmissions within 30-days. This places potentially overwhelming costs directly back to Medicare/Medicaid provider institutions.

4. Medical institutions are being sold an ever-growing array of increasingly complicated and costly powered mattress systems ostensibly developed to further reduce pressure ulcer risks with each new design. New mattress designs are still based on interface pressure mapping and do not produce measurable improvements in patients' deep-tissue, oxygen-saturation levels, or improved patient comfort, or reduced insomnia.

5. The pathophysiology of pressure ulcer development is just beginning to be understood. The true dynamics of repetitive ischemia/reperfusion injury as they relate to deep-tissue oxygen/nutrient supply and cell metabolite management are critical to pressure ulcer prevention, development, and wound care.

6. Simultaneous pressure/oxygen saturation testing indicates that there is no positive correlation between increased tissue pressures as measured using patient/mattress interface pressure and decreased blood perfusion in tissue as measured by blood oxygen saturation oximetry. Therefore, interface pressure is not a real-time indicator of relative pressure ulcer risk, and interface pressure alone should not be used to evaluate mattress off-loading. Age, co-morbidities, and hyperemia auto-regulation are pivotal factors in determining what amount of pressure and duration of time will induce ischemia and potentiate a pressure ulcer.

7. All types of mattress systems must be designed to either permit patients to induce normal active hyperemia by patient movement or to simulate patient movement to induce normal active hyperemia. It is estimated that 90% of hospital and long-term care facility patients are capable of normal active hyperemia and should be placed on non-powered equalizing mattress systems.

8. Reactive hyperemia must be avoided. The time and tissue interface pressures required to induce an ischemic/reperfusion (I/R) event vary significantly from patient-to-patient with age, co-morbidities, and functional circulatory auto-regulation.

9. Repetitive ischemic/hyperemic injury is a term the authors developed to describe the cyclic changes in blood flow in an immobile patient's tissue contact area under repeated pressure and off-loading cycles when a patient is periodically turned. This phenomenon can also be induced by a misadjusted alternating mattress.

10. A period of uncontrolled reactive hyperemia before auto-regulation (Figs. 2 and 3) is the hallmark of an I/R event and a precursor to tissue necrosis and pressure ulcer development.

11. Repetitive I/R injury syndrome data supports the hypothesis that repetitive reactive hyperemia inducing I/R produces ever-increasing neutrophil adherence to capillary endothelium that progressively reduces tissue-perfusion and tissue-oxygen tensions. This time and pressure is very variable and requires additional study.

12. A unique somatic active hyperemia "signature" was observed in each of our test subjects as they were placed in supine, sitting, and standing positions. This could be repeated one year later and the subject identified solely by the shape of their oxygen saturation curves.

13. Powered mattress systems were compared with the Oxy-Mat™ in independent clinical trials. Oxy-Mat™ was credited with improved patient sleep, a reduction in pain medication, and the improved ability to participate in P/T rehabilitation.

14. In extensive testing, the Oxy-Mat™ was shown to be functionally superior to Group 2 powered mattresses by consistently lowering averaged interface pressures and permitting increased natural tissue oxygen values as compared to 16 commercially available powered

alternating and low air-loss mattress systems.

15. During independent clinical trials, the Oxy-Mat™ has been credited with improved patient sleep patterns, some reduction in pain medication, and no skin integrity issues. Rashes and dermatitis also noted improvement to clearing. There were no heel pressure issues or stage III and IV ulcers, as well as no advancing breakdowns. Existing heel and posterior wounds improved to a healed condition. STI

AUTHORS' DISCLOSURES

Mr. Butler is a major stockholder for Off-Loading Technologies, Inc. Mr. Kenyon is a consultant for Off-Loading Technologies, Inc. Mr. Vieweg is the test engineer for Life Support Technologies Group which performed the clinical evaluations on mattresses manufactured by Off-Loading Technologies, Inc. Dr. Gorenstein and Dr. Golembe are Co-Medical Directors for Life Support Technologies Group.

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Tel. +1 415 704 3160
Email: info@surgicaltechnology.com
Internet: www.surgicaltechnology.com