



WORLD LEADER IN ADVANCED REHAB TECHNOLOGY

## Pressure Injury Management – An Update on Etiology, Terms and Definitions



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1. Attend the entire course
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3. Complete an on-line course assessment following completion of the course within 2 weeks

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## Objectives of Presentation

- Define suspected deep tissue pressure injury according to the NPUAP staging guidelines
- Explain three differences in progression of superficial versus deep pressure injuries
- Define the new pressure injury staging guidelines according to the NPUAP staging guidelines
- List the four accepted pathological mechanisms causing a pressure injury
- Describe three intrinsic and the four extrinsic risk factors in developing pressure injuries
- Contrast three differences in the pathology of pressure injuries related to deformation versus ischemia



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### Scope of the Problem in US

- Pressure injuries affect 2.5 million adults per year (Atkins, et. al 2016)
- **60,000** deaths per year from pressure-related complications in the US (Thomas, 2014)
  - Nearly twice as many as caused by MVA
- Patients 65 years and older accounted for 72.3 percent of all acute hospitalizations where PI were noted. (Russo, 2006).

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### Scope of the Problem in US

- 60% of those with SCI develop pressure injury during lifetime
- More than 1 in 10 nursing home residents have a pressure injury
- Varying conditions across all age groups
- \$15 BILLION the government spent on PI treatment in 2003

Russo, April 2006

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### Scope of the Problem in US

- Cost of hospitalization of patient with Stage 4 PI: \$124,000
  - Brem et al, 2010
- Cost of healing a non-complicated Stage 4 PI: \$23,000
- Cost of treatment of Stage 4 with osteomyelitis: \$65,000
  - Dealey, 2012
- Cost of \$11 Billion annually (AHRQ, 2016)
  - Additional impact on CMS reimbursement for the ancillary care of hospital-acquired PI (Van Glider, et al 2017)

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### Scope of the Problem in US

- IP cost per year for SCI Veterans
  - with PI: \$91,341
  - without PI: \$13,754
  - +\$77,587**
- OP cost per year for SCI Veterans
  - with PI: \$19,844
  - without PI: \$11,829
  - +\$8,015**

Stroup et al, 2011

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### Acute Care Significance

- CMS changes
- Hospital Acquired Conditions (HACs)
- Present on Admission (POA)
- “. . .beginning October 1, 2008, Medicare will no longer pay hospitals at a higher rate for the increased costs of care that result when a patient is harmed by one of the listed conditions if it was hospital-acquired.”
- Stage 3 & 4 pressure injuries

Deficit Reduction act of 2005

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### Update of DRA on HACs

#### Deficit Reduction Act on Hospital Acquired Conditions

- Effective October 1, 2015, the ICD-10 Version 33 Hospital Acquired Condition (HAC) list replaced the ICD-9-CM Version 32 HAC list
- Stage 3 and stage 4 still on list (HAC #4)
- 49 specific ICD-10 codes related to stage 3 and stage 4 pressure injuries
- FAQ document:

<https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/HospitalAcqCond/Downloads/FAQ-DRA-HAC-PSI.pdf>

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### Update of DRA on HACs

- The following hospitals are still **exempt** from the POA Indicator and HAC:
  - Critical Access Hospitals (CAHs)
  - Long-term Care Hospitals (LTCHs)
  - Maryland Waiver Hospitals\*
  - Cancer Hospitals
  - Children’s Inpatient Facilities
  - Religious Non-Medical Health Care Institutions
  - Inpatient Psychiatric Hospitals
  - Inpatient Rehabilitation Facilities
  - Veterans Administration/Department of Defense Hospitals



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### The Reality of Pressure Injuries

- A global health care concern
- Extends to every care setting
- Entire healthcare team is responsible
- Responsible for **prevention** and treatment of pressure injuries
- As of April 2016 preferred term is **pressure injury**

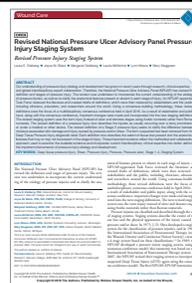


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### Terms and Definitions



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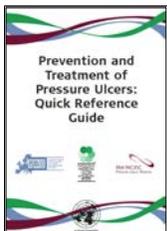
Edsberg, L. E., Black, J. M., Goldberg, M., et al. (2016). Revised National Pressure Ulcer Advisory Panel Pressure Injury Staging System: Revised Pressure Injury Staging System. *Journal of Wound, Ostomy, and Continence Nursing*, 43(6), 585–597.



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### Staging of Pressure Injuries

Revised NPUAP/EPUPAP September 2014



<http://www.internationalguideline.com>



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### Pressure Injuries

#### 2016 Staging Guidelines

All of the following terms and definitions are current as of April 2016

[www.npuap.org](http://www.npuap.org)



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### Pressure Injury (PI) Definition

- Localized damage to the skin and/or underlying soft tissue usually over a bony prominence or related to a medical or other device
  - Can present as intact skin or an open ulcer
  - May be painful
  - Occurs as a result of intense and/or prolonged pressure or pressure in combination with shear
  - Tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue

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NPUAP, 2016



### Stage 1 Pressure Injury

Non-blanchable erythema of intact skin



- Intact skin with a localized area of non-blanchable erythema
- May appear differently in darkly pigmented skin
- Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes

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NPUAP, 2016



### Stage 1 Pressure Injury



- Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure

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NPUAP, 2016



### Stage 1 Pressure Injury

Darker Skin Tones



- Darkly pigmented skin does not have a visible blanching response
- Examine the skin for other changes indicating pressure injury:
  - Discoloration compared to surrounding skin
  - Pain in the area
  - Induration

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Images courtesy of Joyce Black PhD, RN, CWCN, FAAN



### Stage 2 Pressure Injury

Partial thickness skin loss with exposed dermis



- Partial-thickness loss of skin with exposed dermis
- Wound bed is viable, pink or red, moist
- May also present as an intact or ruptured serum-filled blister
- Adipose (fat) and deeper tissues are not visible
- Granulation tissue, slough and eschar are not present

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NPUAP, 2016



### Stage 2 Pressure Injury



- Commonly result from adverse microclimate and shear in the skin over the pelvis and in the heel
- Should not be used to describe moisture associated skin damage (MASD) including incontinence-associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARS), or traumatic wounds (skin tears, burns, abrasions)

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NPUAP, 2016



### Deep Tissue Pressure Injury

Persistent non-blanchable deep red, maroon or purple discoloration

- Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood filled blister
- Pain and temperature change often precede skin color changes



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### Deep Tissue Pressure Injury

- Discoloration may look different in darker skin
- Visibility of necrotic, subcutaneous or granulation tissue, or fascia, muscle or other underlying structures indicates a full thickness PI (Stage 3 or 4 or unstageable)
- Do not use this classification for vascular, traumatic, neuropathic, or dermatologic conditions



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### Deep Tissue Pressure Injury

- Results from intense and/or prolonged pressure and shear forces at the bone-muscle interface
- Wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss

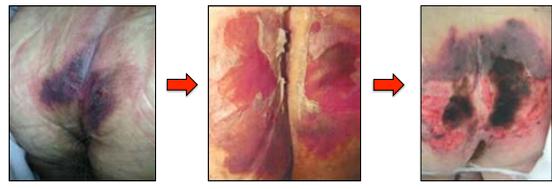


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### Progression of DTPI

How long does it take to go . . .

From this

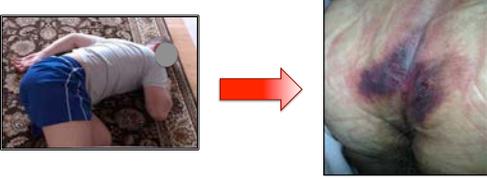


To this

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### Progression of DTPI

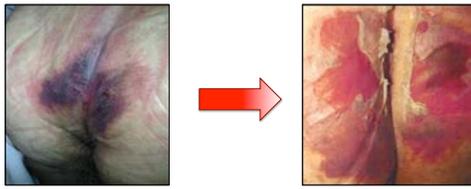


- An event of confinement preceded the first notation of purple skin by 24-72 hours
- ".... most common time frame of 48 hours" - Black, et al., 2015

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### Progression of DTPI



- Within 24-72 hours of the identification of purple skin - thin blisters and/or epidermal sloughing begins
  - Thin blisters, not fluid filled
  - Often misdiagnosed as "Category II" pressure ulcers

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### Progression of DTPI

- Necrotic tissue or full thickness pressure ulcer within 7-10 days after epidermal sloughing

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### Progression of DTPI

Discovery of DTI of buttocks      72 hours later, epidermal lift is present      Development of blood blister at day 10

Black, Brindle & Honaker, 2015

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### Stage 3 Pressure Injury

Full thickness tissue loss

- Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present
- Undermining and tunneling may occur
- Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed

With epibole

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NPUAP, 2016

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### Stage 3 Pressure Injury

- The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds
- Slough and/or eschar may be visible
- If slough or eschar obscures the extent of tissue loss this is an unstageable pressure injury

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NPUAP, 2016

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### Stage 4 Pressure Injury

Full thickness skin and tissue loss

- Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer
- Epibole (rolled edges), undermining and/or tunneling often occur

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NPUAP, 2016

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### Stage 4 Pressure Injury

- Depth varies by anatomical location
- Slough and/or eschar may be visible
- If slough or eschar obscures the extent of tissue loss this is an unstageable pressure injury

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NPUAP, 2016

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### Unstageable Pressure Injury

Obscured full thickness skin and tissue loss



- Full-thickness skin and tissue loss in which extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar
- If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed

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### Unstageable Pressure Injury

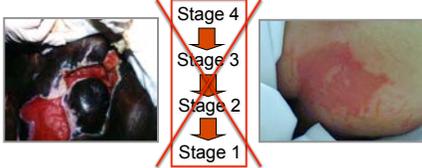


- Stable eschar (i.e. dry, adherent, intact eschar without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed

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### Reverse Staging

- Do not reverse stage a pressure injury
  - Stage 4 pressure ulcer **does not** become a Stage 3, then Stage 2, then Stage 1, then "intact skin" as it heals



- A stage 4 pressure injury that has closed (healed) is classified as "healed stage 4 pressure injury"

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### Reverse Staging

- Reverse staging does not accurately characterize what is occurring physiologically as the wound heals and the depth decreases
  - Wound is filled with granulation (scar) tissue
  - Lost muscle, subcutaneous fat and/or dermis are NOT replaced
  - Risk for subsequent damage is higher in healed wound compared to intact skin with no history of a wound

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### Pathology of Pressure Injuries



What is the actual cause?

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### Evolving Knowledge of Pressure Injuries

"The key to life is accepting challenges. Once someone stops doing this, he's dead." - Bette Davis

"Don't be afraid to change. You may lose something good but you may gain something better." - Unknown

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### Historical Knowledge



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### But, what about these?

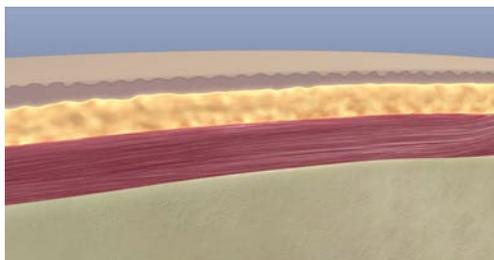


Deep Tissue Pressure Injury

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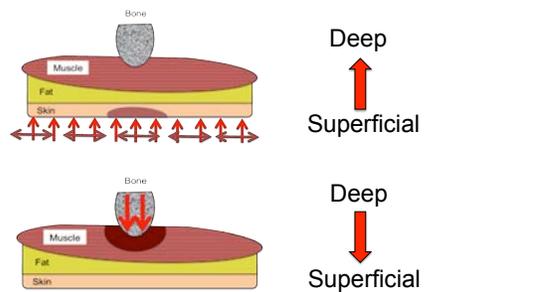
### Evolved Thought Process



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### Two Pathways of Development



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### Extrinsic Risks Factors

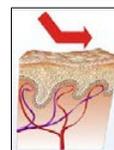
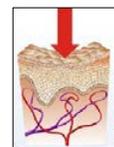


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### Pressure Injury: Extrinsic Risk Factors

- **Pressure**
  - Not changing positions often enough, surface/contact area/immersion, tight or wrinkled clothing, etc.
- **Shear**
  - Less pressure needed to cause skin breakdown when shearing forces are occurring
  - May not be visible on the skin's surface (deep)



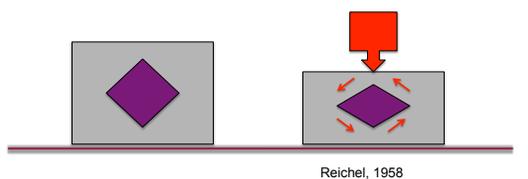
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(Ayello, Baranoski, Lyder, & Cuddigan, 2004)

### Where There is Pressure, There is Shear

- Internal shear
- ALL forces on tissue induce **shear strains (deformation)** within soft tissue



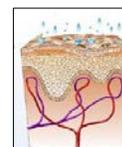
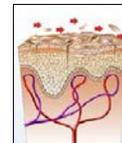
Reichel, 1958

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### Pressure Injury: Extrinsic Risk Factors

- **Friction**
  - Not a primary cause of pressure injuries – but rather surface injuries
  - Can exacerbate existing skin breakdown – contributing factor
  - Can lead to shear stress and deeper issues
- **Moisture**
  - Microclimate (Heat and humidity)
  - Incontinence
  - Wound drainage

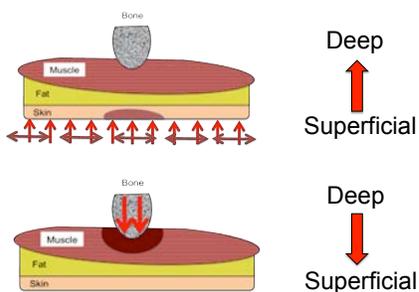


(Ayello, Baranoski, Lyder, & Cuddigan, 2004)

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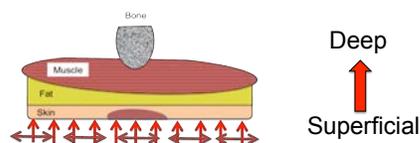
### Two Pathways of Development



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### Superficial Pathway



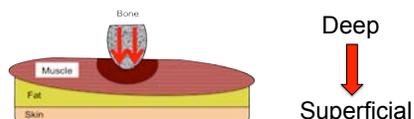
Pressure, **Friction, Moisture, Temperature**

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### Deep Pathway

- **Pressure**
- **Stiffness of surface**
- **Shear**
- **Time**
- **Posture**
- **Intrinsic factors**



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### Intrinsic Risk Factors

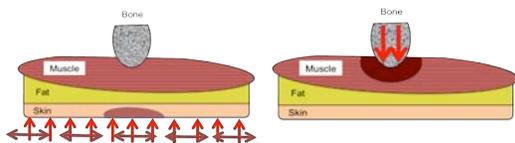
- Age
- Psychosocial
- Malnutrition
- Vascular compromise – **Hypoxia**
- Infection
- Immunocompromise
- Medications – **Steroids**
- Smoking – **Vasoconstriction**
- Diabetes mellitus - **↓ inflammatory response**
- Co-morbidities
- Obesity
- History of pressure ulcers - **Poor tensile strength, ↓ elasticity**



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### Pathway versus Pathology



What is the actual cause?

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### Etiology – Reviewing the Evidence

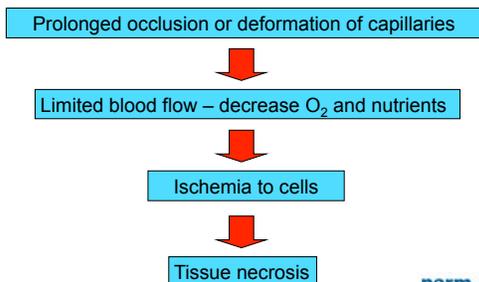
1. Localized ischemia due to extrinsic forces  
(Kosiak, 1961, Dinsdale, 1974, Daniel, 1981)
2. Reperfusion injury  
(Bader 1990, 1988, McCord, 1995, Houwing, 2000)
3. Impaired interstitial fluid flow & lymphatic drainage  
(Reddy, 1981, Miller, 1981, Krouskop, 1983)
4. Sustained cell deformation with increased loads  
(Ryan, 1990, Bouten, 2003, Gefen, 2005, Stekelburg, 2007, Gawlitta 2007, Linder-Ganz, 2007, Gefen, van Nierop, Bader, Oomens, 2012)

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### Localized Ischemia

(Kosiak, 1961, Dinsdale, 1974, Daniel, 1981)



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### How Much Time to Cause Ischemia

≈ 6 Hours

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### Reperfusion Injury

(Bader 1990, 1988, McCord, 1995, Houwing, 2000)

- Concept originally investigated with cardiac patients
- Reperfusion after a period of ischemia results in cellular damage of tissue
- Thought that increased force would prevent blood flow and then when the force is reduced increased return of blood flow may actually disrupt the capillaries
- Also an increase in oxygen free radicals

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### Impaired Interstitial Fluid Flow & Lymphatic Drainage

(Reddy, 1981, Miller, 1981, Krouskop, 1983)

- The increased force may impair interstitial fluid flow & lymphatic drainage
- This impairment of flow may disturb the metabolic waste equilibrium in and around the cells
- This metabolic waste could cause cellular and resulting tissue death

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### Sustained Cell Deformation With Increased Loads

(Ryan, 1990, Bouten, 2003, Gefen, 2005, Stekelenburg, 2007, Gawlitta 2007, Linder-Ganz, 2007, Gefen, van Nierop, Bader, Oomens, 2012)

- High compressive loading not only caused ischemia but also distorted the cells and caused tissue "strain" (damage)
- Stekelenburg in 2007 concluded:
  - "2 hours of compressive loading lead to irreversible damage whereas ischemic loading results in reversible tissue changes"
  - "Large deformation, in conjunction with ischemia, provides the main trigger for irreversible muscle damage."

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### Tissue / Cellular Deformation



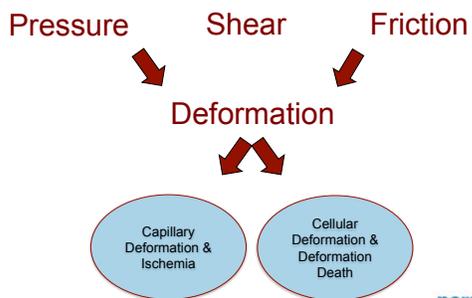
- Low pH and lack of nutrients cause cell death at a longer time scale
- Cell death due to mechanical deformation takes less time than ischemic cell death

Minutes to Hours

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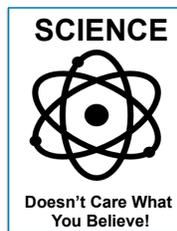
### Tissue Deformation



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### The History of Deformation Theory



How did the research occur to support deformation as a cause of pressure injuries?

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### Meet the Scientists



Dr. Amit Gefen, PhD



Dr. Dan Bader, PhD

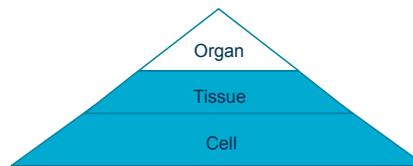


Dr. Cees Oomes, PhD

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### Multi-scale Approach



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### Organ-scale

#### Internal tissue loads during sitting

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Linder-Ganz et al. Journal of Biomechanics 2008

### Multi-scale Approach

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### Production of Tissue-engineered Muscles

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Gefen et al. Journal of Biomechanics 2008

### Production of BAMs from a Cell/Gel Mold

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Gefen et al. Journal of Biomechanics 2008

### The Experimental Setup

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Gefen et al. Journal of Biomechanics 2008

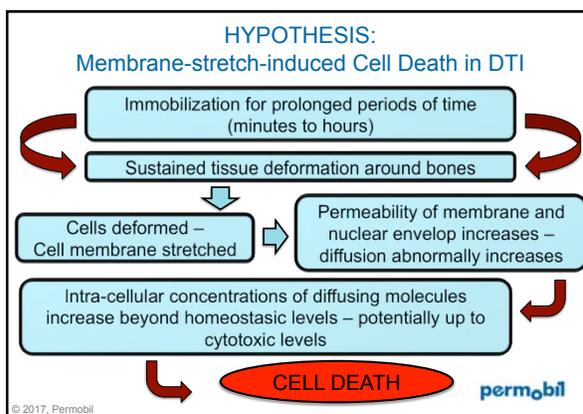
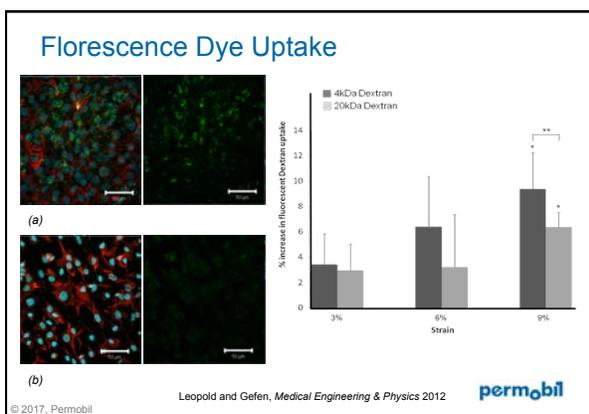
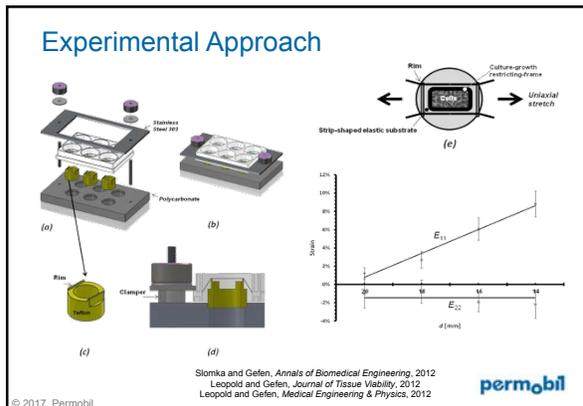
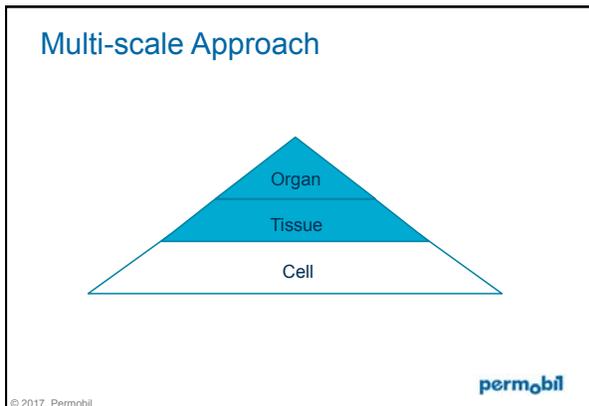
### Measurement of Lowest Deformation Level Causing Cell Death

Propidium iodide was used to fluorescently-stain the development of necrosis in 15 BAMs subjected to strains up to 80%

The lowest strain causing cell-death was determined every 15min, during 285min-long trials, from fluorescence images of the damage region

Time-series of propidium iodide fluorescence images

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Gefen et al. Journal of Biomechanics 2008



### Basic Science Summary

- Decrease in tolerance not necessarily related to ischemia (as traditionally thought)
  - Remember no vasculature in tissue-engineered muscles
- Alternative explanation could be decrease in tolerance related to impaired cellular transport
  - Either at extracellular or intracellular matrix affecting cellular homeostasis

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### Dr. Amit Gefen, PhD

Dr. Amit Gefen, PhD

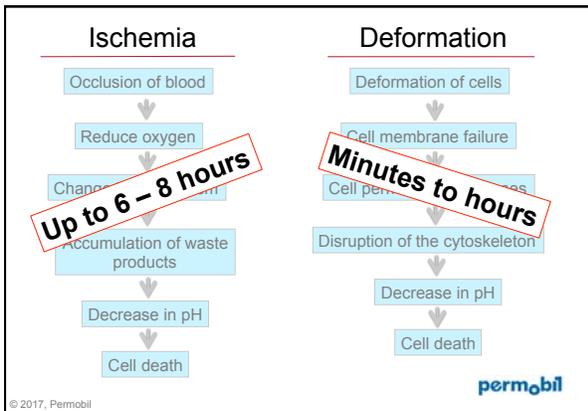
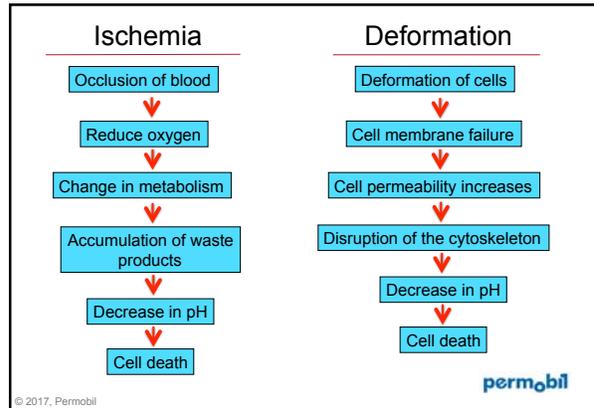
The Musculoskeletal Biomechanics Lab  
Department of Biomedical Engineering  
Faculty of Engineering  
Tel Aviv University

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### Understanding the Research Clinical Decision Making in Pressure Ulcer Management

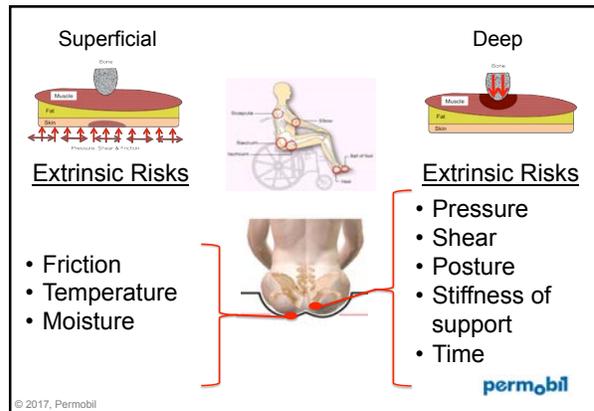


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Up to 6 – 8 hours

Minutes to hours



### Clinical Significance

- Internal damage occurs with deformation
- May not present as a DTI, but damage may be present
- Deformation occurs around bone not only directly over top bone
- If damage is present scarring can continue to occur without outward signs of wound
- With multiple episodes – internal scarring – Eventually less force may cause open wound

INCREASED RISK

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### Consideration in Seating and Positioning

- Consideration of material science and fabrication to manage load: pressure – shear – friction
- Seating system sooner and better than in most cases
- Cushions outside the typical rehab population of diagnoses
- Consider alternative sitting surfaces outside of wheelchair – dialysis, car, toilet etc.
- Utilizing better skin protection cushions and gain stability and positioning through other aspects of mobility device
- Utilization of power seat function and other technology in seating in combination with the cushion to achieve better soft tissue outcomes
- Adaptability and adjustability in seating surfaces

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### Take Home Message

- Two accepted clinical pathways of development
- Pathology of pressure injury is multifactorial
  - No longer just about ischemia
- Internal deformation causes damage – once presents as DTI it is too late
- Management of extrinsic risk is paramount
  - Earlier and better management of deformation
  - Not just about the skin, but muscle and fat too



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### Assessing Your Knowledge

- What is the stage of this pressure injury?



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### Assessing Your Knowledge

- List the extrinsic risk factors in pressure injury development
  - Pressure
  - Shear
  - Friction
  - Moisture
    - Microclimate
    - Incontinence
    - Wound drainage



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### Assessing Your Knowledge

- What are the four accepted pathological mechanisms causing a pressure injury
  - Ischemia
  - Deformation
  - Impaired interstitial fluid flow & lymphatic drainage
  - Reperfusion injury



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### Assessing Your Knowledge

- Differences between ischemia versus deformation
  - Ischemia hours – deformation minutes
  - Deformation damage in presence of blood flow
  - Deformation greater attributed to DTPI
  - High loading greater risk for deformation damage



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### Continuing Education Credit Information



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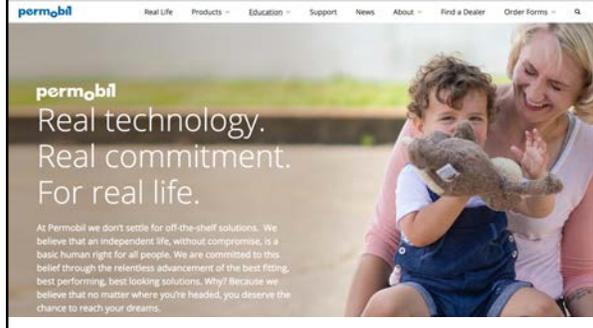
### Online Assessment Info

- **MUST** sign the sign-in sheet
  - Without sign in/out, NO CEUs can be issued
- Online assessments **ONLY** active for **2 weeks!**
  - If not complete within 2 weeks, NO CEUs will be issued
- Access via <http://permobilus.com>
- **NEED** a case sensitive password to access
- **Be ready to PRINT out certificate at the end of the on-line assessment!**

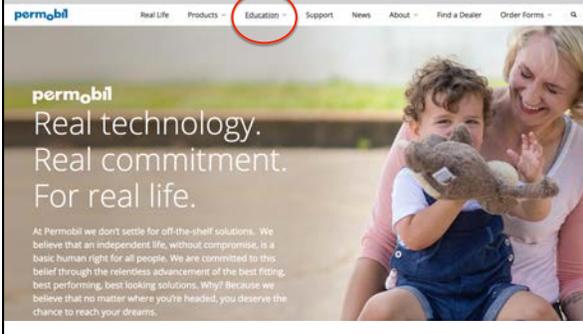


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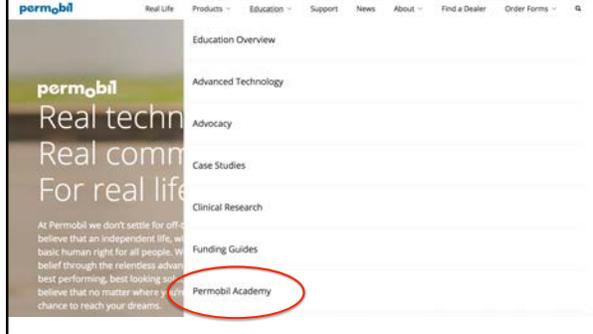
### <http://permobilus.com>



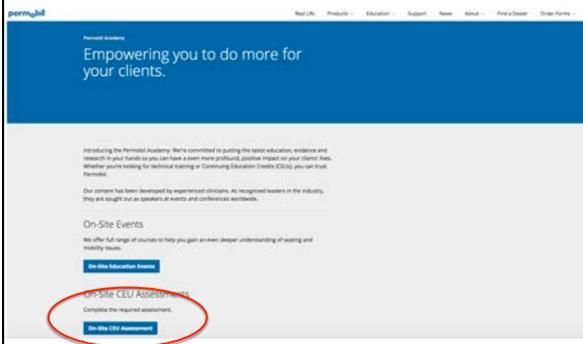
### Click on Education



### Choose Permobil Academy



### Click CEU Assessments



### Click on Take Assessments

After attending an on-site program, complete the corresponding assessment survey to obtain CEU credits.

ASSESSMENTS

Date/Time	Course Topic	Product	Presenter	Location	
01/18/2017	PAES Power Seat Functions		Katherine Petrosi	Daphne Beach, FL	Take Assessment
02/01/2017	Pressure Ulcer Etiology		Katherine Petrosi	Orlando, FL	Take Assessment
02/02/2017	Moving Forward Together		Singer Walls	Richmond, VA	Take Assessment
02/07/2017	PAES Power Seat Functions		Tricia Garmon	Mulliken, TX	Take Assessment
02/09/2017	Fitting the Wheelchair Like a Prosthesis		Singer Walls	Baltimore, MD	Take Assessment
02/17/2017	Power Seat Functions - Good, Better, Best		Janeth Bernheim	Saint Paul, MN	Take Assessment
02/19/2017	Power Seat Functions - Good, Better, Best		Singer Walls	Atlanta, GA	Take Assessment
02/17/2017	Moving Forward Together		Singer Walls	Atlanta, GA	Take Assessment

- The password is case sensitive
  - Password:

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