The use of vapocoolants for relief of pain resulting from activation of trigger areas located in muscles was reported by Travell in 1949. She developed a special technique for the application of ethyl chloride (EC), a vapocoolant. This technique, spray and stretch, entails placing the affected muscle in a position of stretch and spraying in one direction only at an acute angle to coat adjacent skin areas overlying the muscle with the coolant. Gentle passive stretch is applied while spraying. Travell cautioned that EC could freeze the skin and that this topical agent could potentially increase pain by activating latent trigger areas.

Fluori-Methane Spray® (FMS) replaced EC in the early 1960s as the vapocoolant of choice. This agent is a combination of 15% dichlorodifluoromethane and 85% trichloromonofluoromethane and is packaged in a bottle with a finely calibrated nozzle. Evaporation of FMS causes less decrease in skin temperature than that of ethyl chloride; therefore, the potential for skin frosting is decreased. Researchers have ascribed similar effects for FMS and EC; that is, researchers have hypothesized that FMS relieves pain associated with muscle spasm and myofascial trigger points and increases joint range of motion, which is restricted by muscle spasm.

Although many clinical case studies document the effectiveness of FMS, only empirical evidence exists for its mechanism of action. Travell identified three possible mechanisms: receptor adaptation, counterirritation effect, and a neurogenic effect. Receptor adaptation was considered to be a brief "refrigeration anesthesia" of cutaneous receptors that were located in the path of the spray. Counterirritation referred to a barrage of action potentials from cold receptors that would block the noxious input. According to Travell, these two mechanisms contributed to the neurogenic effect. The mechanism of action of vapocoolants needs to be examined in terms of newer scientific evidence and the placebo effect. Based on current literature, a single brief, intense stimulus can produce inhibition within the CNS. Because vapocoolants can be considered brief, intense stimuli, their effect should be similar in increasing passive hip flexion when the range is limited by neurogenic factors.

The purposes of the study were twofold: 1) to replicate one quantitative study on FMS and 2) to examine other brief, cold stimuli applied through the spray and stretch technique to increase passive hip flexion in healthy adults. The two research questions were as follows: Is FMS in combination with stretch more effective than stretch alone? Is there a difference in the type of brief, cold stimuli applied in the spray and stretch technique for increasing passive range of joint motion? Hypotheses formulated from the research questions were 1) FMS combined with passive stretch produces a significantly greater increase in hip flexion than passive stretch alone and 2) no difference exists among the brief, cold stimuli in combination with passive stretch in comparison with stretch alone to increase hip flexion. For the purpose of this study, the brief, cold stimuli were isopropyl alcohol delivered through a small gauge needle, EC, and FMS. Hip flexion was measured by the pelvifemoral angle. This method is consistent with other studies examining FMS to increase passive hip flexion or examining isometric contraction versus passive stretch to increase passive hip flexion.

**METHOD**

**Subjects**

Eighty-four college-age subjects with no known orthopedic or neurologic condition that would limit hip flexion volunteered to participate in the study. Each subject signed a
Procedure

**Question one.** Each subject was positioned side lying on the table with the right lower extremity on the secondary table on the 0-degree line. The proximal tip of the greater trochanter was aligned with the plumb bob. I then secured the subject's pelvis by applying stabilization to both anterior superior iliac spines and to the sacrum. An ankle cuff was attached 1-in superior to the lateral malleolus and a cable was attached from the cuff to the load cell. The housing of the load cell pivoted to maintain a 90-degree angle between the long axis of the lower extremity and cable throughout the procedure. The lower extremity was then secured to a scooter board to maintain knee extension and to facilitate movement of the leg by me.

After the subject was positioned, the threshold force for passive hip flexion was obtained. I moved the leg approximately 5°/sec until the subject felt a slight "pull" in the popliteal fossa. At that point, both the angle and force were recorded. These values served as the pretest values.

Subjects in the control group maintained the position for 40 seconds. Subjects in the experimental group received a standard spray procedure. Six applications of FMS, each five seconds in duration, were applied in a distal to proximal manner to skin on the posterior aspect of the thigh. The entire spray procedure lasted no longer than 40 seconds. I then passively moved the leg until the pretrial force reading was matched. When the force readings were the same, I recorded the angle. The use of the force readings eliminated the need to rely on the subject to replicate the same "pull" sensation in the popliteal fossa.

**Question two.** The brief, cold stimuli in the experimental groups were FMS, EC, and isopropyl alcohol. Isopropyl alcohol was delivered by a syringe through a small gauge needle to mimic the delivery system of FMS and EC. I used the same procedures for securing the subject, applying the treatment, and obtaining the pretest and posttest measures as described for question one.

Data Analysis

To test the first hypothesis, a one-tail t test \( (p < .05) \) was performed on the mean difference between the pretest and posttest values for the control and experimental groups. To test the second hypothesis, the difference between the pretreatment and posttreatment angles was used in a one-way analysis of variance (ANOVA) \( (p < .05) \). \(^{10}\)

## RESULTS

For the first question, the pretest and posttest group means for passive hip flexion in the experimental group were \( 112.2^\circ \) and \( 121^\circ \), respectively, and for the control group, \( 118.4^\circ \) and \( 123.5^\circ \), respectively. The mean difference in angle of hip flexion before and after treatment for the experimental group was \( 8.78^\circ \) \( (s \pm 4.97) \) and the mean difference for the control group was \( 4.86^\circ \) \( (s \pm 4.51) \). The result of the one-tail \( t \) test was that FMS in combination with stretch did not increase passive hip flexion \( (p < .05) \). These results do not support the earlier study on FMS. \(^{7}\) For the second question, results of the ANOVA to test the hypothesis pertaining to the use of any brief, cold stimulus to increase hip flexion are presented in the Table. No significant difference \( (p < .05) \) was noted for

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\(^{†}\) Gould-Statham Products, 2230 Statham Blvd, Oxnard, CA 93030.

\(^{‡}\) Data Precision Corp, Andover, MA 02115.
the combination of any brief, cold stimulus and passive stretch as opposed to passive stretch alone.

DISCUSSION

Although brief, cold stimuli have been demonstrated to increase passive joint range of motion in cases of muscle spasm, this is apparently not the case in the individual not exhibiting pain. The rationale for the findings of this study may be described in terms of the effects of vapocoolants on a quiescent CNS as opposed to a CNS with heightened excitability from pain and muscle spasm. In the pain-spasm-pain cycle, a positive feedback mechanism can be established that increases (potentiates) the effects of the noxious stimulus. The gamma loop mechanism has been implicated as a contributing factor to this self-sustaining reflex hypertonnia. We could hypothesize that brief, cold stimuli activate a negative feedback loop that could decrease (depotentiates) the amount of noxious stimuli reaching various levels of the CNS. Another example of a negative feedback loop is the assumption of the analgesic posture. Movement out of this position would be limited by pain.

One procedure for interrupting the pain-spasm-pain cycle by a brief, intense cold stimulus was proposed by Ellis. He considered the cold stimulus to bombard “central pain receptor areas” and swarm out the pain impulses. This phenomenon could occur by the “gating” or blocking of pain impulses at the spinal or higher center level. Higgins and colleagues supported this view and noted that when a painful electrical stimulus and a tactile stimulus were applied concurrently, perception of the electrical shock was attenuated. Furthermore, Satran and Goldstein noted a reduction of the somatosensory cortical evoked potential when a vibratory electrical stimulus was paired with an electrical shock. The gating mechanism could also be the basis for the effectiveness of the vapocoolant spray as a counterirritant. Transmitting fewer pain impulses to nerve centers where they are perceived and interpreted would permit an increase in the range of motion that the patient previously considered painful. Based on this postulate, the individual without a pain-spasm-pain cycle would not demonstrate a significant increase in joint range of motion. This was supported in my study.

An idea that warrants further exploration is related to the use of stimuli for their psychogenic effects. Travell considered that some but not the total effects of EC for alleviation of acute “stiff neck” were due to psychogenic factors. Thorsteinsson and colleagues have demonstrated that other forms of cutaneous stimuli such as transcutaneous electrical nerve stimulation applied as a placebo are effective in producing analgesia. In 32% of the trials, analgesia was produced by placebo TENS application in comparison with 48% of the trials for the actual TENS stimulation. A similar consideration with positive feedback by the therapist could potentiate the effects of the vapocoolant spray.

CONCLUSION

The results of the study demonstrated that a single application of a brief, cold stimulus did not increase passive hip-flexion range of motion in healthy subjects. The clinical observation by Travell, however, that a single application could increase range of motion was not refuted by this study because no patients exhibiting pain and muscle spasm were included. I refined a technique for obtaining reliable measurements of hip flexion. These results can serve as a basis for studying the effects of therapeutic interventions with patients who exhibit limited range of hip flexion associated with pain and the pain-spasm-pain cycle.

REFERENCES


| TABLE
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*p < 0.5.