Local Skin Cooling as an Aid to the Management of Patients with Breast Cancer Related Lymphedema and Fibrosis of the ARM
HN Mayrovitz and JAYZer

Background and Objectives

Topical skin cooling causes local vasoconstriction that persists after skin temperature normalizes and also reduces the normal post-ischemic hyperemic response from tissue indentation loading. Skin cooling also causes systemic vasoconstriction that combined with locally induced vasoconstriction decreases capillary-to-interstitial fluid filtration and promotes post-capillary fluid reabsorption. Such enhanced processes tend to reduce interstitial fluid volume. Further, given the use of cooling to help treat and blunt edema formation and effects of elevated environmental temperatures on lymphedema (LE), we were surprised that cooling has not been used as a therapeutic modality for LE. Also, LE is often associated with co-present inflammation and fibrosis processes so that skin surface cooling, which can cool to a 2 cm depth, might be a way to have positive impacts on these processes. In treating patients who have developed breast cancer treatment related LE (BCRL) we have noted that skin tissue areas most bothersome to patients were areas with sensed elevated skin heat suggesting underlying inflammatory processes often in conjunction with palpable fibrosis. In an effort to provide relief to these patients topical cooling was integrated into their physical therapy and lymphedema treatment session. An unforeseen yet welcome observation associated with that topical cooling was the apparent reduction in tissue firmness as judged by palpation and effort expenditure during treatment. The purpose of the present research was to systematically and quantitatively evaluate the impact of skin tissue cooling on skin tissue water content and skin indentation resistance in women with documented BCRL.

RESULTS

SUBJECTS: 20 women referred for BCRL therapy of arm LE participated; All signed an IRB approved consent. Baseline features of this group are summarized in Table 1.

Table 1. Baseline Pre-Cooling Parameters: Entries are mean ± SD for 12 breast patients. \( F_{1.3} \) and \( F_{4.0} \) are forces to indent skin tissue to 4.0 and 1.3 mm. The \( %H_2O \) is skin water percent to a depth of 2.0–2.5 mm at force measurement sites. TDC is skin tissue dielectric constant (dimensionless). TSK is breast skin temp at 5 Breasts of Arm

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control Arm</th>
<th>Affected Arm</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Force ( F_{4.0} ) (N)</td>
<td>2.16 ± 0.70</td>
<td>3.92 ± 1.04</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Force ( F_{1.3} ) (mN)</td>
<td>56.9 ± 14.4</td>
<td>111.6 ± 53.1</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>( %H_2O )</td>
<td>43.3 ± 9.5</td>
<td>76.0 ± 19.3</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>TDC</td>
<td>32.8 ± 7.2</td>
<td>57.5 ± 14.7</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>TSK ( ^\circ C )</td>
<td>32.5 ± 1.3</td>
<td>32.4 ± 1.4</td>
<td>0.251</td>
</tr>
<tr>
<td>Girth (cm)</td>
<td>23.6 ± 4.0</td>
<td>28.8 ± 4.9</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

TABLE 1

Measurement Methods

- **F_{4.0:**
  - Force Recording
  - Measuring Indentation Force

**F_{1.3:**
  - Base Plate
  - Skin

- **Girth:**
  - Skin tissue width

- **HT:**
  - Skin tissue %H

- **TDC:**
  - Tissue dielectric constant (TDC) values. Device is the MoistureMedterD Compact.

**Method:**

- Skin tissue %H

**Protocol:**

- By adding cold...to the end of MLD session.

**Main Results:**

- Results show that arm skin cooling softens lymphedematous and fibrotic tissue by about 24% to 28% depending on indentation depth. This appears to occur without a significant change in skin fluid content at least to a depth of about 2.5 mm. Although the precise mechanism linking cooling to softening is as yet not fully understood the fact that tissue is softened carries with it many potential benefits to patient and therapist. The near immediate tissue softening is associated with less pressure on underlying nerve endings and lost input to sensory nerves thereby interrupting the pain cycle resulting in rapid pain relief. The rapidly softened tissue and decreased perception of pain offers the patient hope and encourages their therapeutic journey to reclaim functional use of their affected further. Because softer tissue becomes more pliable, myofascial lengthening, scar tissue releasing and other aspects of treatment are easier for the therapist to perform thereby reducing treatment time and effort while achieving improved functional mobility. The suitability of cooling and its optimal treatment parameters as a standard component to lymphedema therapy and self-management needs to be prospectively determined via further research.

**Subjects:**

- All signed an IRB approved consent.

**Initial PROCEDURES:**

- Skin tissue %

**Sequence PROCEDURES:**

- Post-cooling measurements were done at the target sites as indicated in Fig. 4.

**Conclusion:**

- Skin temperatures (TSK) measured at the target sites; Error bars are SEM. Pre-cool TSK does not differ between control and lymphedematous (LE) arms, but skin cooling reduces TSK from 32.4 ± 1.4 (SD) to 23.7 ± 2.0 for an average reduction of 8.7 ± 2.1 °C. TSK remains less than pre-cooling at the end of the MLD session.

Fig 1. Tiss-U-Press indentation force to a depth of 4.0 mm (\( F_{4.0} \)) in N

Fig 2. Indentation force using the SkinFibrometer to a depth of 1.3 mm (\( F_{1.3} \)) in mN

Fig 3. Skin tissue \( %H_2O \) estimated to a depth of 2.0–2.5 mm using tissue dielectric constant (TDC) values. Device is the MoistureMedterD Compact.

Fig 4. Sequential Procedures: All measurements were made at target sites at least in triplicate before and after cooling and treatment as schematized in the above diagram.

Fig 5. Skin Cooling Effects: Data show cooling effects on indentation forces to A) 4.0 mm (\( F_{4.0} \)) and B) 1.3 mm (\( F_{1.3} \)) and C) skin TDC associated with treated arm patients with the contralateral control arm as reference. Error bars are SEM. Main observable effect is a significant reduction in indentation forces with small additional effects of MLD. Cooling showed essentially no effect on skin water percentage as assessed by TDC.

Fig 6. Skin Temperature Profile: Data show skin temperatures (TSK) measured at the target sites; Error bars are SEM. Pre-cool TSK does not differ between control and lymphedematous (LE) arms, but skin cooling reduces TSK from 32.4 ± 1.4 (SD) to 23.7 ± 2.0 for an average reduction of 8.7 ± 2.1 °C. TSK remains less than pre-cooling at the end of the MLD session.

Fig 7. Absence of Skin Wetting Effect: Data show an indentation force to 4.0 mm (\( F_{4.0} \)) associated with skin wetting in 5 arm and 5 breast patients. Skin temperatures shown are those measured at the end of the wetting, cooling and MLD sessions. Main observable effect is a non-significant (NS) change in F4.0 due to simple wetting but a significant reduction due to cooling.

For Table 1, see image 108x2305 to 402x2580.

Fig 7. Absence of Skin Wetting Effect: Data show an indentation force to 4.0 mm (\( F_{4.0} \)) associated with skin wetting in 5 arm and 5 breast patients. Skin temperatures shown are those measured at the end of the wetting, cooling and MLD sessions. Main observable effect is a non-significant (NS) change in F4.0 due to simple wetting but a significant reduction due to cooling.