AAWC Pressure Ulcer Summit (2018): A Recap

Gregory A Bohn, MD MAPWCA, ABPM/UHMS

Department of Surgery
Central Michigan School of Medicine
Tawas, Michigan
Activities:

• President American Board of Wound Healing
• Past President: Association for the Advancement of Wound Care (AAWC)

Disclosures

Medical/Scientific Boards:
• Medline
• Convatec
• Acelity
• ULURU

Consultant:
• ULURU
Objectives

1. Describe the purpose of the AAWC 2018 PrU Summit.
2. Interpret the clinical significance of a conceptual framework for the mechanism of pressure induced tissue damage.
3. Identify opportunities to improve detection and description of pressure induced tissue damage, the process of PI/PU monitoring and risk assessment.
4. Discuss opportunities for collaboration among interprofessional organizations in the pursuit of reducing and eliminating pressure induced tissue damage.
Proceedings of the Association for the Advancement of Wound Care’s First Annual Pressure Ulcer Summit

• OWM, 64:4 (April 2018)

AAWC PrU Summit: Contributing Factors for Developing

1. Fall 2015- May 2016:
   • Change in language and definitions with *minimal* input/response by clinical and academic interprofessional experts.

2. Existing system for quantifying PrU severity continues to be flawed

3. Who speaks for pressure ulcer prevention and treatment?
   • Many clinical and academic professionals with expertise in wound management

4. Patients still get pressure ulcers!
AAWC Response

• **Mission:** To advance the care of people with and at risk for wounds

• **Literature Search** to explore still unaddressed commonly cited problems in the PrU research and clinical practice literature.

• **Strategies:**
  • 1. Assist in accurate assessment of PrU’s distinct from other common etiologies of skin damage AND to standardize a reliable method of determining PrU severity.
  • 2. Create a forum for experts representing the diverse professional organizations dedicated to wound care to explore the most recent science regarding PrU formation.
Gaps Identified in PrU Prevention

1. Staging system:
   • Suggests progression of tissue damage from outside in
   • Based on visual assessment
     • Inter-rater reliability: 42% - 52% at Consensus Conference
     • 2012: Review of Literature (1488 patients extracted data)
       1. “Reliability limited and highly variable.”
       2. “Difficult for nurses to distinguish pressure from other types of wound.”
• Several studies estimate substantial rates of inaccurate staging, including DTI and unstageable PrU, with rates ranging from 48% to 79.8%.

• A prospective study has associated inaccurate staging with approximately $1.7 million in revenue loss.
3. Collaboration among Clinical and Research Experts
   • Based on current research: bench and bedside
   • Based on current experience of expert clinicians
   • Do we know what each is doing?
Bring together wound care leaders and stakeholders to:

1. Review the latest research.
2. Explore challenges and innovations in clinical care.
3. Identify opportunities in advancing the science of pressure ulcer prevention and management.
4. Seek to identify pathophysiological models that clinicians can understand and use to make bedside decisions.
5. Compare and contrast recent science with our current practice patterns to identify opportunities for improvement.
Validity Testing of the Pressure Ulcer Description (PUDT) Tool

Task Force

Gregory Bohn, MD MAPWCA
President AAWC, CMU School of Medicine

Ruth A. Bryant PhD, RN, CWOCN
Director Nursing Research, Abbott Northwestern Hospital, Minneapolis, MN

Tim Paine , PT
Treasurer AAWC

Barbara Bates Jensen, PhD, RN
Professor of Nursing UCLA, Secretary AAWC

Kara Couch MS, CRNP, CWS
George Washington University Hospital Medical Faculty

Richard Simmon, MD

Joy Schank, RN, MSN, ANP,CWOCN

Eric Lulove DPM

Karen Bauer, RN
University of Toledo

Donna Cartwright, MPA, MPA, RHIA, CCS, RAC, FAHIMA.
RHIA, CCS, RAC, FAHIMA.
Purpose of PUDT

- Guide the bedside clinician through the assessments necessary to determine the most likely etiology of the current skin condition in the peri-rectal and fleshy buttocks area.
- Quickly and simply determine the type of skin damage present (i.e., pressure ulcer, incontinence associated dermatitis, virus, etc.).
- NOT a severity scale
Structure of the PUDT

• Three (3) Domains:

1. Skin damage without open ulcer
   - White
   - Red
   - Purple/blue/Gray
   - Deeper hue of individual’s usual skin tone
   - Change in individual skin characteristics (edema, induration, etc.)

2. Blisters
   - Serum
   - Blood

3. Skin damage with open ulcer
   - Superficial open ulcer
   - Deep open ulcer
   - Necrotic Ulcer
<table>
<thead>
<tr>
<th>Skin damage without open ulcer</th>
<th>Clinical Description</th>
<th>Pressure Ulcer Detection Tool (PUDT)</th>
<th>ICD-10 code</th>
<th>Crosswalk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Skin discoloration, no open ulcer</strong></td>
<td>White – Skin may appear white from excess moisture, i.e. maceration or dermatological conditions like fungal infections</td>
<td>1 Maceration 2 Fungal Infection</td>
<td>1 Friction Shear 2 Maceration 3 Incontinence</td>
<td>1 Early Pressure Ischemia</td>
</tr>
<tr>
<td><strong>Skin discoloration, no open ulcer</strong></td>
<td>Red – Inflammation of the skin from external mechanical force, unresolved exposure of moisture, or infection (bacterial, fungal, viral)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Skin discoloration, no open ulcer</strong></td>
<td>Purple, blue, grey – Represents deep tissue damage or ischemia</td>
<td>1 Early Pressure Ischemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Skin discoloration, no open ulcer</strong></td>
<td>Deeper hue of individual's usual skin tone – Represents damage in darkly pigmented skin</td>
<td>Discoloration in darker pigment skin</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Skin discoloration, no open ulcer</strong></td>
<td>Change in individual skin characteristics – Skin change such as temp, edema, Induration, bogginess and pain could represent deep tissue necrosis without skin color change</td>
<td>1 Early pressure damage without skin change</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Blisters</strong></td>
<td>Serum Blister – Blistering of skin results from friction, shear, adhesive damage and does not represent pressure damage. Serum filled, no blood</td>
<td>1 Friction Shear</td>
<td>1 Friction Shear</td>
<td></td>
</tr>
<tr>
<td><strong>Blisters</strong></td>
<td>Blood Blister – Blister may have blood due to deeper dermal injury or due to anticoagulant use</td>
<td>1 Friction Shear with deeper dermal injury</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Definition of Open Ulcers**

**Superficial open ulcer**

- Anatomical loss of epidermis/dermis WITHOUT exposure of deeper structures like subcut, muscle, bone, vital structures

**Deep open ulcer**

- Anatomical loss of epidermis/dermis WITH exposure of deeper structures like subcut, muscle, bone, vital structures
## Relevance Ratings by Expert Panel N = 41

<table>
<thead>
<tr>
<th>PUDT Item</th>
<th>1: not clinically relevant, delete</th>
<th>2: item confusing unable to assess relevance without further information</th>
<th>3: clinically relevant, needs minor improvements on wording</th>
<th>4: clinically relevant as written</th>
<th>Combined rating of 3-4</th>
<th>Item Revision?</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>1 (2.4)</td>
<td>6 (14.6)</td>
<td>11 (26.8)</td>
<td>22 (53.7)</td>
<td>33 (80.5%)</td>
<td>Yes</td>
</tr>
<tr>
<td>Red</td>
<td>1 (2.4)</td>
<td>4 (9.8)</td>
<td>15 (36.6)</td>
<td>20 (48.8)</td>
<td>35 (85.4%)</td>
<td>Minor edits</td>
</tr>
<tr>
<td>Purple</td>
<td>-</td>
<td>3 (7.3)</td>
<td>13 (31.7)</td>
<td>24 (58.5)</td>
<td>37 (90.2%)</td>
<td>Minor edits</td>
</tr>
<tr>
<td>Deeper hue</td>
<td>-</td>
<td>8 (19.5)</td>
<td>14 (34.1)</td>
<td>18 (43.9)</td>
<td>32 (78%)</td>
<td>Yes</td>
</tr>
<tr>
<td>Change in individual skin characteristics</td>
<td>-</td>
<td>4 (9.8)</td>
<td>10 (24.4)</td>
<td>26 (63.4)</td>
<td>36 (87.8%)</td>
<td>Minor edits</td>
</tr>
<tr>
<td>Blister serum</td>
<td>-</td>
<td>4 (9.8)</td>
<td>11 (26.8)</td>
<td>25 (61)</td>
<td>36 (87.8%)</td>
<td>Minor edits</td>
</tr>
<tr>
<td>Blister blood</td>
<td>-</td>
<td>4(9.8)</td>
<td>8 (19.5)</td>
<td>28 (68.3)</td>
<td>36 (87.8%)</td>
<td>Minor edits</td>
</tr>
<tr>
<td>Superficial open</td>
<td>-</td>
<td>2 (4.9)</td>
<td>10 (24.4)</td>
<td>28 (68.3)</td>
<td>38 (92.7%)</td>
<td>Minor edits</td>
</tr>
<tr>
<td>Deep open</td>
<td>-</td>
<td>-</td>
<td>7 (17.1)</td>
<td>33 (80.5)</td>
<td>40 (97.6%)</td>
<td>Minor edits</td>
</tr>
<tr>
<td>Necrotic ulcer</td>
<td>-</td>
<td>3 (7.3)</td>
<td>8 (19.5)</td>
<td>29 (70.7)</td>
<td>37 (90.2%)</td>
<td>Major edits</td>
</tr>
</tbody>
</table>
PUDT: Next Steps

1. Validity Testing
   ✓ First pass: 85% of respondents minor or no changes
   ✓ Second pass: 90% no changes
   ✓ Wording finalized

2. Reliability Testing
   • Finalize best pictures to use
   • Design protocol to use tool in variety clinical settings with variety different clinicians

3. Present to CMS
1. Validity of present staging system, pathophysiological models, and assessment tools.

2. Longstanding inconsistencies exist in PrU risk assessment, staging definitions, classifications for adverse event reporting, and definitions for quality metrics across health care settings.

3. These contradictions place a daily burden on clinical resources in health care organizations and have not significantly advanced the mission of improved patient safety.

4. Unclear how recent changes in pressure ulcer staging will cascade to provide clarity or what alternative approaches can improve this gap.
Mechanisms of Pressure Induced Tissue Damage: Historical Approach

- Ruth Bryant PhD, RN, CWOCN
  - Histopathological process?
  - Cone shaped pressure gradients at the bone/soft tissue interface?
  - Is the epidermal or shallow dermal ulcer a different co-occurring condition?
  - Model needed to convey the series of events associated with pressure induced tissue damage:
    - Vessel occlusion
    - Thrombosis
    - Tissue hypoxia/ischemia
    - Metabolic accumulation of wastes
    - Interstitial fluid accumulation
    - Reperfusion injury
Mechanical loading and tissue compression

Below the skin surface

Visible tissue changes e.g., rubor

Epidermal tissue rupture e.g., Stage II-IV PU

Tactile tissue changes e.g., calor, tumor

Subepidermal tissue damage

Interstitial fluid accumulation

Hypoxia

Vascular permeability

Apoptosis/necrosis

Inflammatory Processes

Oxidative Stress

Toxic metabolites

Nutrient depletion

Ischemia

Lymphatic dysfunction

Deformation

Mechanical loading and tissue compression

At the skin

Window of Prevention

Manifestation Threshold

Nutrient depletion

Inflammatory Processes

Oxidative Stress

Reperfusion injury

Ischemia

Lymphatic dysfunction

Deformation

Damage Threshold

Toxic metabolites

Lymphatic dysfunction

Nutrient depletion

Ischemia

Reperfusion injury

Oxidative Stress

Inflammatory Processes

Mechanical loading and tissue compression

Below the skin surface

Hypoxia

Vascular permeability

Apoptosis/necrosis

Subepidermal tissue damage

Interstitial fluid accumulation

Hypoxia
Focusing PU Prevention Research on Informing Clinical Interventions

• Stephen Sprigle, PhD, PT
  • Rehabilitation Engineering and Applied Research Lab
  • Georgia Institute Of Technology

1. Individualized PU risk can be defined as a person’s Biomechanical risk
   • The characteristic of an individual’s soft tissue to deform in response to external forces
   • Hypothesis: A person with greater biomechanical risk is more vulnerable to PrUs

2. Advocate for prevention research: The imbalance between prevention and treatment is embarrassing

3. PrUs, by definition, are caused by external forces. Thus research should seek an understanding of external forces as a means to ameliorate their effects within clinical interventions.

4. Focus on individual difference of external force effects

5. Develop and deploy sensors.

6. Providers and payers need to take the lead to work together to extend the state of the science.
Sustained deformation lethal to tissues

The skeleton of the cell (cytoskeleton) breaks down

Compression
Plasma membrane
Actin

Stretching

Deformation is a cell killer!

Cell death by necrosis
Cell death by apoptosis
Cell Survives

Weihs and Gefen, *Medical Engineering & Physics* 2016
Direct deformation damage onsets faster than damage due to an ischemic insult.

**Ischemia**
- Impaired perfusion
- Decrease in pH
- Accumulation of waste products
- Cell death
- Loss of homeostasis

**Deformation**
- Deformation of cells
- Cell membrane failure
- Cell permeability increases
- Cell death
- Deformation is a cell killer!

**Up to 6 – 8 hours**

**Minutes to hours**

Weihs and Gefen, *Medical Engineering & Physics* 2016
Muscle quality, specifically fat infiltration, impacts local tissue quality and resilience.

Muscle composition impacted skin blood flow component with significant correlations with gluteal intramuscular fat.
Focal disruptions along basement membrane in elderly skin together with change in orientation of collagen fibers may identify initial changes that lead to development of pressure ulcers in elderly population.

Inflammasome is multiprotein complex

- Expressed in keratinocytes
- Component of Innate immune system
- Responsible for activation of inflammatory processes \(\rightarrow\) IL1
- IL1 suppressed in aged and loaded skin

Inflammasome components are suppressed in elderly indicating greater risk for PrUs
Quality Metrics

- Matt Scanlon MD, CPPS
  - Professor of Pediatrics, Critical Care
  - Medical College of Wisconsin

- Indicators (gas tank or oil level) VS Measures (thermometer)
- Accuracy of coding: 97 per Nursing Documentation → 6 MS documented → 10 with ICD 9 codes
- Rate of coding Stage 2 or higher:
  - Administrative data: 0.15%
  - Surveillance data: 2%
  - Meddings et al 2013 Annals of Internal Medicine

- This presentation provided information on tools and metrics used through ICD-9 and ICD-10 coding, EHRs, National Nursing Quality Indicator surveys, Collaborative Alliance for Nursing Outcomes surveys, and local incident reports
- Although these quality metrics may be useful, they lack validity, and more data analysis is needed to define which factors are truly related to nursing quality
- The comparative analysis of quality metrics should be used to drive future policy in the identification and prevention of PrU in all settings

ICD 9 and ICD 10 codes are not intended to substitute for Quality Measures
Typical Healthcare Safety Approach

Identify Harm (or errors) ➔ Retrospective Investigation ➔ Report # Bad Things

# of bad things
# of patient days
Typical Non-Healthcare Safety Approach

- Identify Hazards ➔ Eliminate Hazards/Mitigate Harm ➔ Report # Successes

# of eliminated hazards ➔ # of identified hazards
REMINDER: Definitions of Deep Tissue Injury (DTI)

According to NPUAP (abv.):

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**.

According to CMS (abv.):

Localized area of discolored intact skin. Area of discoloration may be preceded by tissue that is painful, firm, mushy, boggy, or...

**...warmer or cooler as compared to adjacent tissue.**

**KEY LANGUAGE:**

*a sign/symptom that precedes visual recognition*
Early Detection: DTI

Richard Simman, MD, FACS, FACCWS

Technology: *Long-Wave Infrared Thermography*

**Easy to Use**
- Handheld, lightweight and ergonomic
- Non-radiating and non-contacting
- Can scan multiple areas/wounds in ~5 minutes

**Observe & Evaluate the Invisible**
- Reveals *patho* physiologic markers invisible to naked eye
- Objectively visualize & quantitatively measure circulation, perfusion, and metabolic activity

**The Benefits**
- Assess signs/symptoms of DTI and infection
- Assess wound healing/response to treatment
- Assess amputation levels
- Others

Non-visible signs/symptoms of DTI *(ischemic response)*

Non-visible signs/symptoms of DTI *(inflammatory response)*
Early Detection:

• Recall the Paradigm of Pressure Induced Tissue Damage

• How Can We Detect the Damage occurring before we can see it at the skin level?
Localized edema onsets at sites of cell breakdown and then evolves

The cascade from microscopic to macroscopic edema

- Capillary widening
- Increased permeability
- Attraction of leukocytes
- Systemic response
- Increased blood flow
- Fluid release into tissues
- Extravasation of leukocytes to site of injury
- Fever and proliferation of leukocytes

Sub-epidermal moisture (SEM), which cannot be detected through visual skin assessment, is the early sign of macroscopic edema

Pending FDA decision, not for sale in the US
THE WORD “INJURY”

Injury is not just a medical term. It is also a legal term used in and defined by Jury Instructions

The harmful connotation is compounded when used in conjunction with the term “Never Event”

May imply “harm” and/or abuse
- Many cases involving pressure wounds also include claims of elder abuse and neglect

Not the same as Traumatic Brain Injury or Spinal Cord Injury
- These injuries rarely, if ever, occur while in the care of a healthcare provider while pressure wounds routinely do
Summary Key Points

1. Individual and Unique Risk Factors need to be considered and studied
2. Tissue and cellular deformation matter – system physiology cannot be ignored
3. Human physiology is based on a hierarchy of micro- and macroscopic systems that are all acting at different time scales. Thus, one cannot extrapolate phenomena happening in a patient from what is observed in cellular systems.
4. It is likely that existing definitions and categorizations for PrU do not account for what is happening below the skin. Simply observing the skin might not be enough. Tools are in development.
5. More collaborative work and discussions are needed!
Certification Path to Fellowship

Become Wound Care Certified
Thank you!

- Greg Bohn, MD MAPWCA
- gregbohn2@aol.com
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