

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

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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-interstitium 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 palpated 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 experienced 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.

Protocol

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**.

MEASUREMENTS: All were bilateral and made prior to cooling, after cooling and after MLD. Skin temperature (TSK) was measured with an IR thermometer and arm girth measured with a Gulick tape measure. Forces needed to indent skin tissue to 4.0 mm ($F_{4.0}$) were measured as shown in **Fig 1** and forces to indent to 1.3 mm ($F_{1.3}$) was measured as shown in **Fig 2**. Skin tissue water was assessed via tissue dielectric constant (TDC) measurements and expressed as %H₂O as indicated in **Fig 3**.

INITIAL PROCEDURES: As part of pre-treatment assessments, arm girths were measured at 5 sites on both arms. Lower and upper arm volumes were calculated from a frustum model and ratios of affected to control arm volumes determined. The group had upper arm volume ratios of 1.114 ± 0.088 and lower arm ratios of 1.139 ± 0.119 . This corresponds to edema volumes of 13.9% and 11.4%. Then, an arm area that was determined by palpation to be the most fibrotic was chosen as the target site.

SEQUENTIAL PROCEDURES: The pre-cooling measurements were done at the target site as indicated in Fig 4. Steps for cooling consisted of preparing an ice water bath in a large bowl and allowing the ice-water bath to temperature equilibrate. Washcloths were then dunked into the cold bath for about five seconds then removed, wrung out and laid over the affected area. Depending on size of the area to be covered 2-4 washcloths were utilized for optimal coverage with an overall cooling time of about 12-15 minutes. The patient was asked to touch the cold washcloths prior to first-time draping of the affected area to prepare for placing of the cold washcloth and decrease the impact of the cold sensation on initial skin contact. If requested by the patient the temperature of the water into which the washcloths were dunked was adjusted with room temperature water to accommodate for varying patient sensitivity to cold water. This process was repeated 3-4 times until a drop in skin temperature of 7-9 °C was achieved. Following the collection of the post-cooling data the patient received MLD and physical therapy care per individual patient Plan of Care.

Table 1. BASELINE PRE-COOLING PARAMETERS: Entries are mean \pm SD for 12 breast patients. $F_{4.0}$ and $F_{1.3}$ are forces to indent skin tissue to 4.0 and 1.3 mm. The %H₂O is skin water percent to a depth of between 2.0-2.5 mm at force measurement sites. TDC is tissue dielectric constant (dimensionless), TSK is breast skin temp at sites of force and %H₂O measurements.

Measurement Methods

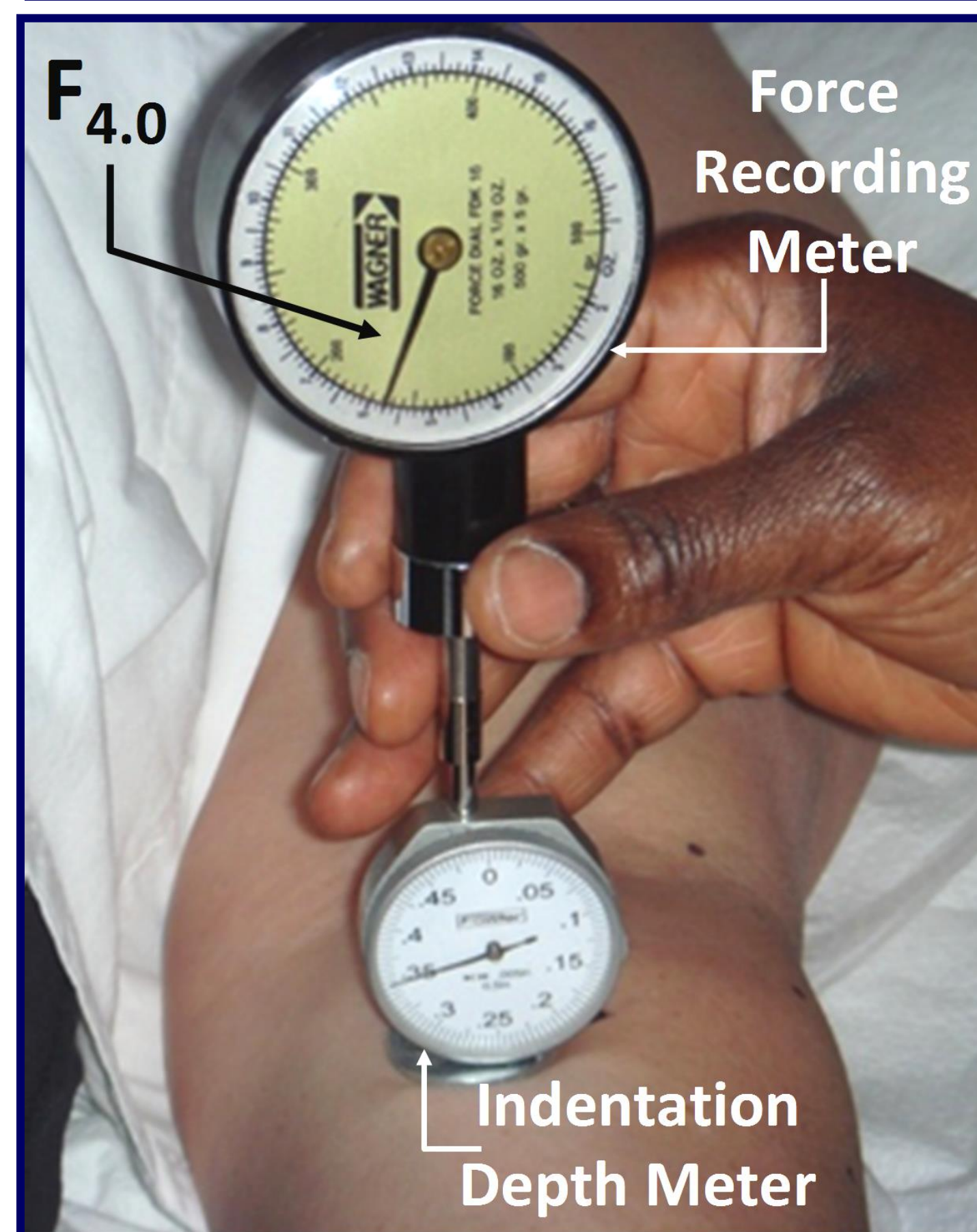


Fig 1. Tiss-U-Press indentation force to a depth of 4.0 mm ($F_{4.0}$) in Newtons (N)

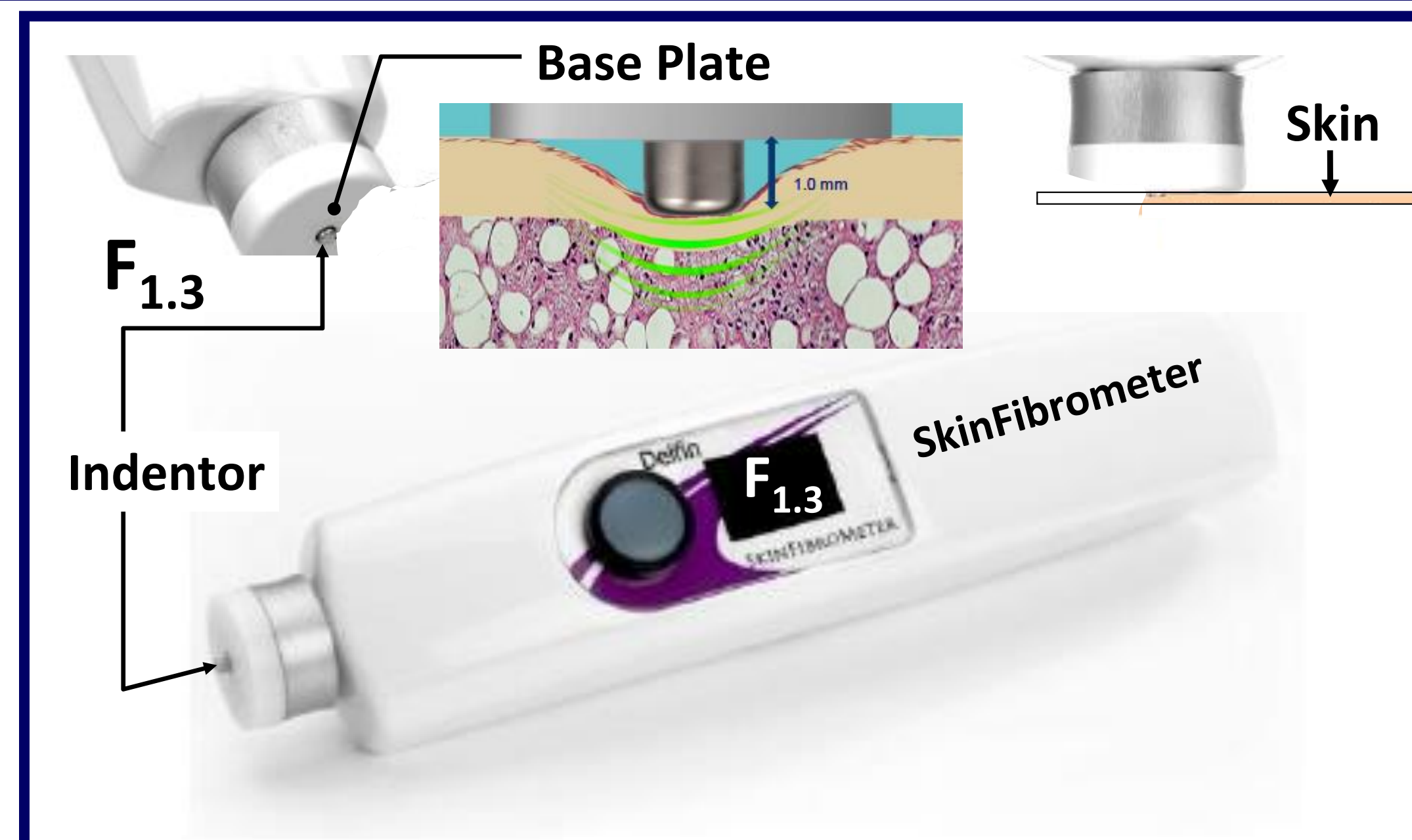


Fig 2. Indentation force using the SkinFibrometer to a depth of 1.3 mm ($F_{1.3}$) in mN

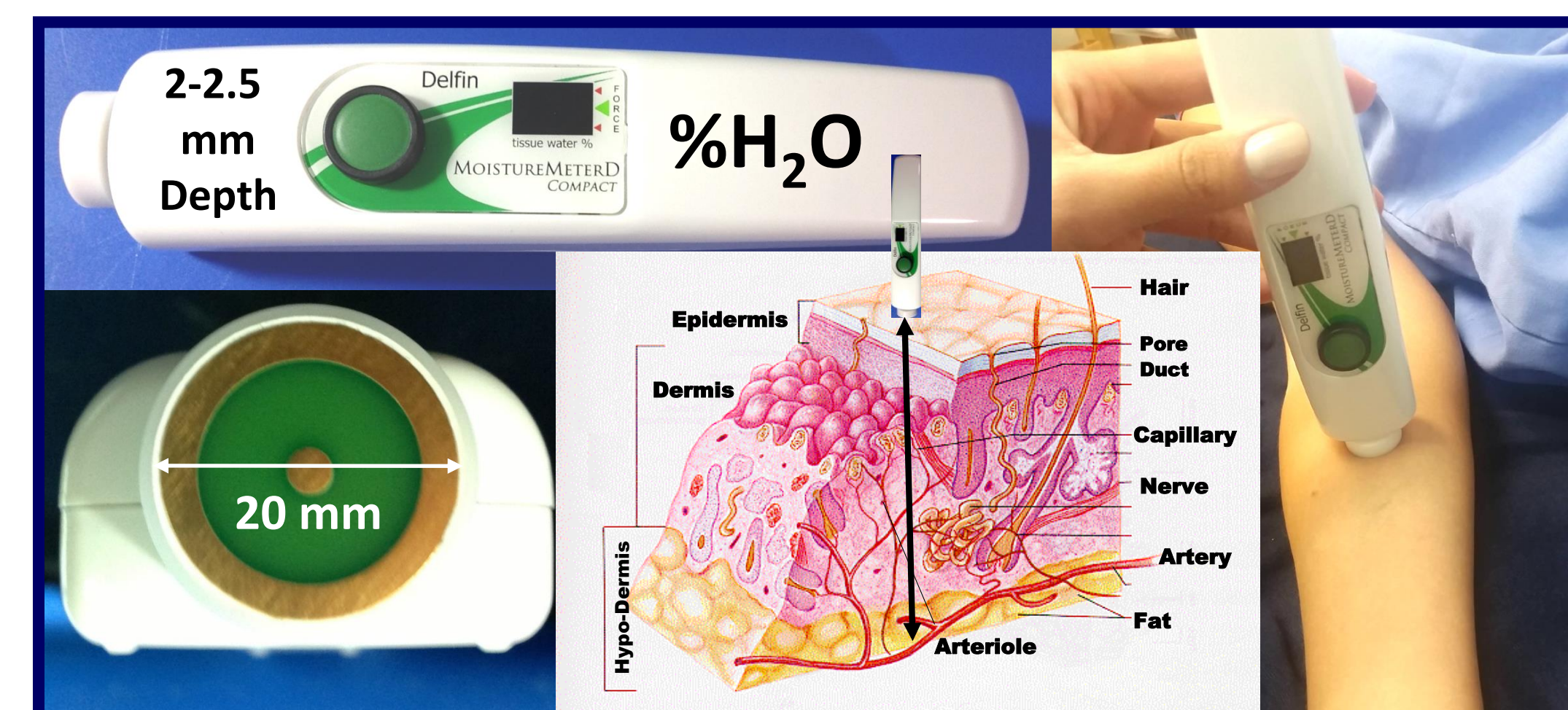


Fig 3. Skin tissue %H₂O estimated to a depth of 2.0 – 2.5 mm using tissue dielectric constant (TDC) values. Device is the MoistureMedterD Compact.

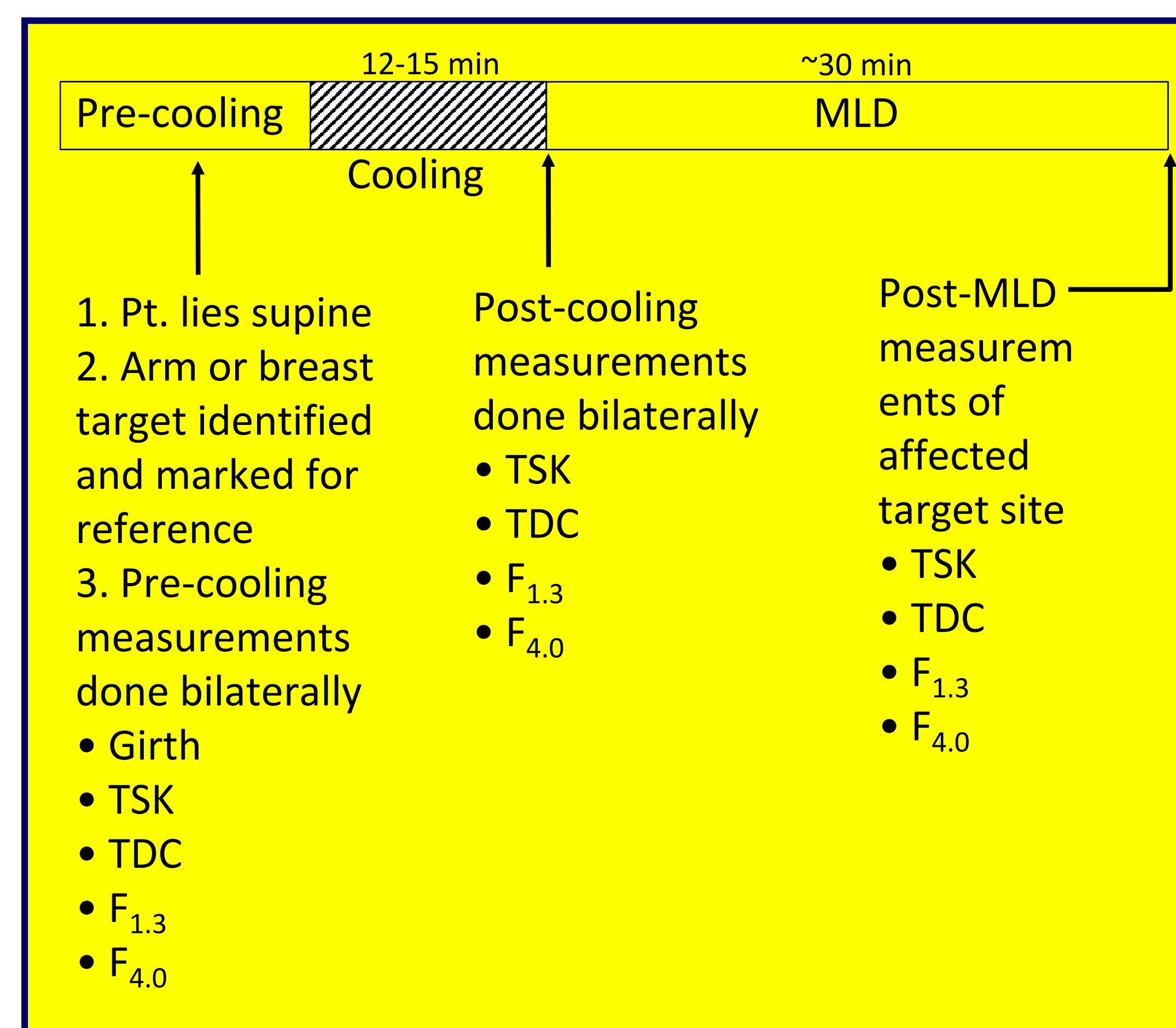


Fig 4. Sequential Procedures: All Measurements were made at target sites at least in triplicate before and after cooling and after treatment as schematized in the above diagram

TABLE 1	Control Arm	Affected Arm	p-value
$F_{4.0}$ (N)	2.16 ± 0.70	3.92 ± 1.04	< 0.0001
$F_{1.3}$ (mN)	54.9 ± 14.4	111.6 ± 53.1	< 0.0001
%H ₂ O	43.3 ± 9.5	76.0 ± 19.5	< 0.0001
TDC	32.8 ± 7.2	57.5 ± 14.7	< 0.0001
TSK °C	32.5 ± 1.3	32.4 ± 1.4	0.251
Girth (cm)	23.6 ± 4.0	28.8 ± 4.9	< 0.0001

Main Results

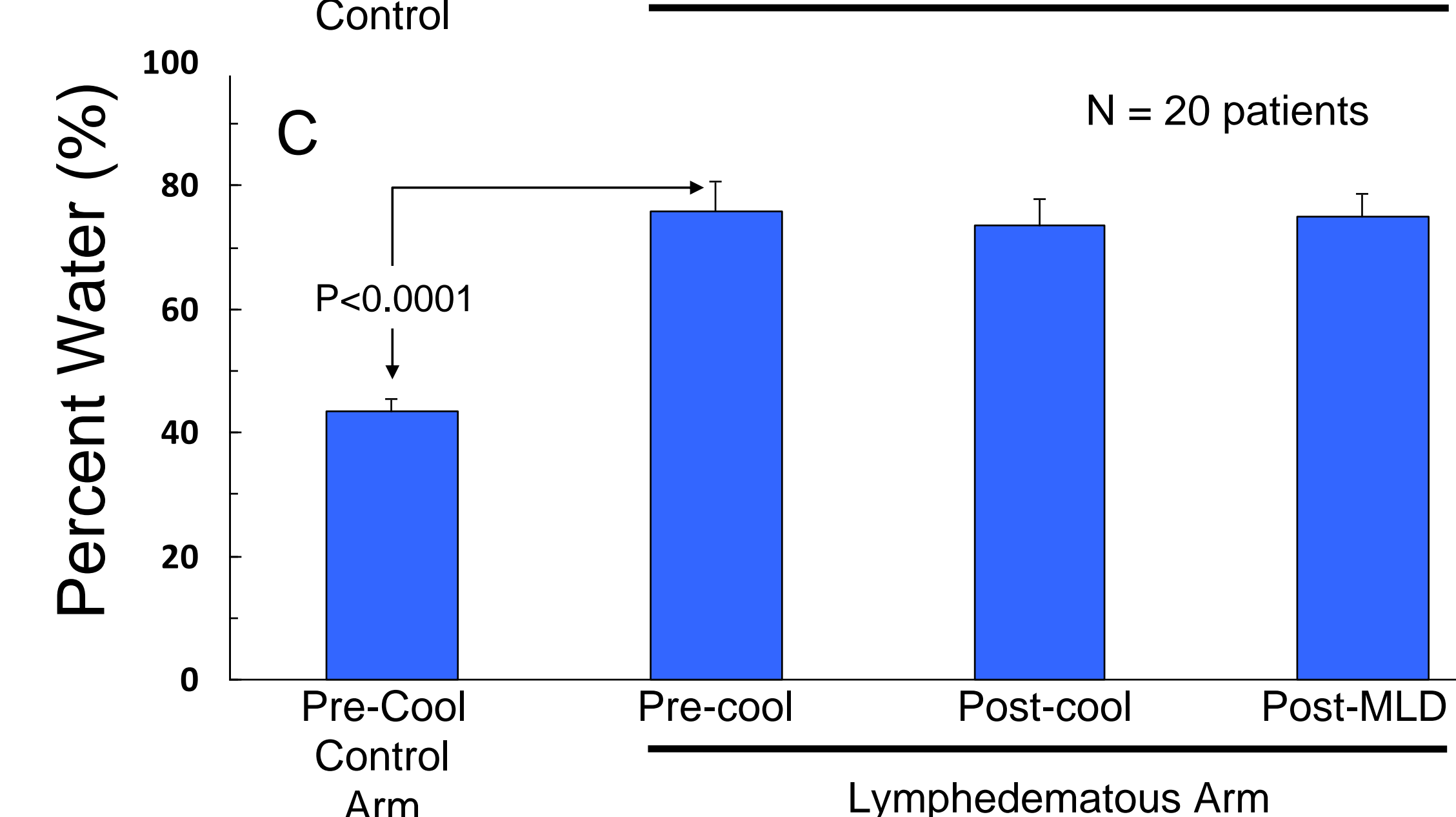
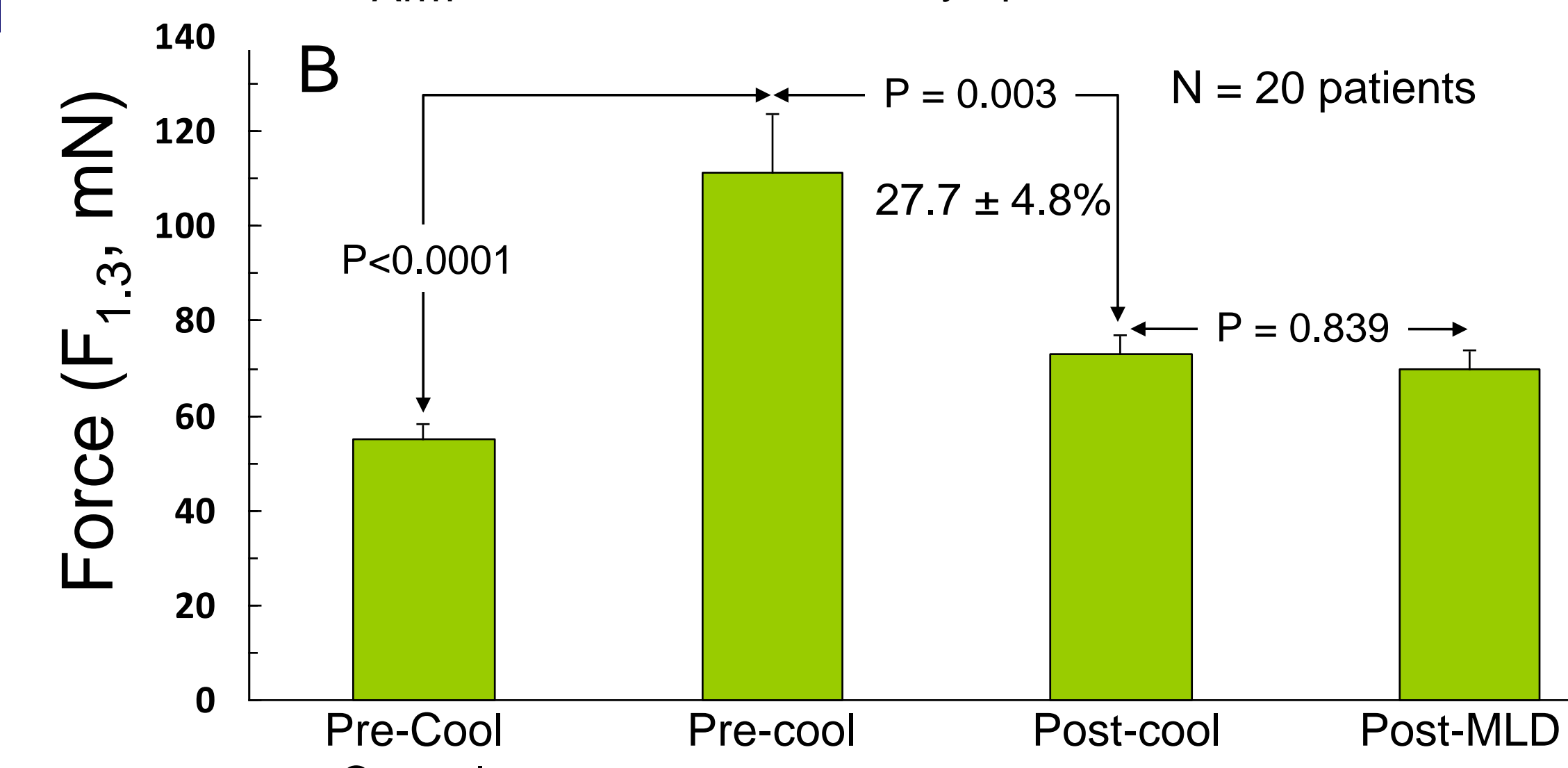
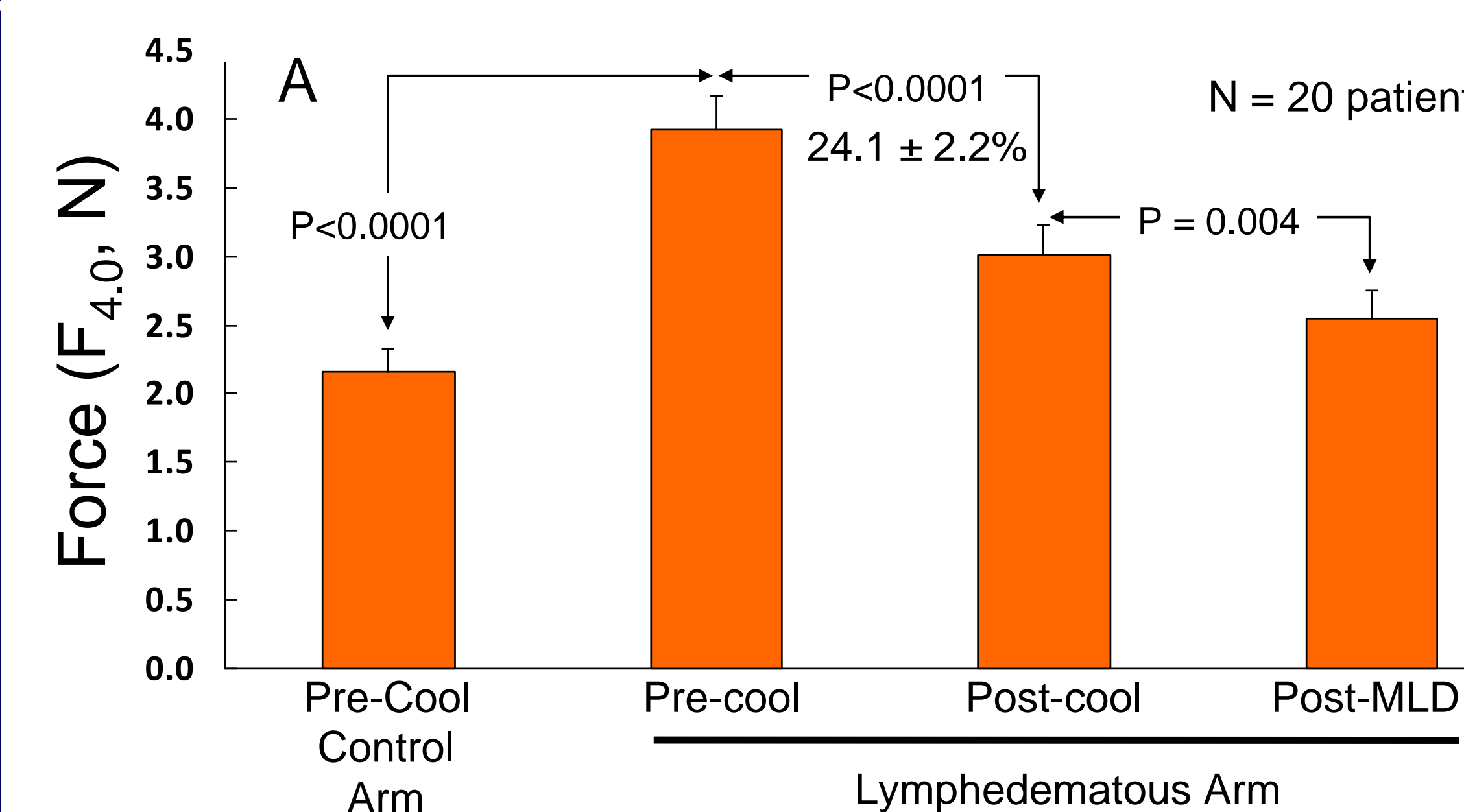


Fig 5. Skin Cooling Effects: Data show cooling effects on indentation forces to A) 4.0 mm ($F_{4.0}$) and B) 1.3 mm ($F_{1.3}$) and C) skin %H₂O associated with treatment of 20 arm patients with the contralateral control arm as reference. Error bars are SEM. Main observable effect is a significant reduction in indentation forces with small or no additional effects of MLD. Cooling showed essentially no effect on skin water percentage as assessed by TDC.

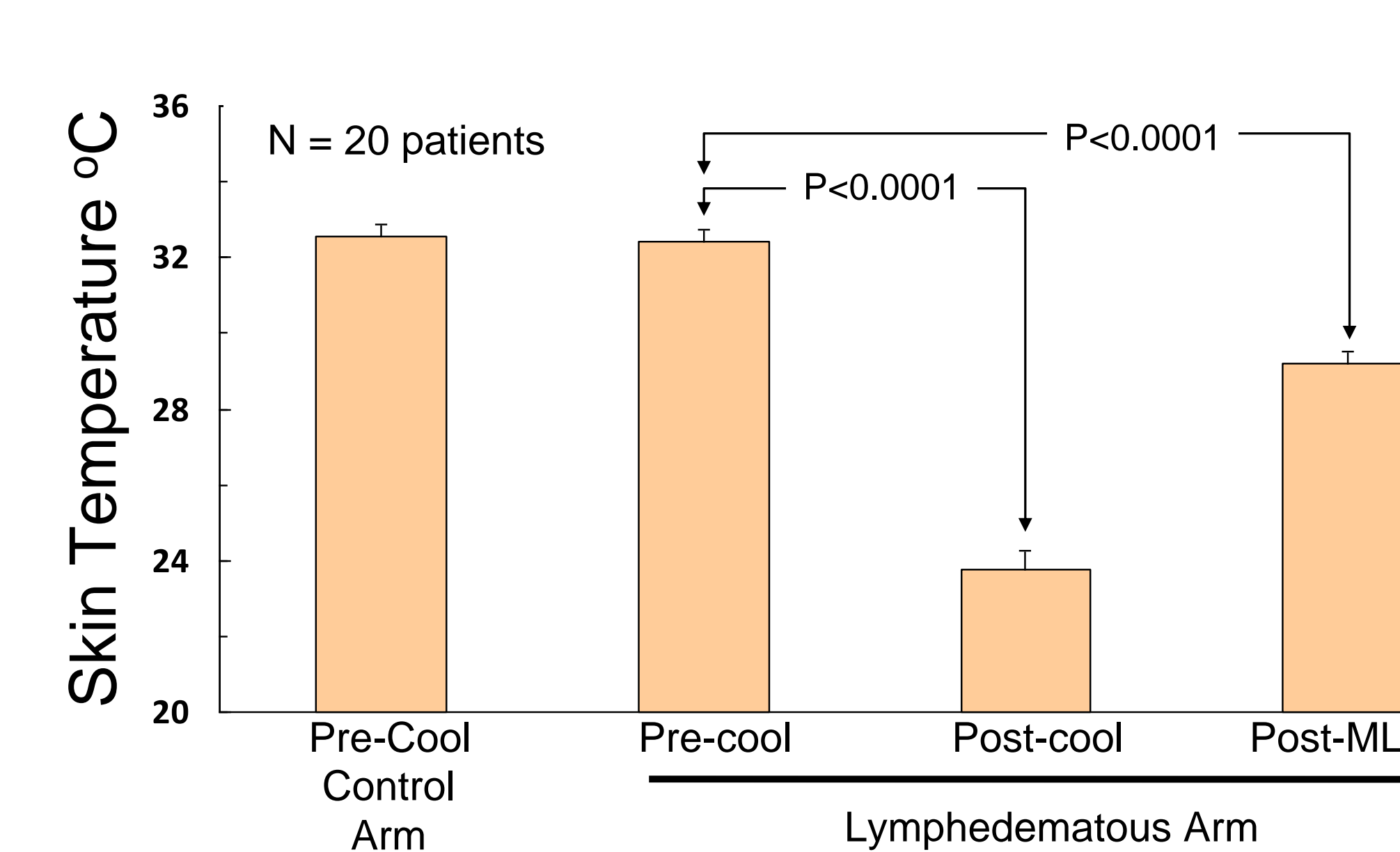


Fig 6. Skin Temperature Profile: Data show skin temperatures (TSK) measured at the target sites; Error bars are SEM. Pre-cool TSK does not differ between control and lymphedematous (LE) arms but skin cooling reduces TSK from 32.4 ± 1.4 (SD) to 23.7 ± 2.0 for an average reduction of 8.7 ± 2.1 °C. TSK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

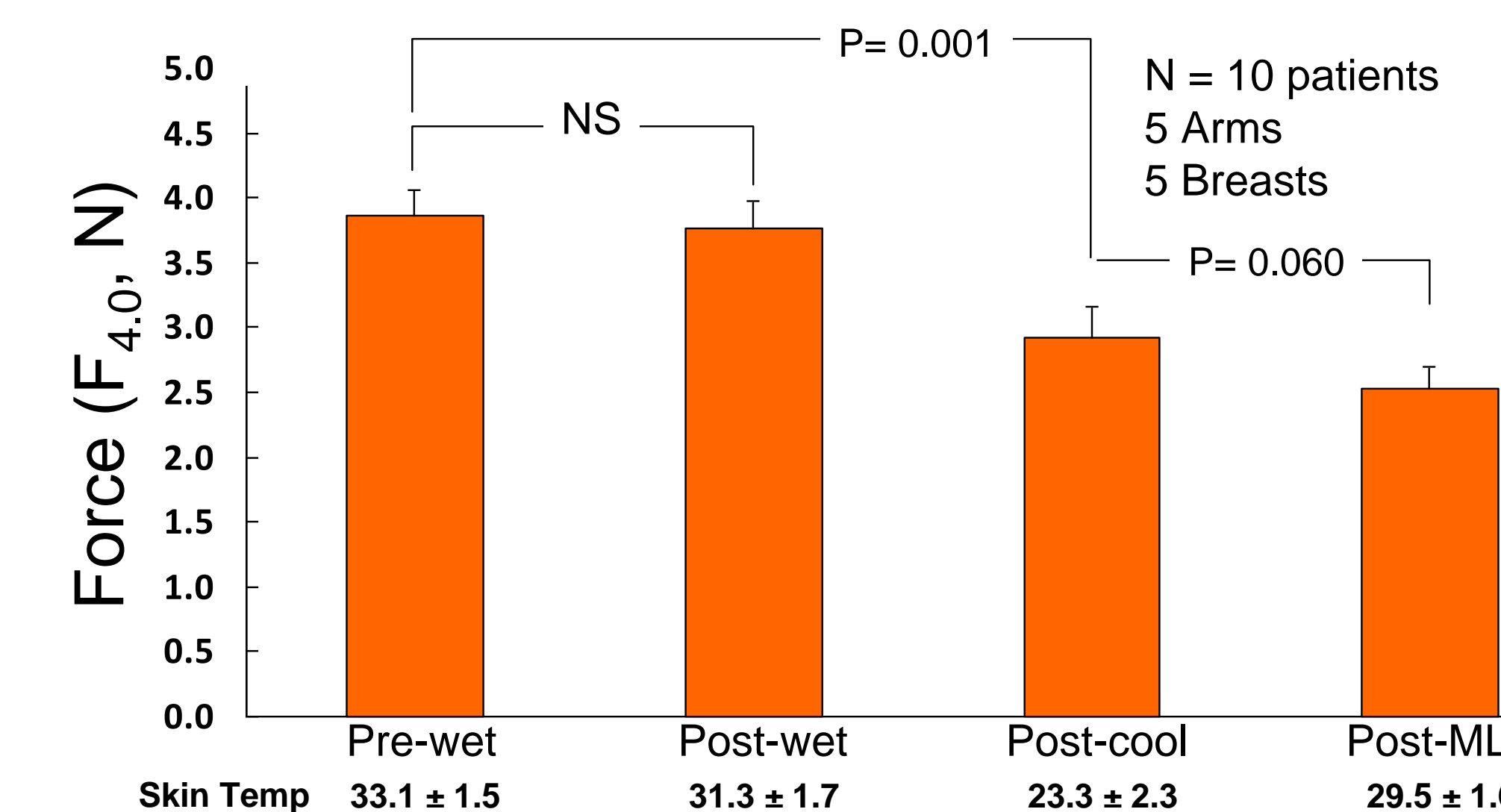


Fig 7. Absence of Skin Wetting Effect: Data show effects on indentation force to 4.0 mm ($F_{4.0}$) associated with skin wetting in 5 arm and 5 breast patients. Skin temperatures shown are those measured at the end of the wetting, cooling and MLD intervals. Main observable effect is a non-significant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

Conclusions

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 less 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 encouragement in their therapeutic journey to reclaiming functional use of their affected body. 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.