

Reliability and Validity of Therapy Localization as Determined from Multiple Examiners and Instrumentation

Anthony L. Rosner^{1,*}, Gerry Leisman^{2,3},
James Gilchrist^{4,5}, Eugene Charles⁶,
Matthew G. Keschner⁶, and
Michael Minond⁷

¹International College of Applied Kinesiology,
USA

²The National Institute for Brain and Rehabilitation
Sciences, Nazareth, Israel

³Universidad de Ciencias Médicas de la Habana,
Facultad Manuel Fajardo, Cuba

⁴The Institute for Brain and Rehabilitation
Sciences, Gilbert, AZ, USA

⁵School of Health Sciences, Touro College,
Bay Shore, NY, USA

⁶Private Practice, New York, NY, USA

⁷Private Practice, Kew Gardens, NY, USA

Abstract

Objective: Therapy localization demonstrates a change in muscle function when a patient's hand is placed over an area of suspected involvement. In manual muscle testing, is there agreement between examiners (reliability) and is there instrumental confirmation (validity) of their muscle classifications produced by therapy localization? **Methods:** Three diplomates in applied kinesiology performed manual muscle tests of the middle deltoid of 30 volunteers with or without neck pain to assess the degree of examiner concurrence and documentation by surface electromyography. An additional 5 patients with neck pain were tested by the same protocol by 2 diplomates to assess forces applied with a clench transducer, degree of arm movement with an electrogoniometer, and vibration of the middle deltoid by vibromyography. **Results:** Statistical tests revealed no significant differences among examiners in their muscle classifications or in the forces applied during muscle testing. Clear distinctions between weak and strong muscle classifications both in the absence or presence of therapy localization were shown by both electrogoniometry and vibromyography. The presence of neck pain was associated with a significantly greater presence of therapy localization. **Conclusions:** In manual muscle tests of the middle deltoid performed by applied kinesiologists, both the reliability and validity were supported by concurrence among examiners and correlations with the results shown with clench transducers, electrogoniometry, and vibromyography.

Keywords: applied kinesiology, manual muscle testing, therapy localization; reliability, validity, electrogoniometry, vibromyography

* Correspondence: Dr. Anthony Rosner, 156 School Street,
Watertown, MA, 02472-4149 USA Email:
arosner66@aol.com

Introduction

A key component of applied kinesiology emerged when Goodheart observed that the results of manual muscle tests (MMT) changed when a patient touched an area of dysfunction, which became known as *therapy localization (TL)*. Specifically, a muscle that previously tested “weak” would become “strong” under those circumstances, [1] pointing to dysfunctions involving any of the following: Reflexes, subluxations, soft tissue injuries, meridian points, and nerve receptors [2]. Similarly, an area touched by the patient that led to the weakening of a muscle that previously tested “strong” suggested a subclinical involvement; i.e., a reflex that is active but not engaged enough to cause a muscle to test “weak” without the TL [2].

In the language of neurophysiology, the essence of TL is that input from low-threshold mechanoreceptors in the skin can modulate ongoing activity in muscles. In other words, stimuli that are applied to different somatic sites can interact in such a manner that one stimulus controls the neural activity recorded at another site. This has been demonstrated in a variety of approaches involving objective measurements in the basic sciences.

In laboratory rats, for instance, colorectal distention produced a visceromotor reflex, as quantified by taking electromyographic (EMG) recordings from the external oblique muscle of the upper abdomen [3]. Elsewhere, it was observed that afferent inputs from the skin and viscera affected both the activity of the bladder and skeletal muscle surrounding the urethra [4].

In cats, there is further evidence supporting the crosstalk and efferent activity in different regions of the body, a central component of TL. Specifically: (1) Microelectrode recordings in the thoracic cord revealed that cells located in the lamina 5 respond to both the fine myelinated afferents from the splanchnic nerve as well as to afferents from the skin, suggesting the convergence of signals; [5] and (2) thermal and mechanical stimulation of the skin at various

segmental levels elicited reflex changes in the heart rate [6].

Further evidence supporting the validity of TL is provided in human studies:

- 1 Strong synaptic coupling exists between the tactile afferents in the sole of the foot and motoneurons supplying muscles that act about the ankle. This was done with microelectrodes which were inserted percutaneously into the tibial nerve of human subjects, in which reflex modulations of whole muscle electromyography (EMG) were observed for each of 4 classes of low-threshold cutaneous mechanoreceptors. Simply stated, this study demonstrates that stimulation of the skin may be responsible for changes in muscle strength, which is the basic tenet of TL. Indeed, the cutaneomuscular reflexes observed may be themselves a part of the mechanism of TL [7].
- 2 A neuroreflexology-based screening test (Medex device) was shown to have a significant degree of correlation with conventional medical evaluation in assessing internal organ pathologies. With 150 patients participating in the study, high sensitivity (>70%) was measured for cardiovascular, respiratory, gastrointestinal and genitourinary diseases. Correlation was significant ($p < 0.01$) for all categories except for blood and lymphatic disease. In other words, electrodermal reflexes of the skin may be indicative of internal organ pathologies—a phenomenon which constitutes a major portion of TL [8].
- 3 EMG recordings in fifteen patients demonstrated that stimulation of the median nerve reduced the size and number of descending corticospinal volleys that were evoked by transcranial magnetic stimulation in relaxed or active muscle. This suggested that mixed or cutaneous input from the hand

can suppress the excitability of the motor cortex at short latency, which may contribute to the initial inhibition of the cutaneomuscular reflex. This may be reflected in changes in muscular strength in MMT, which would be the essence of TL [9].

- 4 In patients with chronic cervical radiculopathy, light pressure in the symptomatic arm is painful and accompanied by a widespread increase in EMG activity. Palpation of adjacent soft tissues is painless and unaccompanied by EMG activity. The light pressure applied is similar to what happens in TL when the patient gently touches an area of suspected injury or dysfunction, producing a change in muscle function that can be useful in diagnosis [10].
- 5 In two separate populations (23 normal [random] and 17 athletic [strong]), a modified shoulder abduction manual muscle test demonstrated strength changes following the tactile stimulation of the skin. Specifically, scratching applied inferior to the clavicle on the clavicular head of the pectoralis major muscle after maximum contraction revealed decreases in isometric strength as quantified by a dynamometer system (Cybex II). The neurophysiologic inhibition of strength following the tactile stimulation of the skin represents the essence of TL [11].

Yet in all these studies, a clear and reproducible physiological confirmation of the clinical effect of TL in human examinations is lacking. A plausible approach to providing such information is to exploit a set of circumstances in human examination that confirms the validity of “strong” and “weak” results in manual muscle testing. This was partially accomplished by Caruso and Leisman [12, 13] who provided evidence that the classifications of muscles as weak and strong as determined by examinations by the applied kinesiologist are both objective

and reproducible with sufficient experience and training. These investigators examined patterns of force, timing, and movement for over 700 muscle tests with specially designed equipment (pressure transducers and electrogoniometers). They used simple mathematical applications to find potential patterns of force and displacement that would correspond to patterns of “weak” muscle tests obtained from healthy volunteers. The result was the creation of a model that was not only able to clearly discriminate between “strong” and “weak” muscles, but also was accurate 98% of the time for applied kinesiology practitioners with 5 or more years of training and experience [12].

Other instrumental evaluations of the muscle testing procedure used in AK recorded somatosensory evoked potentials (SEP) on limbs contralateral to the stimulated side. In all subjects the baseline in which no muscle test was performed and the control (“strong”) muscle test recordings were comparable, while the pattern from the “weak” muscle test displayed increased amplitudes. The suggestion was that a neurologic basis existed for manual muscle testing [14].

For TL itself, however, no such methodologies have been found in the literature. Our approach was to perform TL using the middle deltoid as the test muscle on a series of patients, confirming the results of MMT among independent practitioners and with instrumentation designed to measure forces applied, muscle movement, and a technique known as vibromyography (VMG) capable of non-invasively assessing voluntary muscle effort by extracting specific components of the vibration spectrum that correlates best with muscle activity [15]. Finally, it sought to confirm whether a positive TL test is associated with neck pain, a musculoskeletal disorder with which weak MMT results have been linked [16].

Materials and Methods

The institutional review board for human research (from the Winthrop University Hospital

in Mineola, NY) approved this project, all subjects having provided written consent to participate after being presented with the experimental protocol and offered a modest financial incentive to complete the study. Three clinicians with at least five years of muscle testing experience were recruited from the New York metropolitan area to conduct the investigation in one of the practitioner's private offices. They recruited 30 volunteers from their patient base who experienced neck or shoulder pain for a minimum of two days preceding muscle testing, following the measurement and analytical techniques and using the test muscle as previously described by the author [12]. Patients with neck or shoulder pain were expected to yield the highest percentage of positive TL results, facilitating the completion of this investigation.

The *middle deltoid* muscle was chosen for testing for two reasons: (1) Good inter-examiner reliability of the deltoid muscle has been demonstrated; [17] and (2) with the patient in a

seated position with head and neck kept within a neutral position, TL may be performed with a minimum of contortion and substitution that could independently affect the results of the muscle test.

For determinations with instrumentation, the patient was fitted on an area of skin over the middle deltoid of the right arm with one of the following: (a) a clench force (bulb) transducer (SS56L) to measure forces applied by the clinician, (b) an electrogoniometer (SS21L) and disposable paired cloth electrode (EL500) to determine arm movement, or (c) a vibromyography transducer (TSD250) to measure vibration of the middle deltoid. All were connected to a data acquisition board capable of performing vibromyography (VMG36R2WSW). All instrumentation was provided by BioPac Systems Inc., Santa Barbara, CA as shown in Figure 1. The patient was maintained in a seated position with the head and neck in a neutral position.

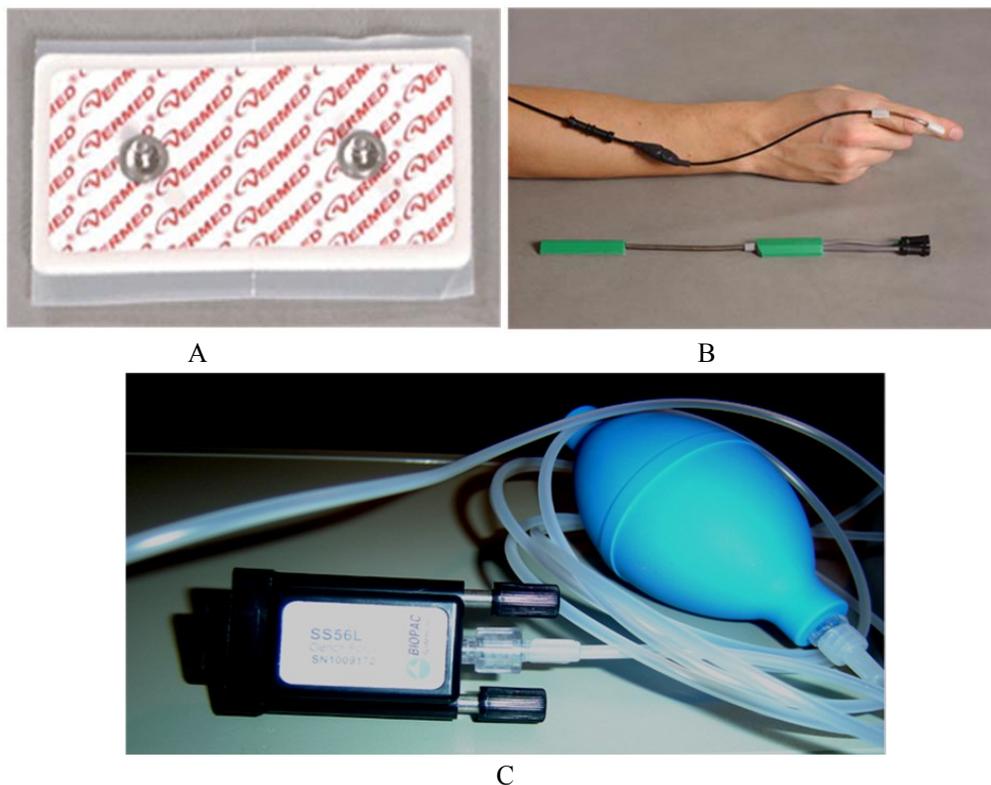


Figure 1. (Continued).



D



E

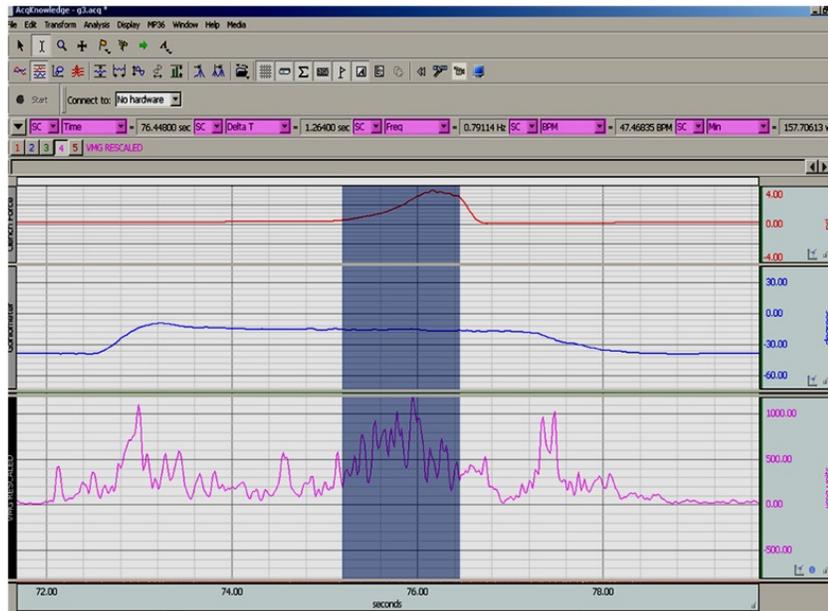
Figure 1. Instrumentation to assess validity of middle deltoid test classification:

- A: Disposable paired foam electrode (BioPac EL500).
- B: Electrogoniometer (BioPac SS21BSL Twin Axis Goniometer, 150 mm).
- C: Clench force (bulb) transducer (BioPac SS56L).
- D: Vibromyography (VMG) transducer (BioPac TSD250).
- E: 2-channel VMG amplifier/integrator (BioPac VMG36R2WSW).

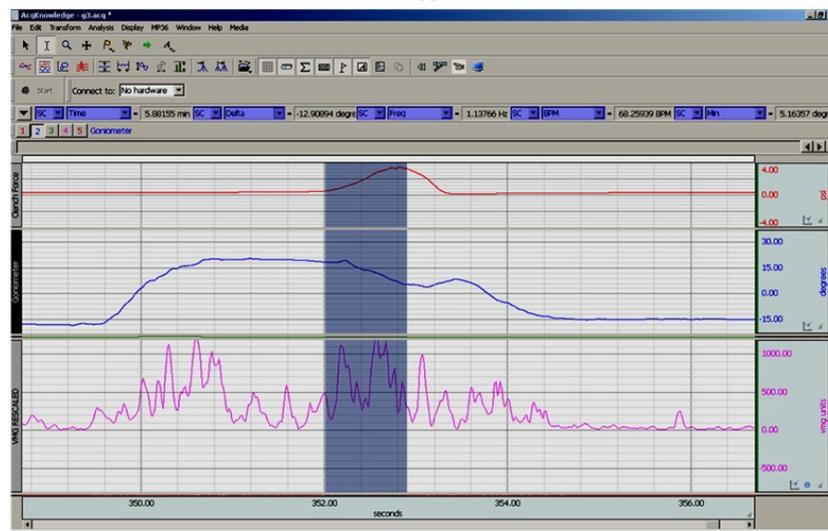
The maximum voluntary contraction of the patient's arm was first computed in order to allow for the normalization of all subsequent tests. The MMT itself was conducted as a submaximal break test, with resistance applied by the patient to increasing test pressure by the examiner over a 1-3 second period. The test was stopped when a "lock" (full resistance) was perceived by the tester, [2, 18-20], with the result (strong or weak) surreptitiously conveyed to the operator of the VMG apparatus for correlation with the instrumental data out of sight of the patient. The MMT was next repeated by having the patient perform TL by touching the myotomes for the middle deltoid muscle at C4 to

T1. Head and neck position of the patient were maintained in the neutral position, and blinding of the patient to the test result was maintained.

After a five-minute recovery period, the same patient was retested by the second of the three examiners to confirm inter-examiner reproducibility by first being tested in the clear (without TL) and then touching T1 to C4 as described above. The entire procedure was ultimately performed as previously described by the third clinician to provide yet further data on inter-examiner reproducibility. The sequence of the three examiners was randomized by the application of a random number generator program [21] to prevent ordering effects.



A



B

Figure 2. A: Strong deltoid muscle response shown by instrumentation. Shaded area denotes area sampled for all measurements. TOP: Force in pressure per square in (psi) for force bulb measurement used by clinicians to push on subject's arm. Rising edge is the applied force, peak force is sustained for a few seconds, while the falling edge to the right is the termination of the force. MIDDLE: Goniometer measurement in degrees of movement. Line drops very little during the application of force, indicating very little arm movement; hence, categorization was "strong."

BOTTOM: VMG in VMG units. Raw waveform was processed through a vibromyograph filter (Biopac VMG36RSWSW). B: Weak deltoid muscle response shown by instrumentation. Shaded area denotes area sampled for all measurements. TOP: Pressure per square in (psi) for force bulb measurement used by clinicians to push on subject's arm. It can be seen that the amplitude of the wave is the same as that seen in the strong muscle response (TOP, Figure 1a). MIDDLE: Goniometer measurement in degrees of movement. Line drops significantly during the application of force by the clinician, indicating that the arm dropped significantly during the applied force; hence, categorization was "weak." BOTTOM: VMG in VMG units. Raw waveform was passed through a Vibromyograph (Biopac VMG36RSWSW). Statistical analysis revealed significant difference from "strong" response.

The time profiles of force and displacement were determined by measuring the displacement of the patient's arm (read by the accelerometer) and the clinician's force (transmitted by the transducer inserted between the clinician's measuring hand and the patient's limb being tested). Analog information from the accelerometer and force transducer were fed by direct coupling into the industry-standard analog VMG data acquisition board.

For each clinician/patient pair, an average threshold between "strong" (conditionally facilitated) and "weak" (conditionally inhibited) was established. To reconfirm the validity of the clinical evaluation of the examiners who were blinded to the recording results, the clinician's assessments of the muscle's condition were recorded in a notebook for each data file for the recorded test. For the binary judgments from the clinicians as to strong and weak responses, all results were subjected to Chi-Square analyses. Where instrumentation was involved for all EMG, force bulb, electrogoniometric, and VMG measurements, the raw data was correlated with the clinicians' strong or weak categorizations of participant responses by noting the time on the waveforms accompanying each muscle test. The particular clinician making the observation was noted as well. Because further instrumentation calibrations had to be made with time limitations on the subjects' availability, a total of 2 clinicians and 5 patients were utilized for the force bulb, electrogoniometric, and VMG measurements.

The bulb force recording measurements were used as the standard against which other waveform measurements were collected. These represented the action of the clinician pressing down (applying force) on the participant's arm either in the absence or presence of therapy localization. The bulb force waveform shown in Figures 2A and 2B were selected from the beginning of trough to end of peak, representing the amount of time that the clinician was actually applying force. The falling edge (post peak) of the bulb force waveform was not included, since it was observed that the clinician had ceased applying force abruptly such that the

force included in this region was negligible. Once the appropriate sections of waveforms were selected, the relevant data (mean, maximum, minimum) were downloaded from the Biopac software to an Excel spreadsheet for future analysis.

For all instrumentation measurements, 1-tailed t-tests were conducted. To further analyze the EMG determinations, tests for variances consisting of a 1-way, 1-factor 8 group F tests were performed.

Results

For the 30 patients tested for strong or weak middle deltoid muscles, there was no significant difference between the three clinicians in their strong or weak assessments of patient responses. This was observed both in the presence or absence of therapy localization across all strong or weak muscle responses (Table 1). Kappa determinations confirmed close inter-observer agreements, in that k values across all clinicians of 0.70 were obtained for all strong vs. weak categorizations. In the absence of TL, the k value of 0.92 was calculated, whereas in the presence of TL, the k value was 0.94. K values of 0.61-0.80 indicate substantial agreement, which becomes almost perfect when k values fall within the range of 0.80-1.00. For all clinicians involving all participants, there was a highly significant distinction in their classifications of strong or weak muscle responses depending upon whether therapy localization was absent or present (Table 2). When it came to distinguishing patients with or without neck injuries, there were significant differences in the number of strong and weak deltoid responses; i.e., there were statistically significant differences in the number of strong responses in the absence of TL in injured vs. non-injured patients, as were the number of weak responses in the presence of TL in injured vs. non-injured patients (Table 3).

EMG analyses of the deltoid responses of the 30 patients were less conclusive. It was apparent that overall EMG determinations

yielded no significant differences between all strong or weak responses, regardless of whether (a) mean amplitude, (b) mean frequency, or (c) power spectrum density measurements were taken (Table 4). The same was true for these three parameters in either the absence or

presence of TL (Table 5). An additional analysis of variance by means of an F-test failed to disclose differences in either the EMG mean frequency or power spectrum density measurements (Table 6).

Table 1. Reliability between Examiners for Strong vs. Weak Muscle Determinations

A. Absence of Therapy Localization (-TL)

	Clinician 1		Clinician 2		Clinician 3		Totals
	Observed	Expected	Observed	Expected	Observed	Expected	
Strong	83	83.3	86	83.3	81	83.3	250
Weak	7	6.7	4	6.7	9	6.7	20
Totals	90		90		90		270

Null: No statistical difference between clinicians #1, 2, and 3 for strong or weak assessment of patient responses in absence of TL.

Results:

1. **df = 2**
2. **Ch Sq observed = 2.574**
3. **Chi Sq critical = 13.82, p < 0.001**

Conclusion: Null is accepted ($2.574 < 13.82$, $p < 0.001$). There is no significant difference between clinicians for strong or weak assessment of patient responses in the absence of therapy localization across all strong and weak muscle responses.

B. Presence of Therapy Localization (+TL)

	Clinician 1		Clinician 2		Clinician 3		Totals
	Observed	Expected	Observed	Expected	Observed	Expected	
Strong	57	53	48	53	54	53	159
Weak	33	37	42	37	36	37	111
Totals	90		90		90		270

Null: No statistical difference between clinicians #1, 2, and 3 for strong or weak assessment of patient responses in presence of TL.

Results:

1. **df = 2**
2. **Ch Sq observed = 2.008**
3. **Chi Sq critical = 13.82, p < 0.001**

Conclusion: Null is accepted ($2.008 < 13.82$, $p < 0.001$). There is no significant difference between clinicians for strong or weak assessment of patient responses in presence of therapy localization across all strong and weak muscle responses.

Table 2. Reliability of Muscle Testing in Absence or Presence of Therapy Localization.

Session Observations Made by All Clinicians for All Participants:

Absence of therapy localization:
Strong - 252
Weak - 18

Presence of therapy localization:
Strong - 159
Weak - 111

	Strong	Weak	
	B1 Observed - Expected	B2 Observed - Expected	TOTAL
A1 -TL	252 - 1233	18 - 387	270
A2 + TL	159 - 1233	111 - 387	270
TOTAL	411	129	540

Null: No statistical difference between the absence or presence of TL for strong or weak observations made by all clinicians across all participants.

Results:

1. $df = (2-1)(2-1) = 1$
2. **Ch Sq observed = 19324.3**
3. **Ch Sq critical = 10.828, $p < 0.001$**

Conclusion: Null is rejected ($19324.3 > 10.828$, $p < 0.001$). There is a highly significant difference between absence and presence of TL for strong or weak observations made by all clinicians across all participants.

Table 3. Analysis of Variance: Inured vs. Non-Injured Patients

A. Injured vs. Non-Injured in the Absence of TL, Strong Observations:

	INJURED B1	NON-INJURED B2	
	Observed (f) – Expected (F)	Observed - Expected	TOTAL n
-TL STRONG A1	151 – 124.5 $(151-124.5)^2/124.5$ 5.64	98 – 124.5 $(98-124.5)^2/124.5$ 5.64	249
TOTAL	151	98	249

Chi Square (χ^2) Test of Distribution

Assumption: There is no difference between injured and non-injured participants for the number of strong observations made under the absence of TL condition $N = 249$:

f = Observed

f = Predicted, no difference = $249/2 = 124.5$

$\chi^2 = \sum [(f - F)^2 / F]$

$\chi^2 = 5.64 + 5.64 = 11.28$

$\chi^2 = 11.28$

Null: There is no statistically significant difference between injured and non-injured participants for the number of strong observations made in the absence of TL.

Results:

1. **Df (2)-1 = 1**
2. **Chi Sq observed = 11.28**
3. **Chi Sq critical = 10.8276 at $p < 0.001$**

Conclusion: Null is rejected ($11.28 > 10.8276$). There is a significant difference between injured and non-injured participants for the number of strong observations made in the absence of TL at $p < 0.001$.

B. Injured vs. Non-Injured in the Presence of TL, Weak Observations:

	INJURED B1	NON-INJURED B2	
	Observed – Expected	Observed - Expected	TOTAL N
+TL WEAK A4	73 -55 5.89	37 -55 5.89	110

$\chi^2 = 5.89 + 5.89 = 11.78$

Null: There is no statistically significant difference between injured and non-injured participants for the number of weak observations made in the presence of TL:

Results:

1. **Df = (2)-1 = 1**
2. **Ch Sq observed = 11.78**
3. **Ch Sq critical = 10.8276 at p<0.001**

Conclusion: Null is rejected ($11.78 > 10.8276$). There is a significant difference between injured and non-injured participants for the number of weak observations made in the presence of TL at $p < 0.001$.

Table 4. Analysis of Variance of EMG Mean Amplitude, Mean Frequency and EMG Power Spectrum Density of All Strong and Weak Responses: t-test

*Comparing all Strong responses to all Weak responses for all clinicians across all subjects for three separate variables; (A) EMG Mean Amplitude, (B) EMG Mean Frequency, and (C) EMG Power Spectrum Density.

A. EMG Mean Amplitude

**EMG Mean Amplitude measurements: trough to trough mean amplitude values (millivolts).

EMG MEAN AMPLITUDE	STRONG	WEAK
MEAN	-1.443	-1.778
ST DEV	5.427	5.874
MEDIAN	-0.451	-0.605

Null: There is no significant difference between all strong and weak responses for EMG mean amplitude for all clinicians across all patients.

Results:

1. **df = 29**
2. **t-test observed = 0.2748**
3. **t-test critical = 1.699 for p<0.05**

Conclusion: The null hypothesis is accepted ($0.2748 < 1.699$, $p < 0.05$). There is no significant difference between strong and weak responses for EMG mean amplitude (mv) for all clinicians across all patients.

B. EMG Mean Frequency

*EMG Mean Frequency measurements: trough to trough mean frequency values (Hz).

EMG MEAN FREQUENCY	STRONG	WEAK
MEAN	0.681	0.599
ST DEV	0.189	0.189
MEDIAN	0.647	0.575
t-test	9.78×10^{-6}	

Null: There is no significant difference between all strong and weak responses for EMG mean frequency measurements (Hz) for all clinicians across all patients.

Results:

1. **df = 29**
2. **t-test observed = 9.78×10^{-5}**
3. **t-test critical = 1.699 for p<0.05**

Conclusion: The null hypothesis is accepted ($9.78 \times 10^{-5} < 1.699$, 0.05). There is no significant difference between strong and weak responses for EMG mean frequency values (Hz) for all clinicians across all patients.

C. EMG Power Spectrum Density

EMG POWER SPECTRUM DENSITY	STRONG	WEAK
MEAN	5.71×10^{-6}	7.23×10^{-6}
ST DEV	6.05×10^{-6}	7.7×10^{-6}
MEDIAN	3.12×10^{-6}	4.49×10^{-6}

Null: There will be no significant difference between all strong and weak responses for EMG mean power spectrum density frequency (mv / Hz/2) for all clinicians across all patients.

Results:

1. **df = 29**
2. **t-test observed = 9.32×10^{-3}**
3. **t-test critical = 1.699 at $p < 0.05$**

Conclusion: The null hypothesis is accepted ($9.32 \times 10^{-3} < 1.699, 0.05$). There is no significant statistical difference between strong and weak responses for EMG power spectrum density values (mv / Hz/2) for all clinicians across all patients.

Table 5. Analysis of Variance of EMG Mean Amplitude, Frequency, and EMG Power Spectrum Density in Absence or Presence of Therapy Localization; t-test

A. EMG Mean - Trough to Trough EMG Mean Amplitude Value (mv)

EMG MEAN AMPLITUDE	-TL	+TL
MEAN	1.607492	1.433338
ST DEV	5.551790	5.568985
MEDIAN	0.733525	0.229725

Null: No significant statistical difference in EMG mean amplitude (mv) between the absence or presence of TL for strong or weak observations made by all clinicians across all subjects.

Results:

1. **df = 538**
2. **t-test observed = 0.358**
3. **t-test critical = 1.647, $p < 0.05$**

Conclusion: Null is accepted ($0.358 < 1.647, p < 0.05$). There is no statistical difference between the absence or presence of TL as measured by EMG mean amplitude (mv) for all clinicians across all subjects.

B. EMG Frequency - Trough to Trough Mean Frequency Value (Hz)

EMG FREQUENCY	-TL	+TL
MEAN	0.700069	0.624840
ST DEV	0.197638	0.179540
MEDIAN	0.651680	0.607715

Null: No statistically significant difference for mean EMG frequency between the absence or presence of therapy localization for all clinicians across all subjects.

Results:

1. **df = 538**
2. **t-test observed = 2.3×10^{-6}**
3. **t-test critical: 1.6449**

Conclusion: Null is accepted ($2.3 \times 10^{-6} < 1.6449, p < 0.05$). There is no statistical difference between the absence or presence of TL as measured by EMG mean frequency for all clinicians across all subjects.

C. EMG Power Spectrum Density (PSD) - Trough to Trough Mean Value (mv/ Hz/2).

EMG PSD	-TL	+TL
MEAN	5.5×10^{-6}	6.7×10^{-6}
ST DEV	5.8×10^{-6}	7.1×10^{-6}
MEDIAN	3.4×10^{-6}	3.1×10^{-6}

Null: No statistically significant difference for mean EMG power spectrum density between the absence or presence of therapy localization for all clinicians across all subjects.

Results:

1. **df = 538**
2. **t-test observed = 2.3×10^{-6}**
3. **t-test critical: 1.6449**

Conclusion: Null is accepted ($2.3 \times 10^{-6} < 1.6449$, $p < 0.05$). There is no statistical difference between the absence or presence of TL as measured by EMG PSD for all clinicians across all subjects.

Table 6. Analysis of Variance of EMG Mean Frequency and EMG Power Spectrum Density in Absence or Presence of Therapy Localization; f-test

The following F test compares variances between the following 8 groups for significant differences in EMG frequency (Hz).

All clinicians grouped together:

- 3 Clinicians
- 3 Subject responses per clinician for No treatment session
- 3 Subject responses per clinician for Treatment session
- 30 Subjects (18 Injured, 12 Non-Injured)
- Grand Total of Subject Responses = 540

Injured Group	-TL Strong Responses	-TL Weak Responses	+TL Strong Responses	+TL Weak Responses
n = 18	151	7	93	73
Non-Injured Group				
N = 12	98	11	70	37

A. EMG Frequency - Trough to Trough Mean Frequency Value (Hz).

F Analysis Table

Source	Sum of Squares	Df	Mean Square	F
Between Group	2909	7	415.6	0.079
Within Group	2806867	532	5276.1	
Total	2809776	539		

Null: There are no significant differences between all groups as measured by EMG frequency for all clinicians across all subjects.

Results:

1. **df = 7,532**
2. **F observed = 0.079**
3. **F critical = 2.02 at $p < 0.05$**

Conclusion: Null hypothesis is accepted ($0.079 < 2.02$, $p < 0.05$). There are no significant differences between all groups. There are no significant differences between all groups for EMG frequency values.

B. EMG Power Spectrum Density (PSD) - Trough to Trough Mean Value (mv Hz/2).

Source	Sum of Squares	Df	Mean Square	F
Between Group	55027710.15	7	7861101	0.0387
Within Group	108018000000	532	203041353	
Total	2809776	539		

Null: There are no significant differences between all groups as measured by EMG frequency for all clinicians across all subjects.

Results:

1. **df = 7,532**
2. **F observed = 0.079**
3. **F critical = 2.02 at p<0.05**

Conclusion: Null hypothesis is accepted ($0.0387 < 2.02$, $p < 0.05$). There are no significant differences between all groups. There are no significant differences between all groups for EMG PSD values.

Table 7. Determination of Validity of Strong vs. Weak by Electrogoniometry

Number of Strong Responses = 45	Average degree of movement = 0.56°
Number of Weak Responses = 14	Average degree of movement = 12.93°

Null: There will be no statistical significant difference between angular movement (degrees) during strong and weak participant responses by all participants across all clinicians as measured with a Biopac Digital Goniometer.

Results: A t test was performed between all strong and weak measurements.

1. **df = 57**
2. **t-test calculated = 10.87**
3. **t-test critical = 3.232 at p<0.001.**

Conclusion: Null is rejected ($10.87 > 3.55$). There is a significant statistical difference between all strong and weak participant responses as measured by angular movement in degrees by a digital goniometer for all participants across all clinicians at $p < 0.001$.

Further instrumental refinements and calibrations permitted the performance of additional quantitative outcomes, although time and budgetary constraints limited such measurements to only 5 additional patients. Nevertheless, striking distinctions could be made. The application of electrogoniometry with these participants, designed to demonstrate the

angular movement of the arm in degrees, displayed a highly significant difference between all strong and weak deltoid responses across all clinicians (Table 7, Figure 3). Similar statistically significant distinctions between strong and weak responses by electrogoniometry were apparent in the absence or presence of TL (Table 8).

Table 8. Determination of Validity of Therapy Localization by Electrogoniometry

* Frequency of Strong and Weak responses by absence or presence of TL categories:

-TL+TL
 Strong = 30
 Strong = 15
 Weak = 0
 Weak = 15

DEGREES	-TL	+TL
MEAN	-0.5658	5.7971
ST DEV	2.5758	8.5089

Null: There will be no significant difference between the absence and presence of TL as measured by the goniometer in degrees of movement for all participants across both clinicians.

Results:

1. **df = 58**
2. **t-test observed = 3.9202**
3. **t-test critical = 3.232 at $p < 0.001$**
4. **Confidence interval = -1.1161 to 13.8419**

Conclusion: Null is rejected ($3.9202 > 3.232$, $p < 0.001$). There is a statistically significant difference between the absence and presence of TL as measured by the goniometer for all participants across both clinicians at $p < 0.001$

Vibromyography for these patients yielded concurrent contrasts between strong and weak deltoid responses. For all strong vs. weak determinations, VMG displayed a highly significant difference between strong or weak classifications (Table 9, Figure 3). For distinguishing the presence or absence of TL, the degree of confidence in the VMG measurements was 95% (Table 10).

To confirm that the forces applied by clinicians were the same in all strong or weak deltoid determinations, a clench (bulb) force transducer was able to demonstrate that the force applied was virtually identical in either all strong vs. weak deltoid tests (Table 11, Figure 4) or specifically in the absence or presence of TL (Table 12).

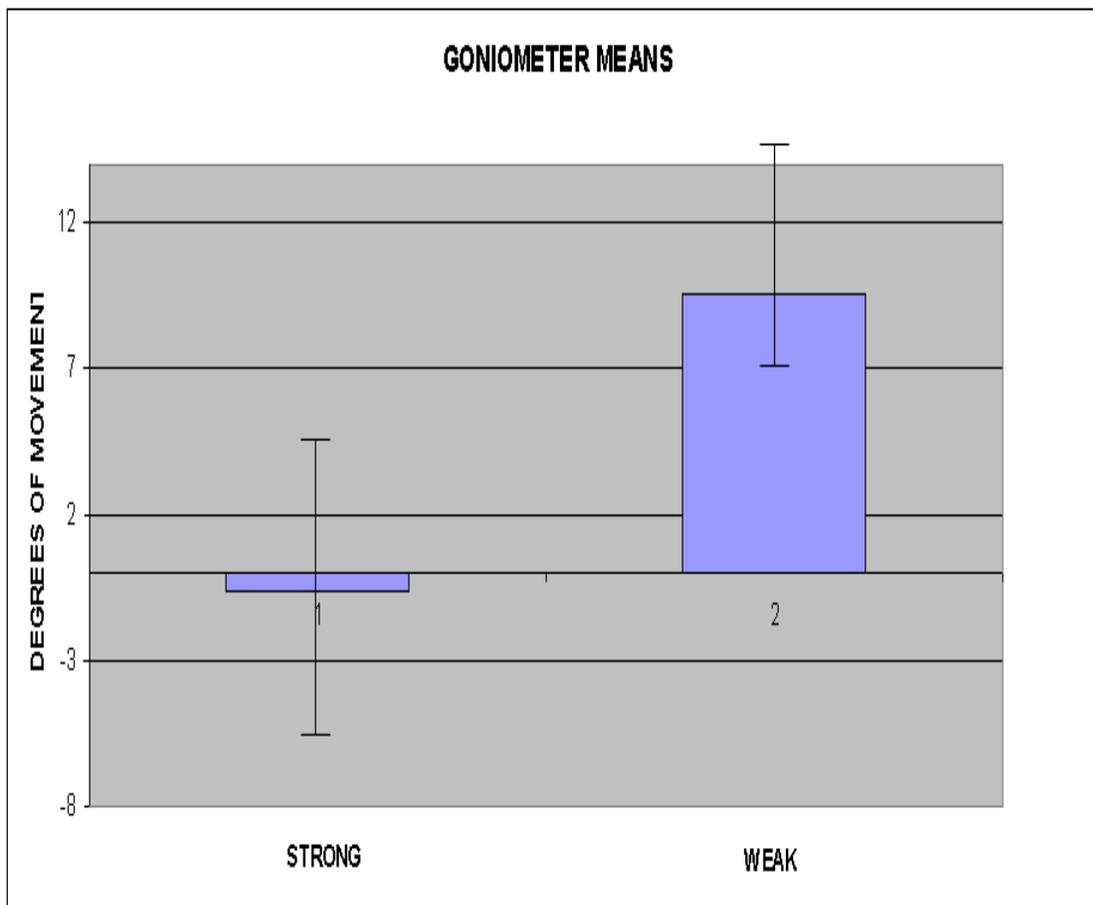


Figure 3. Determination of validity of strong vs. weak muscle response by electrogoniometry.

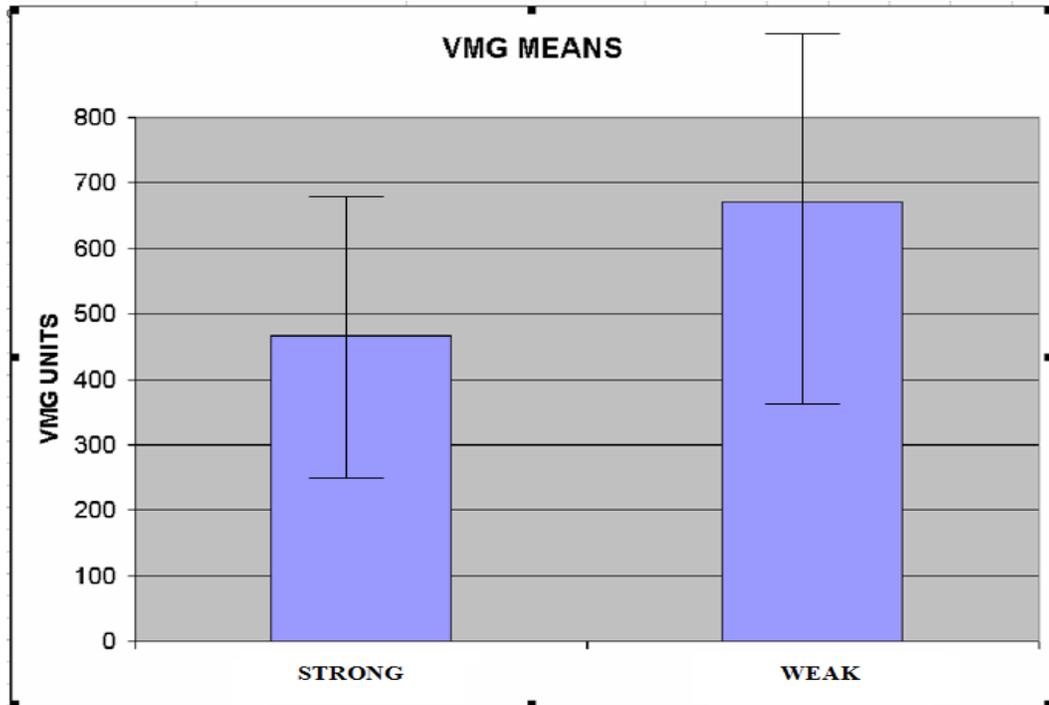


Figure 4. Determination of validity of strong vs. weak muscle response by VMG.

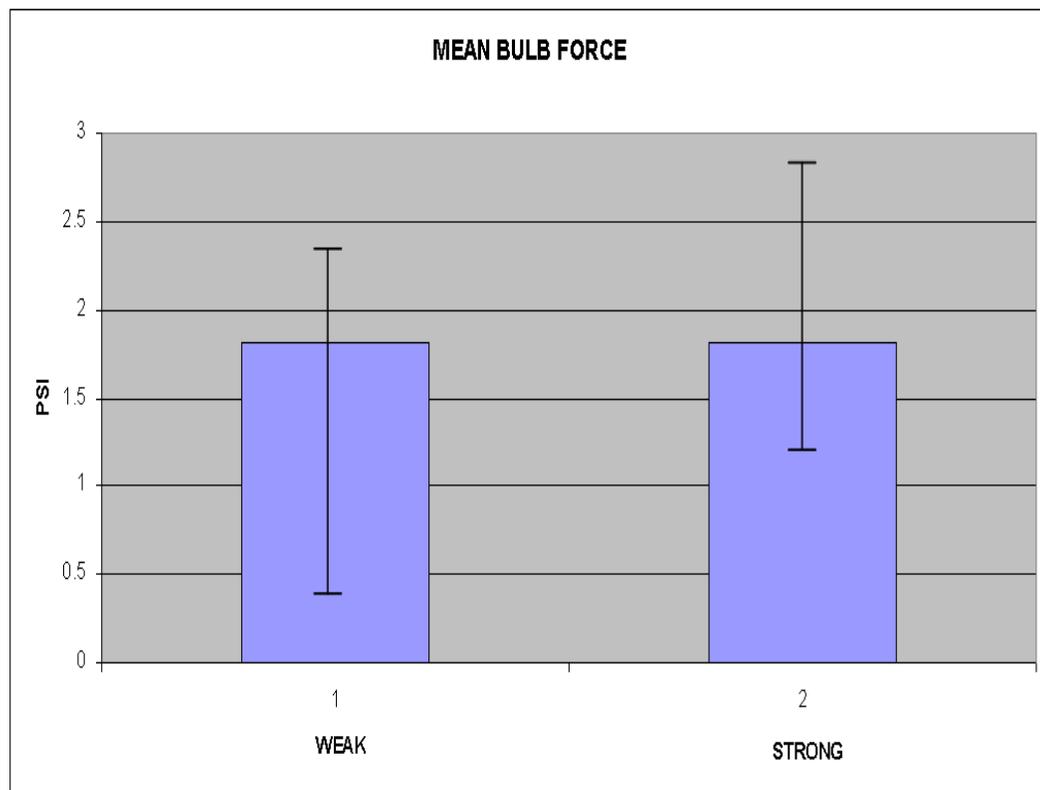


Figure 5. Mean bulb force determinations when eliciting a strong or weak muscle response.

Table 9. Determination of Validity of Strong vs. Weak Muscle Determinations by VMG

	STRONG	WEAK
MEAN	466.31	671.3
ST DEV	129.8	200.4

Null: There will be no statistical significant difference between VMG measurements, units are "muscle activity"- muscle vibration measured as a frequency, for Strong and Weak clinician observations for all participant responses across all clinicians as measured by VMG.

Results: A dependent t test was performed comparing 14 Weak VMG means values to the first 14 Strong VMG mean values.

1. **df = 12**
2. **t calculated = 3.728**
3. **t critical = 3.428 at p<0.0025**

Conclusion: Null is rejected. There is a significant statistical difference between all Strong and Weak participant responses as measured by VMG, ($3.728 > 3.428$, **p < 0.0025, one tail**). The VMG application displays a significant difference between strong or weak participants' responses. The muscle vibrations during strong and weak clinician responses are significantly different.

Table 10. Determination of Validity of Therapy Localization by VMG

	-TL	+TL
MEAN	561.5619	715.9549
ST DEV	219.4901	316.3563

Null- there will be no significant difference between the absence and presence of TL as measured by VMG measurements for all participants across both clinicians

Results:

1. **df = 46**
2. **t-test observed = -1.9644**
3. **t-test critical = 1.68 at p<0.05**
4. **Confidence interval = -139.0752 to 447.8612**

Conclusion: Null is rejected ($1.9644 > 1.68$). There is a significant difference between the absence and presence of TL as measured by VMG at $p < 0.05$.

Table 11. Mean Bulb Force Determinations for Strong and Weak Muscle Responses

PSI	Strong	Weak
MEAN	1.818	1.814
ST DEV	0.342	0.538

Null: there will be no significant difference between strong and weak muscle responses as measured by the amount of force (psi) applied to all participants by all clinicians.

Results:

1. **d1df = 57**
2. **t-test observed = 0.031**
3. **t-test critical = 3.245 at p<0.001, p < 0.001**

Conclusion: Null is accepted ($0.031 < 3.245$, $p < 0.001$). There is no significant difference between strong and weak muscle responses as measured by the amount of force (psi) applied to all participants by all clinicians

Table 12. Force Determinations in Presence and Absence of Therapy Localization

PSI	-TL	+TL
MEAN	1.7554	1.8816
ST DEV	0.2608	0.4812

Null: there will be no significant difference between the absence and presence of TL conditions as measured by the amount of force (psi) applied to all participants by all clinicians.

Results:

1. **df = 58**
2. **t-test observed = 1.263.**
3. **t-test critical = 1.673 at $p < 0.05$**

Conclusion: Null is accepted ($1.263 < 1.673$, $p < 0.05$). There is no significant difference between the absence and presence of TL conditions as measured by the amount of force (psi) applied to all participants by all clinicians

Discussion

In theory, the essence of TL is that input from low-threshold mechanoreceptors in the skin can modulate ongoing activity in muscles. Specifically, stimuli applied to different somatic sites may be capable of interacting in such a fashion that one stimulus controls the neural activity recorded at another site. The rigor of this protocol, requiring in every MMT (a) the same starting point, direction, and magnitude of force, (b) the application of force at a constant rate of speed, (c) the same point of contact on the patient, (d) the same point of contact on the examiner, (e) the same position of the examiner's elbow, arm and forearm, (f) the same plane of the examiner's shoulders, and (g) the same position of the examiner's body has been described in further detail elsewhere [22, 23]. Its observance renders the MMT employed the most likely to avoid many of the pitfalls and criticisms of AK raised elsewhere [22].

In addition to possessing the PAK background, our practitioners each had at least 7 years of clinical practice. It has been demonstrated elsewhere that practitioners with at least 5 years of clinical experience demonstrated 98% agreement in their ability to distinguish strong and weak responses of the pectoralis major in over 750 trials [12].

The high level of training and experience of our PAK practitioners were perhaps key factors which allowed us to demonstrate that the amounts of applied force across all practitioners for either strong or weak muscle responses were virtually identical as shown in Figure 4 and Tables 11 and 12, making the hypothesis that a weak muscle test was the result of a lower force

applied by the clinician unlikely. An additional element supporting the reliability of the deltoid muscle test in these patients was both the Chi-Square (Tables 1 and 2) and kappa statistical results demonstrating a high level of agreement between examiners under all conditions—either in the absence or presence of TL.

Adding confirmatory data from instrumentation supports the validity as well as the reliability of TL in those circumstances involving MMT of the middle deltoid muscle. The magnitude of the ability of both electrogoniometry (Figure 3, Tables 7 and 8) and VMG (Figure 4, Tables 9 and 10) to distinguish strong and weak muscle responses overall and in the absence and presence of TL was so great that statistically significant differences emerged with patient samples numbering just 5.

Goniometry, by measuring degrees of arm movement, serves as an objective indicator of the ability of the test muscle to “lock” in a typical strong response as classified by AK or to “break away” in what has been considered to be a weak response. The very basis of MMT is found in the procedures and principles of Kendall and Kendall, who over half a century ago established that a given muscle when tested from a contracted position against increasing applied pressure from the examiner either maintained its position or broke away.²⁴ Those muscles which maintained their position were graded as facilitated, or “strong,” while those which gave way were classified as inhibited, or “weak” [25]. Muscle strength *per se* is not regarded to be a significant issue with clinical syndromes in PAK, nor is it considered to be a factor in back pain or the onset of chronicity [26-30]. Instead, “facilitated” or “overactive”

refers to the neurological state of the muscle [31, 32].

This reasoning led us to probe deeper into the nature of the deltoid muscle during MMT using surface electromyography (sEMG) and vibromyography (VMG). sEMG has been commonly applied to muscle activity to study fatiguing; [33-38] occurrences of low back, [39,40] shoulder, [41] and general chronic pain, [42] temporomandibular disorders, [43] orthopedic problems, [44] spinal cord injury, [45] osteoarthritis, [46] chronic obstructive pulmonary disease, [47] cerebral palsy, [48] polio, [33] and sacropenia; [49] performance in sports-related activities; [50-54] and methodological issues commonly involving isometric and isokinetic muscle testing lacking the changing pressure and timing of the test that is intrinsic to AK [55-61]. Other instrumental studies have involved the evaluation of the steadiness [62] and loads [63] of the quadriceps muscle under varying conditions.

Previous applications of sEMG directly to AK, on the other hand, have been scanty. Distinctions of examiner-started and patient-started MMT of the middle deltoid by using sEMG to mark the start times were conducted by Conable et. al, yet there was no indication that sEMG was used to distinguish weak from strong patient muscle responses [19]. In extending AK to acupuncture meridians, Moncayo et al. observed that graded sedation produced a graded diminution of signal amplitude, while the opposite effect resulted when antique acupuncture points were used for tonification. [64].

The distinctions sought in weak and strong muscle responses in PAK are of a different nature than all those reported previously. Here it is a matter of timing in subjecting the muscle to a test, within a response time on the order of 1 sec. This does not determine frank muscle strength or endurance, the measurements of which can take up to 30 sec. The nearly instantaneous periods in PAK may have been too limited for the dimensions of sEMG to detect differences between strong and weak responses. Furthermore, the weak response entails

movement of the middle deltoid and arm, which could have confounded the EMG readings. The tentativeness of EMG readings is supported by the observations of Gunendi et al, who suggest that an extended learning of at least 10 test trials may be required to properly assess electromyographic reaction time values [65].

Instead, we turned to vibromyography (VMG) as a more specific probe. Physiologic tremor and motion of the body part under study has hindered previous attempts to correlate muscle vibration with muscle effort, seen with sEMG. The VMG system has been suggested to overcome this difficulty through the use of low-noise accelerometers and electronic bandpass filtering, together with software signal processing capability to extract the specific components of the vibration spectrum that correlates best with muscle activity. Basically, the VMG system has been proposed to correlate muscle body vibrations with absolute muscle effort through the full range of maximum voluntary contraction, requiring only a single point measurement [66, 67]. Previously, a difference in muscle vibration between strong and weak results in MMT was reported with a VMG device, in that a 7-10 Hz vibration in strong muscle (equated to proprioceptive activity) damped out as the muscle broke away in a weak test result [68]. Our VMG results concur with this finding and for the first time have extended the utility of this instrumentation to detecting the presence of therapy localization in the PAK testing of the middle deltoid muscle.

Our demonstration of the significant elevation of the occurrence of TL with neck injury as shown in Table 3 is congruent with the findings of Pollard et al., who reported that TL to the ileocecal valve producing weakness in MMT strongly correlated with the presence of low back pain in individuals. In that investigation the sensitivity (when back pain was present) was 87%, and the specificity (when back pain was absent) was 97%. Accordingly, the authors were able to determine that the likelihood of obtaining a positive TL was 28.6 times more likely to be found in individuals with back pain rather than without [69]. The same

lead author also concurred with our findings regarding the good inter-examiner reliability that was obtained in AK muscle testing [17].

The data in this investigation not only support the validity of TL, but represent a continuing effort to identify and develop the instrumentation that is most capable of representing the attributes of PAK. While not addressing TL itself, several previous investigations have attempted to demonstrate correlations of MMT with either Cybex [70] or manual myometry [71] or dynamometry [72-76].

These methods measure muscle strength but do not take into consideration the rapidity of the MMT or especially the role of the nervous system in adapting the muscle to the changing pressure of the test applied by the examiner in PAK. We suspect that the applications of the electrogoniometer and VMG described in this report are a step closer to following the intricacies of the muscle's response to PAK and thus better equipped to validate the attributes of PAK itself.

Limitations and Suggestions for Future Research

Although the VMG analysis was able to detect the presence of TL at a significance level of $p < 0.05$, this was obtained with just 5 subjects. It is anticipated that higher levels of statistical significance would be obtained with a larger number of participants in future research. To obtain even more robust data in subsequent investigations, the sensor would have to be placed on each subject the exact same way each time, in the same manner that electrocardiogram electrodes are placed in a standard configuration on the patient's chest to obtain valid data. Also requiring further scrutiny is how tightly the sensor is placed on the muscle with tape or ace bandages.

One limitation of the study was that the examiner was not blinded as to whether or not therapy localization was applied. Future investigations would have to involve a third party out of sight of the examiner who would, in

random sequence, ask the patient to (a) do nothing, (b) touch an area that is not considered an active myotome to the muscle being tested, or (c) conduct an actual therapy localization.

A second limitation was that this study was confined to the middle deltoid as a single test muscle. Future research will need to generalize these findings with the application of additional muscles and myotomes in therapy localization.

In spite of these reservations, this remains the first investigation to have confirmed the reliability of TL in PAK and supported its validity with the objective data provided by instrumentation.

References

- [1] Goodheart GJ. *Applied Kinesiology 1974 Research Manual, 10 Edition*. Detroit, MI: Privately published, 1974.
- [2] Walther DS. *Applied Kinesiology Synopsis, 2nd Edition*. Shawnee Mission, KS: International College of Applied Kinesiology-USA, 1988-2000, pp. 37-38.
- [3] Shafton AD, Furnell JB, Ferens D, Bogeski G, Koh SL, Lean NP, Kitchener PD. The visceromotor responses to colorectal distension and skin pinch are inhibited by simultaneous jejunal distension. *Pain* 2006;123(1-2):127-136. Epub 2006 May 16.
- [4] Morrison JF, Sato A, Sato Y, Yamanishi T. The influence of afferent inputs from skin and viscera on the activity of the bladder and the skeletal muscle surrounding the urethra in the rat. *Neurosci Res* 1995; 23(2):195-205.
- [5] Pomeranz B, Wall PD, Weber WV. Cord cells responding to fine myelinated afferents from viscera, muscle and skin. *J Physiol* 1968; 199(3):511-532.
- [6] Kaufman A, Sato A, Sato Y, Sugimoto H. Reflex changes in heart rate after mechanical and thermal stimulation of the skin at various segmental levels in cats. *Neuroscience* 1977; 2(1):103-109.
- [7] Fallon JB, Bent LR, McNulty PA, Macefield VG. Evidence for strong synaptic coupling between single tactile afferents from the sole of the foot and motoneurons supplying leg muscles. *J Neurophysiol* 2005; 94(6):3795-3804.
- [8] Zimlichman E, Lahad A, Aron-Maor A, Kanevsky A, Shoenfield Y. Measurement of electrical skin impedance of dermal-visceral zones as a diagnostic tool for inner organ

- pathologies: A blinded preliminary evaluation of a new technique. *Israel Medical Association Journal* 2005; 7(10):631-634.
- [9] Tokimura H, Di Lazzaro V, Tokimura Y, Oliviero A, Profice P, Insola A, Mazzzone P, Tonali P, Rothwell JC. Short latency inhibition of human hand motor cortex by somatosensory input from the hand. *J Physiol* 2000; 523 Pt 2: 503-513.
- [10] Hall T, Quinter J. Responses to mechanical stimulation of the upper limb in painful cervical radiculopathy. *Austr J Physiother* 1996; 42(4):277-285.
- [11] Nicholas JA, Melvin M, Saraniti AJ. Neurophysiologic inhibition of strength following tactile stimulation of the skin. *Am J Sports Med.* 1980; 14(9):181-186.
- [12] Caruso W, Leisman G. A force/displacement analysis of muscle testing. *Percept Mot Skills* 2000;91:683-692.
- [13] Caruso W, Leisman G. The clinical utility of force/displacement analysis of muscle testing in applied kinesiology. *Intern J Neuroscience* 2001;106:147-157.
- [14] Leisman G, Shambaugh P, Ferentz AH. Somatosensory evoked potential changes during muscle testing. *Intern J Neuroscience.* 1989; 45: 143-151.
- [15] Pittman F. On the state of the art of basic research on muscle tests according to Goodheart. Proceedings of the International Congress of Applied Kinesiology, Berlin, Germany, September 17-19, 2010, p. 14.
- [16] Cuthbert S, Rosner A, McDowall D. Association of manual muscle tests and mechanical neck pain: Results from a prospective pilot study. *J Bodywork Movement Therapies.* 2011;15:192-200.
- [17] Pollard H, Lakay B, Tucker F, Watson B, Bablis P. Inter-examiner reliability of the deltoid and psoas muscle test. *J Manip Physiol Therapeut.* 2005; 28(1): 52-56.
- [18] Kendall FP, Kendall McCreary E, Provance PG. *Muscles: Testing and Function, 4th edition.* Baltimore, MD: Williams and Wilkins, 1993, pp. 179-190.
- [19] Conable K, Corneal J, Hambrick T, Marquina N, Zhang J. Investigation of methods and styles of manual muscle testing by AK practitioners. *J Chiropractic Med.* 2005; 4(1): 1-10.
- [20] Conable KM, Rosner AL. A narrative review of manual muscle testing and implications for muscle testing research. *J Chiropractic Med.* 2011; 10: 157-165.
- [21] <http://www.randomizer.org/form.htm>
- [22] Rosner, AL. Applied kinesiology: Distinctions in its definition and interpretation. *J Bodywork Movement Ther* 2012, doi:10.1016/j.jbmt.2012.04.008.
- [23] Cuthbert SC, Goodheart Jr.,GJ. On the reliability and validity of manual muscle testing: a literature review. *Chiropractic & Osteopathy* 2007;15 (1): 4.
- [24] Kendall HO, Kendall FP. *Posture and Pain.* Baltimore, MD: Williams & Wilkins, 1952.
- [25] Goodheart GJ. *Applied Kinesiology Research Manuals.* Detroit, MI: Privately published yearly, 1964-1995.
- [26] Hunter SJ, Shaha S, Flint D, Tracy DM. Predicting return to work: A long term follow-up study of railroad workers after low back injuries. *Spine* 1998; 23: 2319-2327.
- [27] Hildebrandt J, Pflingsten M, Saur P, Jansen J. Prediction of success from a multidisciplinary treatment program for low back pain. *Spine* . 1997;21:2640-2650.
- [28] Leamon TB. Research to reality: A critical review of the validity of various criteria for the prevention of experimentally induced low back pain disability. *Ergonomics* 1994; 37: 1959-1974.
- [29] Frymoyer JW. Predicting disability from low back pain. *Clin Orthop Ref Fes* 1992; 279: 103-`09.
- [30] Adams MA, Mannion AF, Dolan P. Personal risk factors for first-time low back pain. *Spine* 1999; 24: 2497-2505.
- [31] Janda V. Muscles, central nervous motor regulation, and back problems. In: Korr IM [Ed]. *The Neurobiologic Mechanisms of Manipulative Therapy* 1978; New York, NY: Plenum Press.
- [32] Janda V. Muscles as a pathogenic factor in back pain. *Proceedings of the International Federation of Orthopedic Manual Therapies.* 1980; New Zealand.
- [33] Rodriguez, A., Agre, J. Electrophysiologic study of the quadriceps muscles during fatiguing exercise and recovery: A comparison of symptomatic and asymptomatic postpolio patients and controls. 1991. *Arch Phys. Med Rehabil.* 72(12): 993-7.
- [34] Hsieh J, Gilbertson K. Reliability of mean power frequency and median power frequency in bilateral upper trapezius isometric work. *Proceedings of the International Conference on Spinal Manipulation,* 1993, pp. 21-23.
- [35] Van diesen JH. Asymmetry of erector spinae muscle activity in twisted postures and consistency of muscle activation patterns across subjects. *Spine* 1996;21(22): 2651-61.
- [36] Sparto, PJ, Parnianpour M, Reinsel TE., Simon S. Spectral and temporal responses of trunk extensor electromyography to an isometric endurance test. *Spine.* 1997;22(4):418-26.

- [37] Sparto, P.J., Parnianpour M. An electromyography-assisted model to estimate trunk muscle forces during fatiguing repetitive trunk exertions. *J Spinal Disord Tech* 1999;12(6): 509-18.
- [38] Niermeyer LO, Arnonow HU, Kasman GS. A pilot study to investigate shoulder muscle fatigue during a sustained isometric wheelchair-propulsion effort using surface EMG. *Am J Occup Ther.* 2004;58(5):587-593.
- [39] Ambroz C, Scott A, Ambroz A, Talbott EO. Chronic low back pain assessment using surface electromyography. *J Occup. Environ. Med.* 2000;42(6): 660-669.
- [40] Sihvonen, T., Huttunen, M., Makkonen, M., Airaksinen, O. 1998 Functional changes in back muscle activity correlate with pain intensity and prediction of low back pain during pregnancy. *Arch Phys Med Rehabil* 79(10): 1210-2.
- [41] Michener, L.A., Boardman, N.D., Pidcoe, P.E., Frith, A.M. 2005. Scapular muscle tests in subjects with shoulder pain and functional loss: Reliability and construct validity. *Phys. Ther.* 85(11): 1128-38.
- [42] Maquet, D., Demoulin, C., Forthomme, B., Crielaard, J.M., Crosier, J.L. 2005. Activation EMG pattern during isokinetic knee flexion-extension assessment: Comparison between healthy subjects and chronic pain patients. *Isokinet. Exer. Sci.* 13(1): 5-11.
- [43] Bothelo, A.L., Silva, B.C., Gentil, F.H., Sforza, C., da Silva, M.A. 2010. Immediate effect of the resilient splint evaluated using surface electromyography in patients with TMD. *Cranio.* 28(4): 266-73.
- [44] Brunner, R., Romkes, J. 2008. Abnormal EMG muscle activity during gait in patients without neurological disorders. *Gait Posture* 27(3): 399-407.
- [45] Marciello, M., Herbison, G., Cohen, M., Schmidt, R. 1995. Elbow extension using anterior deltoids and upper pectoralis in spinal cord-injured subjects. *Arch. Phys. Med. Rehabil.* 76(5): 426-32.
- [46] Wu, S.H., Chu, N.K., Liu, Y.C., Chen, C.K., Tang, S.F., Cheng, C.K. 2008. Relationship between the EMG ratio of muscle activation and bony structure in osteoarthritic knee patients with and without patellar misalignment. *J. Rehabil. Med.* 40(5): 381-6.
- [47] Gosselin, N., Matecki, S., Poulain, M., Ramonatxo, M., Ceugniet, F., Prefaut, C., Varray, A. 2003. Electrophysiologic changes during exercise testing in patients with chronic obstructive pulmonary disease. *Muscle Nerve* 27(2): 170-9.
- [48] Ounpuu, S., DeLuca, P.A., Bell, K.J., Davis, R.B. 1997. Using surface electrodes for the evaluation of the rectus femoris, vastus medialis, and vastus lateralis in children with cerebral palsy. *Gait Posture* 5(3): 211-6.
- [49] Tian, S.L., Liu, Y., Fu, W.J., Peng, C.H. 2010. Mechanomyography is more sensitive than EMG in detecting age-related sarcopenia. *J Biomech* 43(3): 551-6.
- [50] Ninos, J.C., Irrgang, J.J., Burdett, R., Weiss, J.R. 1997. Electromyographic analysis of the squat performed in self-selected lower extremity neutral rotation and 30 degrees of lower extremity turnout from the self-selected neutral position. *J. Orthop. Sports Phys. Ther.* 25(5): 307-15.
- [51] Kellis E, Katis A. The relationship between isokinetic knee extension and flexion strength with soccer kick kinematics: An electromyographic evaluation. *J Sports Med. Phys. Fitness* 2007;47(4): 385-94.
- [52] Chapman AR, Vincenzino B, Blanch P, Dowlan S, Hodges PW. Does cycling effect motor coordination of the leg running in elite triathletes? *JSMS* 11(4): 371-80.
- [53] Kotila K, Sveinsson T, Arnason A. Evidence-based testing of the hamstring muscles using wireless surface emg. *Br J Sports Med.* 2011;45(4): 358.
- [54] Briem K, Eythorsdottir H, Magnúsdóttir RG, Palmársson R, Runárásdóttir T, Sveinsson T. Effects of kinesio tape compared with nonelastic sports tape and the untaped ankle during a sudden inversion perturbation in male athletes. *J Orthop Sports Phys. Ther.* 2011;41(5): 328-35.
- [55] Jenp YN, Malanga GA, Growney ES., An, KN. Activation of the rotator cuff in generating isometric shoulder rotation torque. *Am J Sports Med.* 1996;24(4): 477-85.
- [56] Verstappen FT, Veldhuizen JW, Twellaar M, Drost MR, Kuipers H Isokinetic aerobic power output testing of the quadriceps muscle. *Int J Sports Med.* 1988;19(7): 485-9.
- [57] Danneels LA, Cagnie BJ, Cools AM, Vanderstraeten CG, Cambier DC, Witvrouw EE, Cuyper HJD. Intra-operator and inter-operator reliability of surface electromyography in the clinical evaluation of back muscles. *Manual Therapy.* 2001;6(3): 145-53.
- [58] Ekstrom RA, Bifulco KM, Lopau CJ, Andersen CF, Gough JR. Comparing the function of the upper and lower parts of the serratus anterior muscle using surface electromyography. *J Orthop Sports Phys Ther.* 34(5):2004;235-243.
- [59] Howatson G, van Someren KA. The reproducibility of peak isometric torque and electromyography activity in unfamiliarised

- subjects using isokinetic dynamometry on repeated days. *Isokinet Exerc Sci.* 2005;3(2): 103-109.
- [60] Howatson G, van Someren KA. The reproducibility of peak isometric torque and EMG activity in unfamiliarised subjects on repeated days. *Isokinet Exerc Sci.* 2005;13(3):179-186.
- [61] Zito M, Tiberio D, Rhoad J. Electromyographic validation of heel-walking test for ankle dorsiflexor muscle strength. *J Human Muscle Perform.* 1991;1(3):47-53.
- [62] Krishnan C, Allen EJ, Williams GN. Effect of knee position on quadriceps muscle force steadiness and activation strategies. *Muscle Nerve.* 2011;43(4):563-573.
- [63] Earl JE, Piazza SJ, Hertel J. The Protonics Knee Brace unloads the quadriceps muscle in healthy subjects. *J Athl Train* 2004;39(1):44-49.
- [64] Moncayo R, Moncayo H. Evaluation of applied kinesiology meridian techniques by means of surface electromyography (sEMG): Demonstration of the regulatory influence of antique acupuncture points. *Chin Med.* 2009;4(9): 9.
- [65] Gunendi Z, Taskiran OO, Beyazova M. What is the optimal repetition number in electromyographic reaction time studies? *Clin Biomech.* 2005;20(7):754-758.
- [66] Cole JP, Madhavan G, McLeod KJ. Vibromyographic quantification of voluntary isometric contractile force in the brachioradialis. *Proceedings of the IEEE Engineering, Medical, and Biological Society* 2006; 1:1708-1710.
- [67] Matheson GO, Maffey-Ward L, Mooney M, Ladley K, Fung T, Zhang YT. Vibromyography as a quantitative measure of muscle force production. *Scand J Rehab Med.* 1997; 29(1): 29-35.
- [68] Pittman F. On the state of the art of basic research on muscle tests according to Goodheart. *Proceedings of the International Congress of Applied Kinesiology*, Berlin, September 17-19, 2010, p. 14.
- [69] Pollard HP, Bablis P, Bonello R. Can the ileocecal valve point predict low back pain using manual muscle testing? *Chiropr J Austr.* 2006;36: 58-62.
- [70] Lin HT, Hsu AT, Chang JH, Chien CS, Chang GL. Comparison of EMG activity between maximal manual muscle testing and cybex maximal isometric testing of the quadriceps femoris. *J Formos Med Assoc.* 2009;107(2):175-180.
- [71] Schwartz S, Cohen ME, Herbison GI, Shah A. Relationship between two measures of upper extremity muscle strength: Manual muscle test compared to hand-held myometry. *Arch Phys Med Rehabil.* 1992;73(11): 1063-1068.
- [72] Marino M, Nicholas JA, Glenn GW, Rosenthal P, Nicholas S. The efficacy of manual assessment of muscle strength using a new device. *Am J Sports Med.* 1982;10(6):360-64.
- [73] Bohannon RW. Manual muscle test scores and dynamometer test scores of knee extension strength. *Arch. Phys. Med. Rehabil.* 1986;67(6):390-392.
- [74] Wadsworth CT, Krishnan R, Sear M, Harrold J, Nielson DH. Inter-examiner reliability of manual muscle testing and hand-held dynamometer muscle testing. *Phys Ther.* 1987;67(9):1342-1347.
- [75] Hsieh CY, Phillips RB. Reliability of manual muscle testing with a computerized dynamometer. *J Manip Physiol Ther.* 1990;13(2): 72-82.
- [76] Bohannon RW. Measuring knee extensor muscle strength. *Am. J Phys Med Rehabil.* 2001;80(1):13-18

