

The Effect of Thoracic Spine Mobilization On Lower Trapezius Strength Testing

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Abstract: Evidence has shown that muscle strength and function become altered due to motion restrictions. Strengthening muscles with traditional therapeutic exercise will not be completely successful unless inhibition is removed by restoring normal joint mechanics. This study investigated the effect of Grade-IV thoracic spine mobilizations on lower trapezius strength testing in normal subjects. The mobilization for the treatment group (n=20) consisted of posterior-anterior (P-A) oscillations performed from T6-T12 at each segment's end range (Grade-IV). This technique is aimed at restoring normal joint play. The control group (n=20) received a Grade-I mobilization consisting of P-A oscillations performed at the beginning of the joint's range, which is not expected to have articular reflexogenic effects. Before and after the mobilizations, isometric muscle strength of the lower trapezius was measured using a Nicholas Manual Muscle Tester. An independent group t-test comparing the groups demonstrated a statistically significant effect of thoracic spine mobilization on lower trapezius strength testing ($P < .05$).

Key Words: Arthrokinetic Reflex, Muscle Inhibition, Mobilization, Mechanoreceptor, Hypomobility, Thoracic Spine.

A kypholordotic posture is a common finding among the general population, presenting clinically in subjects who have forward head posture, restricted thoracic spine extension, and increased lumbar lordosis^{1,2}. A joint must have normal mobility in order for its corresponding muscles to work efficiently; according to Hurley³, a muscle cannot attain its full function unless inhibition is removed. Previous studies have addressed the effects of mobilization on muscle function, but they have neglected to explain the joint's reflexogenic effects on muscle. The arthrokinetic reflex (AKR) is responsible for these effects as it links the central nervous system to skeletal muscles⁴. The

regulators of this reflex are the articular mechanoreceptors located within synovial joint capsules⁵.

The joint capsule receptors, Types I-IV, exert a reflexive effect on muscle tone⁴. Afferent nerve fibers of these receptors project to motor neurons within the CNS, thereby contributing to the continuous modulation of activity flowing to the muscle spindle. When a stretch on a joint capsule is initiated, the mechanoreceptors exert reciprocally coordinated reflexogenic influences on muscle tone and on the excitability of stretch reflexes in striated muscles⁶. This reflex inhibits muscles from recruiting the maximal number of motor units and protects the body from overstressing restricted joint structures^{4,6}.

Weakness of the lower trapezius is a common clinical finding. This is associated with the upper cross syndrome commonly seen with poor posture⁷. As described by Janda, this pattern is associated with a flattened cervical spine, forward head, protracted and elevated shoulders, and an increased thoracic kyphosis⁷. Limitations in thoracic extension, secondary to localized prolonged flexion,

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leads to muscle tightness and adaptive shortening of the anterior longitudinal ligament and facet capsules⁹.

The anterior-facing superior articular facets assume a superior and anterior position relative to the posterior-facing inferior facets. Tightened capsular and ligamentous tissues restrict spinal motion, limiting full extension. Active thoracic extension causes the superior facets to slide in an inferior direction¹⁰. Maximal closing of the restricted facet joints stretches the surrounding tissues, prematurely activating the AKR mechanoreceptors; this neural response will inhibit agonistic muscles of this movement, including the lower trapezius, which is profoundly affected⁴. As a consequence of restricted thoracic extension from T6-T12, lower trapezius strength is theoretically under neural inhibition; therefore, it would be expected to demonstrate some degree of weakness upon testing.

Joint mobilization has been known to return physiologic and accessory motions to hypomobile structures¹¹⁻¹³. As with typical diarthrodial joints, dense, irregular connective tissue comprises the outermost layer of facet capsules⁹. This layer becomes thickened and immobile in joints that have a capsular pattern of hypomobility. Grade-III and -IV mobilizations will cause plastic deformation of collagen in the outermost capsular layer, thereby restoring its normal length¹⁰. The Grade-IV mobilization used on the experimental group consisted of P-A oscillations performed into the tissue resistance¹⁴. The Grade-I P-A mobilization used on the control group was performed at the beginning of the range and therefore met no tissue resistance.

Review of the Literature

Understanding of the AKR requires a brief review of articular neurology. Embedded within capsule fibers are mechanoreceptors that relay messages for direct reflexive actions on muscles⁴. There are four types of receptors, as described by Freeman and Wyke⁵. Type-I receptors are firmly established in the outer layer of the joint capsule, functioning continuously during static and dynamic movements. Type-II receptors are located deeper within the capsule and fire only during joint movement. Type-III receptors are found on the surfaces of ligaments, as well as joint capsules, and respond to the application of tensile forces^{15,16}. Type-IV receptors are interwoven throughout the entire thickness of the capsule, responsible for evoking joint pain. Though all four receptors mediate reflex effects on muscle activity, the AKR is affected primarily by Type-I and -II receptors.

All synovial joints have predetermined patterns of mechanoreceptors that play a role in reflexes, kinesthesia, proprioception, and nociception¹⁷. The concentration of mechanoreceptors appears in higher densities in areas that undergo extremes of movement¹⁸ rather than midranges of joint motion^{19,20}. The receptors, through reflexive action, maintain the first line of defense in sensing these extremes, alerting the CNS of impending injury¹⁸.

Histological studies on the connective tissues throughout

the spinal column have concluded that specific patterns of receptors exist²¹⁻²³. The facet joints are well innervated by the medial branches of two dorsal consecutive rami²⁴. The Type-I and -II receptors found in the outer capsule layer have been shown to play a role in reflexogenic effects on muscle tone^{6,25}.

Herzog et al²⁵ demonstrated a consistent reflex response on the tone of muscles associated with spinal manipulative treatments. Weakness of a muscle, as depicted by Janda, is due to altered motor regulation from the afferent impulses relayed from tissues surrounding a dysfunctional joint⁷. This "pseudoparesis" is a decrease in strength, which occurs when the CNS regulation limits full firing of a muscle²⁶. Improvements in strength may be regained through mobilization of restricted joints, thus removing inhibitory reflexes⁴.

Studies^{27,28} have examined the effects of joint pathology on muscle strength. Mobilizations on restricted sacroiliac joints (SI) were performed, returning them to proper alignment. As a result of the treatment, both studies found an immediate increase in the strength of muscles responsible for SI joint movement^{27,28}. Removal of the inhibition caused by the AKR in these cases may have resulted in the increases seen.

The purpose of the present study was to assess lower trapezius muscle strength in normal subjects before and after the application of P-A mobilization on thoracic spine levels T6-T12. We hypothesized that a "normal" degree of hypomobility of thoracic vertebrae 6 through 12 is responsible for the neuromuscular inhibition exerted on the lower trapezius. Increasing thoracic extension is expected to remove this inhibition, thereby increasing lower trapezius strength.

Methods

Subjects

A convenience sample of 40 asymptomatic students from New York Institute of Technology between 20 and 45 years of age volunteered to take part in this study. All volunteers were accepted unless they met one of two-exclusion criteria governing selection. The first exclusion criterion eliminated subjects who presented with a flattened thoracic spine as observed by the clinicians; this was determined by the examining clinicians using a postural grid (Figure 1)²⁹. The second exclusion criterion eliminated subjects who currently had shoulder pathology, causing pain and/or limited movement.

Approval for this research was granted by the Institutional Review Board at the New York Institute of Technology in Old Westbury, New York. Informed consent was obtained.

Procedure

Subjects underwent a standardized interview, a musculoskeletal assessment of the upper quarter, and a

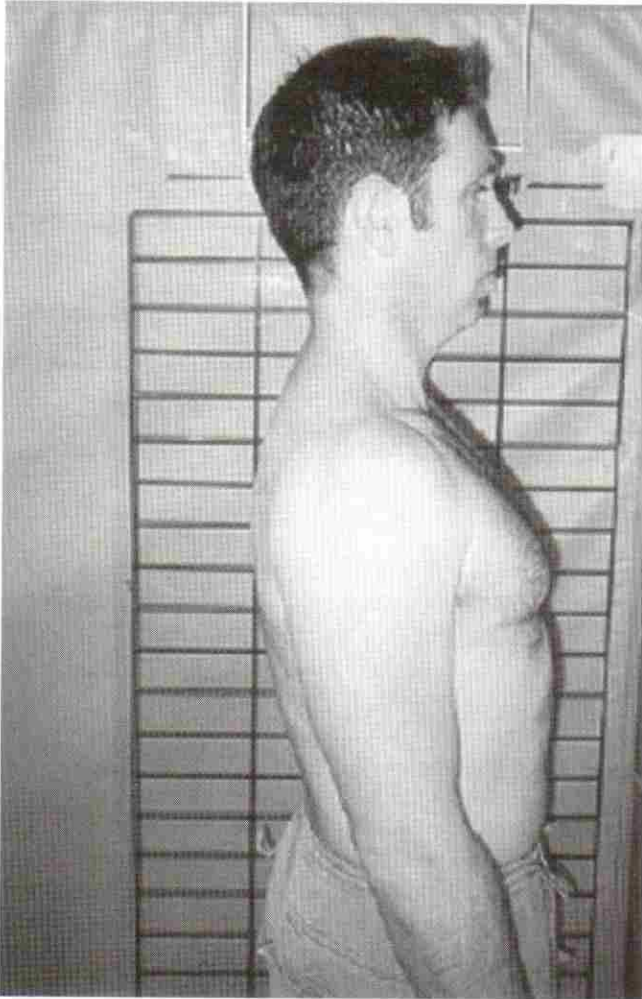


Fig. 1: Postural grid

baseline strength measurement of the lower trapezius bilaterally. This study used double-blind techniques for all measurement procedures³⁰. The strength of the lower trapezius was measured by testing the subject's ability to depress/adduct the scapulae in a position of bilateral shoulder elevation. With the subject in supine, the shoulders were guided to a position of 150° of abduction, bilaterally (Figure 2). This setup placed the lower trapezius muscle fibers in line with the test movement; this ensured that thoracic spine extension was maintained at all times. Isometric torque in shoulder elevation, producing scapular depression/adduction, was measured at a position close to the end range of both upper extremities for three trials. The highest value of the three contractions was accepted as the peak torque measurement.

The baseline strength and subsequent final strength were measured using a handheld Nicholas Manual Muscle Tester (Lafayette Instrument Company, Lafayette, IN). It was stabilized to an inanimate fixed support structure with an adjustable base. Intrarater reliability for this muscle

test was established during pre-testing procedures, assessing the reproducibility of the technique. Relative to the support table, the device's distance from the subject was recorded for use again during the final testing of each individual. This ensured replication of proper body and upper extremity position during the final strength test.

The Nicholas Manual Muscle Tester, reported to have $\pm 0.5\%$ of full scale by the manufacturer, was used for its high accuracy. Magnusson et al³¹ have shown that the Nicholas dynamometer has excellent interday and intraday reliability. This dynamometer has also shown valid and highly reliable results between trials and days.³²

The start position used for muscle testing involved the subject lying supine on a plinth with arms adducted, with hips and knees supported and flexed to 90°. This precise position of the subject's spine was necessary for accurate results, as we needed to measure the strength of the lower trapezius following similar arm motion as standard tests require³³, while ensuring thoracic extension. Flexing and supporting the lower extremities reduced any contributions from the lower body, neutralizing the pelvis and minimizing lumbar lordosis. The operator passively moved the subject's arms into full flexion and 150° of abduction while keeping the elbows straight. The dynamometer was placed just proximal to the wrist joint line for all subjects, while the forearm was in pronation with the palm facing the ceiling. Adjustments of the support structure were done accordingly, and the subject was returned to the start position as described above. The subject was then instructed to reach overhead and push solidly into the manual muscle tester (Figure 2).

After an individual's baseline measurement was completed, the subject was randomly placed into the experimental or control group using a table of random numbers³⁰. All subjects were placed prone on a plinth promoting thoracic extension. Mobilizations were performed on the thoracic spinous processes of vertebrae T6-T12



Fig. 2: Test position for MMT

(Figure 3). The experimental group received a Grade-IV P-A mobilization. The control group underwent a Grade-I mobilization, as this does not promote tissue deformation but rather only decreases pain in acute stages of inflammation^{1, 10, 14}. Two different operators performed the mobilizations, one for the Grade-I and one for the Grade-IV. The area just distal to the pisiform bone was used to direct the spinous process in a P-A direction^{4, 10}. Each of the seven segments was mobilized for 30 seconds totaling 3.5 minutes. Immediately after the mobilization, all subjects were repositioned for muscle testing. Final testing followed the same previously described procedure for baseline measurements. These final measures of strength were recorded and analyzed. Care was taken to ensure blinding of the subjects and of the operator performing muscle testing.

Data and Statistical Analysis

The independent variable was the Grade-IV P-A mobilization; the dependent variable was lower trapezius strength. Parametric interval data were recorded and used throughout the analysis. Strength measurements were recorded as the peak of the three trials taken bilaterally. A randomized, placebo-control, double-blind study design was used to compare differences in strength between the experimental and control groups³⁰.

An independent group t-test compared the mean change between the pretest and posttest scores between



Fig. 3: Mobilization on thoracic spinous processes of T6-T12

groups. The analysis of the data was tested at the $P < .05$ level for an increase in lower trapezius strength in the experimental group.

Results

Pretest and posttest measurements of muscle strength testing were assessed for both the control and the experimental groups. The peak strength for pretest and posttest measurements is shown in Table 1.

To compare the experimental and control group, the

Table 1. Peak strength for pretest and posttest measurements.

EXPERIMENTAL		
Subject	Pretest MMT (kg)	Posttest MMT (kg)
5	17.3	17.8
6	13.2	14.7
7	15.7	16.4
9	10.7	12.7
10	7.8	7.2
12	14.4	14.2
13	8.2	10.6
14	7.2	7.7
15	13.6	13.8
20	6.1	5.8
21	10.8	9.2
22	14.9	17.4
23	3.8	3.7
25	7.5	7.8
30	10.1	11.7
31	14.2	14.6
32	6.5	7.2
33	19.2	19.5
34	9.3	11.6
35	8.0	8.0
Mean 10.93		Mean 11.58
S.D. 4.18		S.D. 4.43

CONTROL		
Subject	Pretest MMT (kg)	Posttest MMT (kg)
1	10.2	11.5
2	11.7	9.0
3	9.8	9.5
4	12.0	12.7
8	17.4	20.6
11	11.3	11.1
16	8.5	9.2
17	11.0	11.0
18	12.0	11.9
19	14.1	14.9
24	8.9	8.7
26	7.4	7.1
27	15.3	15.4
28	9.5	9.4
29	7.3	7.1
36	9.2	9.9
37	12.8	14.1
38	11.6	10.1
39	7.7	6.8
40	12.0	10.1
Mean 10.99		Mean 11.01
S.D. 2.64		S.D. 3.31

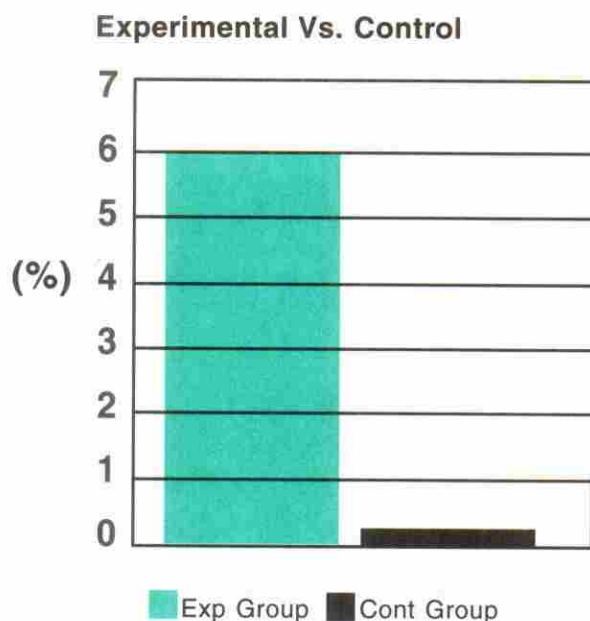


Fig. 4: Average strength change following mobilization

mean difference between the two groups was analyzed. The results demonstrated statistical significance between the experimental and control groups ($t=-1.71$, $P=0.047$). Clinically, these results yielded a 6.0% increase in strength testing scores for the experimental group while the control group showed an increase of 0.2% (Figure 4).

Discussion

These results indicate that the lower trapezius muscle tests stronger after applying Grade-IV mobilizations to the lower thoracic spine. According to the results, immediate increases in strength can be achieved by stretching restricted joint capsules. This finding is significant because clinicians can reverse muscle inhibition through joint mobilizations when weakness is secondary to hypomobility⁴. A 6.0% overall change was observed after only 3.5 minutes of spinal mobilization.

The increase in strength was likely the result of increases in the extensibility of the connective tissues stretched during the experiment, with all other variables controlled to the best of the examiners' ability. However, certain variables were difficult to control, thereby affecting the results. The subject may have learned during the exam to position his/her extremity for a more efficient effort. Human error may have contributed to variability in the data during the pretest and posttest procedures.

Another factor to be accounted for is arm height. This was our most controlled factor, always kept on a stationary platform four inches vertical from the resting surface of the supine subject (Figure 2). However, as explained earlier, the best results were found at the end

range of the subjects' movement to ensure lower thoracic extension. Therefore, it is critical to measure, record, and test at the end range of shoulder elevation at 150° of abduction. The support structure could be improved with a variable height adjustment.

Some subjects may have lacked motivation and may not have pushed to the maximum during the three strength trials, as this study required. A larger sample size might eliminate this problem in a future study. Future studies could also attempt to validate the time frame of this effect and to what degree the time frame is clinically significant. Final strength testing in this study was performed immediately after the mobilization.

The authors performed their testing on the lower trapezius because the lower thoracic spine is often stiff in much of the population. As opposed to other muscles, this muscle is difficult to test for strength because it does not function directly on a major joint. In the future, testers may look at the hip, knee, elbow, or ankle since these joints can easily be stabilized and muscle-tested with conventional devices. In addition, these joints can be mobilized without affecting surrounding muscle tissue.

Lastly, it must be appreciated that the subjects in this study were selected from a normal, asymptomatic population. Future studies may include actual patients with back pain and/or impairment in order to assess the usefulness of this approach in a clinical setting.

Conclusion

This study demonstrated a significant increase in lower trapezius strength testing in response to Grade-IV mobilizations performed on asymptomatic thoracic vertebrae T6-T12. It is theorized that increasing the extensibility of the joint tissues in this region causes an immediate decrease in mechanoreceptor-associated inhibition of the lower trapezius muscles. These findings warrant further research and testing, specifically where mobilization can be used to increase muscle strength about a restricted joint. Clinically, these findings indicate the need for therapists to assess patients' weaknesses and correlate them with possible joint restrictions. Clinicians can use these findings in everyday practice to improve treatment plans by incorporating mobilization with therapeutic exercise.

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REFERENCES

1. Kisner C, Colby LA. *Therapeutic Exercise, Foundations and Techniques*. 3rd ed. Philadelphia, PA: F.A. Davis Company, 1990.
2. Kendall F, McCreary E, Provance PG. *Muscles: Testing and Function*. 4th ed. Baltimore, MD: Williams and Wilkins, 1993.
3. Hurley H, Young A, Stokes M, Iles JF. Effect of joint pathology on muscle. *Clin Ortho* 1987;6:21-27.
4. Warmerdam A. *Manual Therapy: Improve Muscle and Joint Functioning*. Wantagh, NY: Pine Publications, 1999:32-44.
5. Freeman MA, Wyke B. The innervation of the knee joint. An anatomical and histological study in the cat. *J Anat* 1967;101:505-532.
6. Wyke B. Morphological and functional features of the innervation of the costovertebral joints. *Folia Morphologica* 1975;4(23):296-305.
7. Janda V. Muscles, central nervous motor regulation and back problems. In Korr IM, ed. *The Neurologic Mechanisms in Manipulative Therapy*. New York, NY: Plenum Press, 1978:27-41.
8. Petty NJ, Moore AP. *Neuromusculoskeletal Examination and Assessment: A Handbook for Therapists*. Edinburgh: Churchill Livingstone, 1998:33-36.
9. Norkin CC, Levangie PK. *Joint Structure and Function: A Comprehensive Analysis*. 2nd ed. Philadelphia, PA: F.A. Davis Company, 1992:63,142.
10. Saunders DH, Saunders R. *Evaluation, Treatment and Prevention of Musculoskeletal Disorders*. Volume 1; Spine. 3rd ed. Chaska, MN: The Saunders Group, 1993:258-268.
11. Difabio RP. Efficacy of manual therapy. *Phys Ther* 1992;72:853-864.
12. Ottenbacher K, Difabio RP. Efficacy of spinal manipulation/mobilization therapy: A meta-analysis. *Spine* 1985 November;10(9):833-837.
13. Crawford JP. Chiropractic intervention in treatment of the joint and soft tissue disorders. *Canadian J Appl Physiol* 1999;24 (3):279-289.
14. Tomberlin JP, Saunders HD. *Evaluation, Treatment and Prevention of Musculoskeletal Disorders*. Volume 2; Extremities. 3rd ed. Chaska, MN: The Saunders Group, 1995.
15. Schafer SS, Dadfar F, Hartel J, Haupts S, Fischer M. The period of latency before a muscle receptor generates an action potential as a response to a muscle stretch. *Brain Res* 1999;843:36-47.
16. McLain RF. Mechanoreceptor endings in the human cervical facet joints. *Iowa Ortho J* 1993;13:149-154.
17. Guanche CA, Noble J, Solomonow M, Carole SW. Periarticular neural elements in the shoulder joint. *Orthopedics* 1996;22(6):615-627.
18. Zimny ML. Mechanoreceptors in articular tissues. *Am J Anat* 1988;182(1):16-32.
19. Clark FJ, Burgess PR. Slowly adapting receptors in the cat knee joint: Can they signal joint angle? *J Neurophysiology* 1975;38:1448-1463.
20. Giles LGF, Taylor JR. Innervation of lumbar zygapophyseal joint synovial folds. *Acta Orthop Scand* 1987;58:43-46.
21. McLain RF, Picker JG. Mechanoreceptor endings in human thoracic and lumbar facet joints. *Spine* 1998;23(2):163-173.
22. Ashton IK, Ashton BA, Gibson SJ, et al. Morphological basis for back pain: The demonstration of nerve fibers and neuropeptides in the lumbar facet joint capsule but not in the ligamentum flavum. *J Ortho Res*;10(1):72-78.
23. Vandenabeele F, et al. Encapsulated ruffini-like endings in human lumbar facet joints. *J Anat* 1997;191:571-583.
24. Bogduk N. The innervation of lumbar spine. *Spine* 1983;8:286-293.
25. Herzog W, Scheele D, Conway PJ. Electromyographic responses of back and limb muscles associated with spinal manipulative therapy. *Spine* 1999;24(2):146-152.
26. Janda V. Muscle weakness and inhibition (pseudoparesis) in back pain syndromes. In: Greive GP, ed. *Modern Manual Therapy of the Vertebral Column*. New York, NY: Churchill Livingstone, 1986:197-201.
27. Suter E, McMorland G, Herzog W, Bray R. Decrease in quadriceps inhibition after sacroiliac joint manipulation in patients with anterior knee pain. *J Manipulative Physiol Ther* 1999;22(3):149-153.
28. Cibulka MT, Rose SJ, Delitto A, Sinacore DR. Hamstring muscle strain treated by mobilizing the sacroiliac joint. *Phys Ther* 1986;66 (8):1220-1233.
29. Kendall HO. *Muscle Testing and Function*. 2nd ed. Baltimore, MD: Williams & Wilkins Co., 1971.
30. Portney LG, Watkins MP. *Foundations of Clinical Research; Applications to Practice*. Stamford, CT: Appleton & Lange, 1993.
31. Magnusson SP, Gleim GW, Nicholas JA. Subject variability of shoulder abduction strength testing. *Am J Sports Med* 1990;18(4):349-352.
32. Trudelle-Jackson E, Jackson AW, Frankowski CM, et al. Interdevice reliability and validity assessment of the Nicolas hand-held dynamometer. *J Ortho Sports Phys Ther* 1994;20(6):302-316.
33. Hislop HJ, Montgomery J. *Daniel's and Worthingham's Muscle Testing*. 6th ed. Philadelphia, PA: W.B. Saunders Company. 1995:73-75.