Early Intervention for Deep Tissue Injuries

MIST® Ultrasound Healing Therapy

Pre-MIST®

Deep Tissue Injury

Post 5 MIST® Treatments

MIST Ultrasound Healing Therapy
What is a Deep Tissue Injury (DTI)?

**NPUAP DEFINITION:**

The National Pressure Ulcer Advisory Panel (NPUAP) defines a suspected Deep Tissue Injury as a, “Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue.”

- DTIs are a form of pressure ulcer that can form over any area of pressure, but are most commonly seen on the sacrum and heels.

- The tissue injury occurs from the inside out. Changes on the surface are not seen until later, when the tissue undergoes necrosis.

- DTIs require rapid identification, as they may quickly progress to Unstageable, Stage III and IV pressure ulcers despite aggressive and optimal treatment.

- It is difficult to confirm an injury is a deep tissue injury until it runs its full course. The word “suspected” may be used in front of DTI until this confirmation takes place.

Who is at risk for a Deep Tissue Injury?

**High Risk Populations**

DTIs usually occur in the most compromised patients, but can be found in almost any care setting where a patient is allowed to remain in one physical position for extended periods of time.

Patients with a cognitive deficit that impairs their ability to sense pressure (stroke, anesthesia, coma, spinal cord injury, etc.) are at the greatest risk.

**Things to look for:**

- Confinement for more than 3 hours in the OR or cath lab
- History of being “down at the scene” prior to admission
- Unable to turn; leg immobile (due to total hip or total knee), fractured hip, spinal cord injury
- Leg numbness from stroke or neuropathy
- Use of medical devices: O₂ mask, CPAP, braces etc.
- Complex medical conditions: acute cardiac event, pneumonia, obesity, etc.
How do Deep Tissue Injuries Differ from other Pressure Ulcers?

**SUPERFICIAL PRESSURE ULCERS**
Low Pressure – Extended Time

- **Extrinsic Factors**
  - Moisture (urine & stool)
  - Heat
  - Friction
  - Shear

- **Intrinsic Factors**
  - Impaired motor-sensory
  - Poor nutrition
  - Infection

- **Pressure**
  - Lower pressure (<200mm Hg) over long periods of time (10 hours)

**Damage is created from OUTSIDE > IN**

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**DEEP TISSUE INJURIES**
High Pressure – Short Time

- **Extrinsic Factors**
  - Posture/Positioning
  - Time on hard surface
  - Stiffness/Firmness of the support surface

- **Intrinsic Factors**
  - Impaired motor-sensory
  - Muscle atrophy

- **Pressure**
  - High pressure (>300mm Hg) over short periods of time (3 hours)

**Damage is created from INSIDE > OUT**

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**ISCHEMIA**

Ischemia is caused from a prolonged mechanical load that reduces local perfusion by collapsing capillaries within the skeletal muscle. This mechanical load also alters the interstitial environment, causing fluids and nutrients needed for cellular function and metabolism to shift to adjacent lower pressure areas in the interstitial space.\(^{11,12}\)

Ongoing ischemia causes a shift to anaerobic metabolism, increasing the production of lactic acid and other metabolic wastes that lead to cellular and interstitial acidosis and cellular death.\(^{12,13}\) While considered the primary cause of cell death associated with deep tissue injuries, the short time required to achieve cell death in a DTI suggests that ischemia is not the only factor contributing to tissue damage.\(^{13-17}\)

**ISCHEMIC REPERFUSION INJURY**

Ischemic-reperfusion injury is a two-step process that begins with the cessation of blood-flow to the muscle, leading to an oxygen deficit and buildup of toxic metabolites normally removed by the flowing blood. Restoration of blood flow stops and reverses the ischemic damage, but gives rise to a cascade of inflammatory responses including free radical production from the sudden influx of significant quantities of oxygen.\(^{17-20}\)

Reintroduction of oxygenated blood into an ischemic site may cause oxygen molecules to bind with the waste products of anaerobic metabolism. This produces a variety of oxygen-derived free radicals such as superoxide, hydrogen peroxide, hypochlorous acid, and more.\(^{12,21,22}\) Normally antioxidants protect cells against these oxygen-derived free radicals, but production of higher concentrations during sudden reperfusion of ischemic tissue may overwhelm the antioxidant system, resulting in irreversible cell damage.\(^{22}\)

Oxygen free radicals also have the potential to damage the vessel endothelium causing thrombosis which further contributes to ischemia. In addition, they produce inflammatory mediators and leukocyte adhesion molecules which compromise blood flow by occluding capillary openings. This congestion causes local capillaries to become more permeable, which contributes to the edema noted in tissue surrounding deep tissue injuries.\(^{22}\)

**SUSTAINED CELL DEFORMATION**

Evidence is growing to support cellular deformation as a key causative factor in the cell death associated with deep tissue injury.\(^{13-18}\) As a specific pressure threshold is exceeded, the cell is stretched and the pores of the cell membrane allow an increase in metabolites to move into and out of the cell until an imbalance occurs leading to cell death. The degree of damage is determined by the level of cell deformation and exposure time. In addition, the combination of pressure and shear can compress the cell membrane to the point of rupture.

**SUMMARY**

As research into the causative factors of deep tissue injury expands, it is clear that the tissue damage previously thought to be the result of ischemia alone is due to a combination of factors. The damage process in skeletal muscle tissue is caused by the level of deformation during short loading periods, however, during prolonged loading, ischemia and reperfusion will ultimately play a more important role.\(^{15}\) This damage can result from a single event or a series of ischemic cycles that results when the tissue is not allowed to fully recover.\(^{18}\)
What are the Treatments for Deep Tissue Injuries?

STANDARD TREATMENTS

- Pressure Relief/Off Loading
  - Repositioning schedules with side to side turning
- Support surfaces (i.e. low air-loss mattress, static air overlay)
- Heels in off-loading boots
- Nutritional Support

ADVANCED TREATMENTS

- MIST® Ultrasound Healing Therapy

MIST® Therapy is low frequency ultrasound delivered through a noncontact saline mist. The sound waves of this non-thermal, painless treatment penetrate into and below the surface to mechanically stimulate cells. As a result, healing barriers are removed and cells are stimulated to accelerate the normal healing process.23-29

<table>
<thead>
<tr>
<th>CAUSE OF DTI</th>
<th>IMPACT OF MIST®</th>
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<tbody>
<tr>
<td>ISCHEMIA</td>
<td>Increased Vasodilation</td>
</tr>
<tr>
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<td>Improves perfusion by dilation of the capillaries surrounding the wound bed to increase circulation, enhancing healing on a macro level.20</td>
</tr>
<tr>
<td>ISCHEMIA REPERFUSION INJURY</td>
<td>Increased Angiogenesis</td>
</tr>
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<td>The resulting stress placed on the capillaries leads to the stimulation of new capillaries on a micro level. Significantly more blood vessels (p&lt;0.05) were present in the granulation tissue of mice treated with MIST Therapy.31</td>
</tr>
<tr>
<td>CELL DEFORMATION</td>
<td>Reduction in Inflammation</td>
</tr>
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<td></td>
<td>Reduces in inflammation through the reduction of pro-inflammatory cytokines 26,27 and increases nitric oxide (NO) production which alleviates the negative impact of oxygen free radicals.21</td>
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<td>Cell Stimulation</td>
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<td>Cell stimulation is noted in cells treated with MIST Therapy by an increase of ERK (extracellular regulated kinase) and JNK (c-Jun N-terminal kinase) and the ERK/JNK ratio, which is thought to regulate cell proliferation and cell survival mechanisms.22</td>
</tr>
</tbody>
</table>

![Laser Doppler Perfusion Effects](image)

![Angiogenesis](image)

![Reduction of Pro-Inflammatory Cytokines](image)
Even with the best standard of care, the majority of deep tissue injuries will break down to become full thickness wounds requiring healthcare providers to deal with the associated negative outcomes.

<table>
<thead>
<tr>
<th></th>
<th>Baharestani Data Retrospective 2009 (N=200)</th>
<th>North Carolina WOCNs* Prospective2011 (N=42)</th>
<th>Honaker Retrospective 2012 (N=63)</th>
<th>N = 305</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneously Resolved</td>
<td>1%</td>
<td>5%</td>
<td>2%</td>
<td>7%</td>
</tr>
<tr>
<td>Progressed to Stage I/II</td>
<td>1%</td>
<td>Not Reported</td>
<td>20%</td>
<td></td>
</tr>
<tr>
<td>sDTI</td>
<td>26%</td>
<td>67%</td>
<td>30%</td>
<td>32%</td>
</tr>
<tr>
<td>Progressed to Stage III/IV</td>
<td>27%</td>
<td>28%</td>
<td>8%</td>
<td></td>
</tr>
<tr>
<td>Progressed to Unstageable</td>
<td>45%</td>
<td>Not Reported</td>
<td>40%</td>
<td>61%</td>
</tr>
</tbody>
</table>

*2 DTIs could not be assigned based on information provided

61% of DTIs progress to full thickness wounds with standard of care alone.

The addition of MIST Therapy to standard of care treatment significantly reduces the number of DTIs that will degrade to Unstageable, Stage III/IV Pressure Ulcers.

<table>
<thead>
<tr>
<th></th>
<th>Honaker Retrospective 2012 (N=64)</th>
<th>Honaker Prospective 2013 (N=43)</th>
<th>N = 107</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneously Resolved</td>
<td>18%</td>
<td>14%</td>
<td>75%</td>
</tr>
<tr>
<td>Progressed to Stage I/II</td>
<td>62%</td>
<td>54%</td>
<td></td>
</tr>
<tr>
<td>sDTI</td>
<td>5%</td>
<td>30%</td>
<td>15%</td>
</tr>
<tr>
<td>Progressed to Stage III/IV</td>
<td>6%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>Progressed to Unstageable</td>
<td>9%</td>
<td>2%</td>
<td>10%</td>
</tr>
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</table>

Only 10% of DTIs progress to full thickness wounds when MIST Therapy is added to Standard of Care.
**Financial Benefit of Early DTI Intervention with MIST® Therapy**

**STANDARD OF CARE (SOC)**

- Spontaneously Resolved Stage 2: 7%
- sDTI: 32%
- Unstageable Stage 3-4: 61%

**MIST® THERAPY + SOC**

- Spontaneously Resolved Stage 2: 75%
- sDTI: 15%
- Unstageable Stage 3-4: 10%

Adding MIST Therapy provides $36,000 saving per DTI patient

<table>
<thead>
<tr>
<th>COST ANALYSIS – 20 DTI PATIENTS</th>
<th>SOC</th>
<th>MIST® + SOC</th>
<th>Cost Saving</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cost of Stage I/II/DTI</td>
<td>($55,400 / 20) / 39%</td>
<td>($55,400 / 20) / 90%</td>
<td>$2,008,946 - $1,275,696 = $733,250</td>
</tr>
<tr>
<td>Cost of Stage III/IV/UN</td>
<td>($129,248 / 20) / 61%</td>
<td>($129,248 / 20) / 10%</td>
<td></td>
</tr>
<tr>
<td>Cost of MIST®</td>
<td>$0</td>
<td>($100 / 10 / per patient)</td>
<td></td>
</tr>
<tr>
<td>Total Overall Cost</td>
<td>$2,008,946</td>
<td>$1,275,696</td>
<td>$733,250/20 = $36,663</td>
</tr>
</tbody>
</table>
References:


For more information, contact your local Celleration representative or call 866.307.MIST (6478).