

# THE EVOLVING CHARACTERISTICS OF CLINICAL TEST DEVELOPMENT IN MUSCULOSKELETAL PRACTICE WITH 3 NOVEL CLINICAL TEST EXAMPLES

By Deepak Sebastian

# **A DISSERTATION**

Presented to the Department of ORTHOPAEDIC PHYSICAL THERAPY program at Selinus University

Faculty of NATURAL HEALTH SCIENCE in fulfillment of the requirements for the degree of Doctor of Philosophy in ORTHOPAEDIC PHYSICAL THERAPY

2023

# **CONTENTS:**

- **1. PRELUDE AND THESIS OBJECTIVE**
- 2. WHAT IS METHODOLOGICAL RESEARCH

# 3. PHASE 1: HYPOTHESIS GENERATION AND DESCRIPTION PHASE OF A CLINICAL TEST (EXAMPLE: ACTIVE SUPINATION DORSIFLEXION TEST)

- Conceptual basis of the first phase (description) of clinical test development
- Title
- Abstract and Introduction
- Materials and methods
- Tables and Figures (embedded throughout)
- Contents and results
- Discussion and defense of the description of the test
- Conclusion
- Bibliography

# 4. PHASE 2: REPRODUCIBILITY TESTING PHASE OF A CLINICAL TEST (EXAMPLE: SCAPULA BACKWARD TIPPING TEST)

- Conceptual basis of the second phase (reliability) of clinical test development
- Title
- Abstract and Introduction
- Materials and methods
- Tables and Figures (embedded throughout)
- Contents and results
- Discussion and defense of the reliability of the test

- Conclusion
- Bibliography

# 5. PHASE 3: VALIDATION TESTING PHASE OF A CLINICAL TEST (EXAMPLE: SITTING ACTIVE PRONE PASSIVE LAG TEST)

- Conceptual basis of the third phase (validity) of clinical test development
- Title
- Abstract and Introduction
- Materials and methods
- Tables and Figures (embedded throughout)
- Contents and results
- Discussion and defense of the validity of the test
- Conclusion
- Bibliography

# 6. ACNOWLEDGEMENTS

# **PRELUDE AND THESIS OBJECTIVE**

Methodological studies provide information on the design, conduct, analysis or reporting of primary and secondary research and can be used to appraise quality, quantity, completeness, consistency (reliability) and accuracy (validity) of health research. One area related to methodological research involves the development and testing of clinical tests for use in musculoskeletal practice. Musculoskeletal clinicians utilize testing methods to identify the presence or absence of musculoskeletal dysfunction. These clinical tests are the basis of the identification of the presence or absence of musculoskeletal dysfunction but are not always confirmatory. When not confirmatory they may at least improve the probability of the presence or absence of the dysfunction. The label of confirmation is called a diagnosis and is typically made by clinicians trained or certified to do so.

Hence the question is, how are these musculoskeletal clinical tests created and what are the stages in the evolutionary process of these musculoskeletal clinical tests. In a nutshell at a very grassroot level clinical tests in musculoskeletal practice are born out of a passionate and hands on musculoskeletal clinician that is constantly observing, palpating, feeling, and moving body parts. Relevant to a specific body part, the clinician may develop and adopt a personal style or method of evaluation, relevant to a particular diagnosis. Examples include a unique palpation technique, an instrument ingeniously designed to measure muscle or joint function, eliciting a pattern of movement or presentation seen consistently in a particular category of patient or diagnosis, etc. It may be a single maneuver or a set of findings but nevertheless a consistent pattern. The word 'consistent' has more than just a vernacular sense here as this consistency may be the beginning of the establishment of a novel screening or diagnosis idea. It may evolve in stages as follows: 1. Firstly, identifying a consistent pattern of presentation and researching the literature to see of the pattern has been previously described

2. Continuing to apply this consistent pattern (observing its continued consistency) and seeing if it truly has clinical benefit and then proclaiming it (description).

3. As the clinician (in this case myself) is the only individual that 'sees' it, requesting colleagues to join the process and observe if they see what I see. If so, pursuing the process of establishment of reproducibility (reliability).

4. If reproducible, then utilizing it on patients who have the relevant diagnosis for its 'presence' consistently, and more importantly applying it on patients who do not have the relevant diagnosis to observe for its 'absence' consistently. Thus, establishing truthfulness (validity).

5. At every level, be it description, reliability, or validity, publishing the finding in the interest of enhancement of patient care and contribution to the body of literature.

Hence the objective of my thesis is to describe and narrate 3 novel and original clinical tests in musculoskeletal practice, originally described by me as a result of clinical intervention and laboratory and data analysis which are already published in peer reviewed journals. The authenticity of this thesis is that all 3 tests (besides being a personal original description of work done over several years), aptly follow the 3 stages of clinical test development in musculoskeletal practice, namely:

**1.** Description and case reporting

- **2.** Reliability testing
- 3. Validity testing

The 3 tests and their published citation is as follows:

- Sebastian D. The Active Supination Dorsiflexion Test Guided Therapeutic Intervention for Shin and Calf Pain: A Case Report. J Musculoskelet Disord Treat. 2020. 6;078: 1510078.
- Sebastian D, Chovvath R, Malladi M. The scapula backward tipping test: An inter-rater reliability study. Journal of Bodywork & Movement Therapies. 2017; 21: 69-73.
- 6. Sebastian D, George, Tsang Z. The sitting active and prone passive lag test: A Validity Study in a Symptomatic Knee Population. Journal of Physical Therapy Science (accepted for publication in May 2023 issue).

These 3 clinical tests have been published at these 3 individual stages, one at a descriptive stage, the other that was evaluated for reliability after description, and finally the last one that was validated after description and reliability was established. The thesis will thus exemplify the evolutionary process of the establishment of clinical tests in musculoskeletal practice which may even apply to clinical tests that may have the ability to reach the status of a gold standard.

# METHODOLOGICAL RESEARCH

## METHODOLOGICAL RESEARCH:

As mentioned in the previous chapter, one area related to methodological research involves the development and testing of clinical tests for use in clinical practice. validity.

Clinicians utilize testing methods to identify the presence or absence of clinical conditions. Validated testing methods are learnt during clinical training via classroom sessions, courses, textbooks, journals, and peer interaction. Although, tests are the basis of the identification of the presence or absence of disease but are not confirmatory. The label of confirmation is called a **diagnosis** and is typically made by an individual that has been trained and certified in that area to do so. One needs to understand that an individual does not need to be unwell to undergo a test leading to a diagnosis. In the era of prevention and wellness tests are performed as an evaluation, assessment, or screening, on individuals who are currently well but with potential risk. Hence the term diagnosis may not apply to an individual who is currently well. For this reason, understanding the operant terms for the **'identification'** of a clinical problem requires explanation. This is mainly since the term's examination, assessment, evaluation, testing, screening, and diagnosis are often used interchangeably. They require clarification as they are not synonymous.

An **examination** in clinical practice is the first examination performed by an appropriate health provider. It helps the health provider to determine the initial disease status and directs the provider to additional testing with the goal of diagnosing a disease or dysfunction.

A **clinical test** is performed as a component of the examination process and is used in clinical practice (with satisfactory accuracy leading to a diagnosis) to identify a disease, disorder, or dysfunction to be able to provide timely and appropriate management. Tests may be simple or procedural. A simple test may require the consideration of appropriateness and cost but not the risks of performing it. For example, most hands-on tests performed in musculoskeletal and rehabilitation practice may be appropriate, inexpensive with potentially low to no risk. A procedural test, on the other hand, usually implies an invasive procedure that carries some risk. Cardiac catheterization, laparoscopy, or colonoscopy may be examples. In these cases, the benefit often outweighs the risk.

Clinical tests take many forms from a simple hands-on test to a cluster of such tests, to a procedural test, to a battery of tests that may be a combination of a hands-on test, lab test and procedural test all put together. The goals of these combinations are simply to improve the accuracy of determining the presence or absence of a disease, disorder, or dysfunction. The reason why clinical tests are given special importance is that they eventually form the basis, set or reference standard to confirm the absence, presence, potential risk of a disease or the cause of a disorder, dysfunction, or death. They are utilized as a process leading to a clinical diagnosis and to also perform periodic evaluations and assessments.

A **diagnosis** is the clinical conclusion that a provider would make based on the initial examination and clinical testing. Simply put, this conclusion when drawn on a symptomatic individual results in a diagnosis. On the other hand, when done on an asymptomatic individual during an annual physical or an epidemic it is called a **screening**. Nevertheless, the basis and goal of what a clinical test purports to accomplish remains.

An **evaluation** in clinical practice focuses on making a judgment about values, numbers, or the performance of an individual. The judgement is made based on set qualitative or quantitative standards. It gauges the performance of that individual, to determine abilities with reference to a set standard (e.g. normal values on a clinical test).

In clinical practice examples of reference or set standards may be normal values which indicate normal health, i.e., normal blood pressure, lab levels, range of motion and strength of a motion segment etc.

An assessment in clinical practice is the process of appraising an individual and is often made to identify the level of performance of an individual. The process may involve collecting information from an evaluation (quantitative and qualitative) to set goals and use that information for improving quality. In clinical practice this may be done after the diagnostic process and management plan has been established, during an interim progress evaluation or during discharge.

#### Disease versus diagnosis of impairment-based dysfunction:

A disease is any harmful deviation from the normal structural or functional state of an organism characterized by signs and symptoms and specific findings in a validated diagnostic test like lab analysis or imaging. These tests are usually high in specificity that may confirm the presence or absence of a disease process. They may also hence serve as a gold standard for diagnosis. A gold standard however only refers to a benchmark and is not 'the' perfect test, but the best available one that has a standard with known results (Jefferson Rosa Cardoso).

The conceptual basis of movement impairment-based dysfunction is that repeated or prolonged activity and postures or sustained alignment in a non-ideal position and repetitive movements in a specific direction are thought to be associated with several musculoskeletal conditions. While this occurs, what compounds the problem is the lack of awareness about where the dysfunctional patterns in the body are. They could be in the form of weakness, tightness, altered mobility patterns etc. When paired with continued and increasing load demand, those non-optimal patterns can create inefficiencies. They may also create a cascade of compensatory movement, joint stress and risk for injury. Interestingly this could be the premise of the conventional diagnosis we see in everyday clinical practice like tendinitis, nerve entrapments and soft tissue tears. Hence identifying these dysfunctional patterns prior to the establishment of a pathology requires testing that typically cannot be performed by imaging or lab analysis. They typically involve a clinician performing these tests that involve anthropometric alignment or movement testing. These tests hence recognize risk or when combined with other similar tests may increase the likelihood of the presence or absence of a dysfunction. Hence seldom are these tests a gold standard and hence lack specificity.

In the process of investigating impairment based musculoskeletal dysfunction, screening tests are more frequently used in comparison to confirmatory tests. Sometimes more than one of these screening tests are used as a cluster of tests which when positive or negative as a majority, can further increase the likelihood of the presence or absence of the dysfunction in question. There hence may be a difference in examining the presence of disease as opposed to an impairment-based dysfunction.

### The screening / diagnosis idea:

In clinical practice 'identification' begins with an 'index of suspicion' or in an 'at risk' situation. This may be an individual with actual symptoms or is at risk for certain conditions based on age, gender, anthropometry, demographics, race etc.

The term screening and diagnosis are frequently encountered in clinical practice. Although mutually synonymous they differ completely in their objective. Screening tests may precede diagnostic tests and may be performed on healthy individuals. They may also be performed on a symptomatic individual in the process of elimination to zero into a particular problem. Hence a screening test is to detect early disease, risk factors for disease or to zero into a particular disease process. They are usually done in large numbers of mostly healthy individuals but can also be done in a symptomatic individual in the process of diagnostic elimination. Hence, screening test may also serve as a diagnostic test or as part of a test cluster. A diagnostic test, however, may be used to establish the presence or absence of disease. It may be done as a basis for treatment decisions in symptomatic individuals or in individuals who tested positive in a screening test. It is hence used as a confirmatory test to establish a diagnosis. In summary, screening tests may lean toward being more sensitive, while diagnostic tests aim at being specific to a pathological process.

A passionate and hands on musculoskeletal clinician is constantly observing, palpating, feeling, and moving body parts. Relevant to a specific body part each clinician may have a personal style or method of evaluation relevant to a particular diagnosis. This may be a unique auscultation sound, or an instrument that one had ingeniously designed in the garage meant to measure a physiological function, a noel lab test or a variation of an existing lab test, a pattern, movement or presentation seen consistently in a particular category of patient or diagnosis. It may be a single maneuver or a set of findings but nevertheless a consistent pattern. The word 'consistent' has more than just a vernacular reference here as the consistency may be the beginning of the establishment of a novel screening or diagnosis idea. While this may not occur all the time, what does one do if it does and especially if it may be of clinical value that might benefit patients. In that case it ideally must be shared with the clinical community and there is a method, a scientific method to it and the steps are as follows:

# 1. Identifying a consistent pattern and researching the literature to see of the pattern has been previously described

The clinician is recommended to pursue and identify this unique or novel finding during clinical practice and side by side research the literature to see it has been previously described.

# 2. Continue to apply this consistent pattern and observe to see if it truly has clinical benefit

In the process of following the identified unique method its clinical usefulness carries the most weight. While the clinician continues to pursue and identify this unique or novel finding during clinical practice, its clinical benefit must be investigated.

3. As the clinician is the only individual that 'sees' it, request a colleague to join the process and observe if they see what you see. If so, may require validation for reproducibility.

Sometimes the passion and zeal for ones craft may lead to the perceiving of findings that may be farfetched or grandiose and the subsequent vehemence of defending its position. This is where reproducibility matters

4. If reproducible, then apply it on patients who have the relevant diagnosis for its 'presence' consistently, and more importantly apply it on patients who do not have the relevant diagnosis for its 'absence' consistently. This will require validation for validity.

5. If reproducible and valid, publish your finding.

# **Steps to validating a clinical test:**

# **Reliability**:

A test is considered reliable when it is reproducible consistently. This means a positive or a negative finding can be established consistently when an examiner performs the test repeatedly on a given condition or when two examiners perform it at the same time for the same condition. For example, when an individual presents with a fracture of the shaft of the femur, a plain radiograph may show the fracture no matter how may times the radiograph is repeated by the examiner if the fracture is present. This is known as intrarater reliability. Assuming once the diagnosis is established as a 'positive' for a fracture of the shaft of the femur, if the radiograph is presented to another clinician with the same level of experience, the same diagnosis may be agreed upon repeatedly with more clinicians with similar experience. Since the radiographic finding was consistent between two or more examiners the credit for being a reliable test may be directed to the testing method, the radiograph, as it not only picked up an accurate diagnosis but also established consistency between two or more examiners with similar experience. This is known as inter-rater reliability.

# **Recognizing Test Reliability:**

The reliability of a clinical test is assessed by performing a category of statistics called analysis or relationships. We may loosely assume that it an analysis of how close a relationship exists between the disease or clinical condition and the test so it identifies it. The is the opposite of experimental trials that compare the efficacy of two treatment procedures to identify if one is better than the other. This would be a category of statistics called analysis or differences. of a clinical test is the extent to which results are consistent between different and blinded examiners across different occasions of testing. The weighted kappa was used to measure association as the scale of measurement was ordinal. Thus, inter-rater reliability was determined using the kappa statistic (Raghavan, Fosler and Lai, 2012).

## Validity:

Validity in clinical test parlance means usefulness. A blunt knife may be reliable if it consistently fails to cut raw meat. Although a reliable finding it lacks usefulness as a knife should do what it is made to do, cut. Similarly, a clinical test, despite being reproducible or reliable it should do what it purports to do, accurately detect a clinical problem when it is present or not detect its presence when it is absent. A valid test can do this and if the level of accuracy close to being flawless, it may be considered the gold standard or reference standard.

### **Establishing reliability and validity:**

**Test Reliability:** 

### **Reliability evaluation:**

- AA: Both examiners identified positive
- BB: Both examiners identified negative
- AB: A identified positive and B identified negative
- BA: B identified positive and A identified negative

2x2 Contingency Table for Reliability

	A	В	
A	AA	AB	Total
В	BA	BB	Total
	Total	Total	

Kappa= Range 0-1

# **Test Validity:**

The data collected consisted of True positives (TP), False positives (FP), False negatives (FN) and True negatives (TN), and were represented and calculated on a 2x2 contingency table (Hassanzadeh J and Rezaianzadeh A, 2012) (Fig. 5).

# 2x2 Contingency Table for Validity

Test	Present	Absent	Total
+VE	ТР	FP	TP+FP
-VE	FN	TN	FN+TN

Total	TP+FN	FP+TN	Total

The parameters of relevance were Sensitivity, Specificity, Predictive Values and Likelihood Ratios. Sensitivity (Sn) is the percentage with a knee dysfunction who test positive on the dysfunctional side and is calculated as Sn=TP/(TP+FN). A test that is highly sensitive is good at ruling out disease or an impairment and are good for screening purposes. Specificity (Sp) is the percentage without a knee dysfunction who test negative on the non-dysfunctional side and is calculated as Sp=TN/(FP+TN). A test that is highly specific is good at ruling in disease or an impairment and are good confirmatory tests. Likelihood Ratio (LR) represents the probability of a patient with a disease or dysfunction having a positive or negative test result in comparison to the probability of a patient without the disease having a positive or negative test result. Positive LR (+ve LR) suggests the number of times more likely a positive test result observed in the symptomatic knee versus the asymptomatic knee. It suggests how well disease is ruled in. Positive likelihood ration is calculated as +ve LR = sensitivity/1-specificity. Negative LR suggests the number of times more likely a negative test result is observed in the symptomatic knee versus asymptomatic knee. It suggests how well disease is ruled out. Negative likelihood ratio is calculated as -LR = 1-sensitivity/specificity. Positive predictive value (PPV) is the percentage of positive test results that are true positives and is calculated as PPV=TP/(TP+FP). Negative predictive value (NPV): It is the percentage of negative test results that are true negatives and is calculated as NPV=TN/(FN+TN).

# **Receiver operating characteristic (ROC)**

ROC curves are a graphical depiction of a test's overall diagnostic performance (Karimollah Hajian-Tilaki, 2013). The Y axis represents Sensitivity, and the X axis represents 1-Specificity. The closer the curve fills out the top left corner, the better the test is performance is quantified by the area under the curve (AUC). An AUC of 0.5 states that the test performs no better than chance (not useful). An AUC of 0.9 would suggest a better-performing test.

# **PHASE 1: HYPOTHESIS GENERATION**

# (DESCRIPTION)

# THE ACTIVE SUPINATION DORSIFLEXION TEST

**ORIGINAL DESCRIPTION:** The Active Supination Dorsiflexion Test Guided Therapeutic Intervention for Shin and Calf Pain: A Case Report. J Musculoskelet Disord Treat. 2020. 6;078: 1510078

# **Conceptual basis of the first phase (description) of clinical test development:**

A clinical testing method in clinical practices is an investigation attempt. For example, a clinician observes a joint to possess a certain degree of movement and decides to make assessment of joint range of motion a testing method. A progression may be made to see a pattern of joint restriction. As an example, observing the calcaneus tending to be restricted in eversion or the talus tending to be restricted in plantarflexion in the presence of flat feet. Justifying this type of a presentation in association with a pathology, consistently, will justify a hypothetical basis for the clinical test. The justification, however, may require clinical support and adequate referencing. The active supination dorsiflexion test was described in a similar manner as it was consistently seen in individuals with pain and dysfunction associated with foot pronation (flat feet). The following is an original description example conducted by me (PhD candidate) that describes how a hypothetical clinical test can be justified and described.

#### **ACTIVE SUPINATION DORSIFLEXION TEST**

**Abstract:** A 66-year-old male experienced right sided shin and calf pain of an insidious onset. The duration of pain was 3 months, aggravated by walking and running. He also reported resting pain especially during the night. A detailed medical evaluation ruled out the presence of a blood clot and electrolyte imbalance. He was diagnosed as having restless leg syndrome and referred for physical rehabilitation. Initial examination revealed positive findings of comparable local tenderness over the right shin and calf. He also presented with foot pronation that persisted throughout the stance phase of gait. Hence his ability to reverse pronation was tested in the standing position. With the knees completely extended, he presented with an inability to actively supinate and dorsiflex the foot on the right side. 8 treatment visits for a period of 4 weeks addressed mechanical dysfunction at the ankle, foot, and hip region, comprising manual therapy, corrective exercise and pain modalities. Reduction of local tenderness, and activity related shin and calf pain was observed. He reported continued discomfort at rest, but of a minimal intensity. Additionally, full range of active supination and dorsiflexion was restored in the standing position with the knees completely extended. The findings in this case report describe a common cause of shin pain. It highlights a novel and easily administered method of evaluating limited mobility in the talocrural joint and the relative inability to supinate the foot that could potentially contribute to symptomatology. Further research to examine the diagnostic utility of this method is recommended.

Key Words: leg pain, pronation, reversal, supination, ASDT

# Introduction

Lower leg, shin and calf pain are frequently encountered in rehabilitation settings. It is a condition prevalent in both active and sedentary individuals (1,2). Individuals presenting to a primary care setting with lower leg, shin and calf pain are evaluated for the possible presence of blood clots (3), chronic exertional compartment syndrome (CECS) (4), stress fractures (5), and infrequently malignancy (6). Other causes for lower leg and shin pain described in the literature are medial tibial stress syndrome (MTSS), popliteal artery entrapment syndrome (PAES) (7,8,9,10), peripheral neuropathy, lumbarradiculopathy, spinal stenosis (9,10), dehydration (11), vitamin B12 and vitamin D deficiency (12,13), exertional rhabdomyolysis (14),

restless leg syndrome (15), varicosities (16), congestive cardiac failure (17) and renal pathology (18).

The most common etiology for mechanical lower leg pain is extended periods of running (1,7,8) and prolonged periods of standing and walking (19,20,21). Although a wide range of diagnoses exist for mechanical and activity-induced leg pain, MTSS, CECS, stress fracture, nerve entrapment, and PAES are the most common (1). Often, more than one of these diagnoses co-exist as in MTSS and CECS alongside ankle and foot pain, owing to similarities in their intrinsic and extrinsic risk or aggravating factors (22,23).

The main aggravator described is the altered foot mechanics during the stance phase of gait. Deviations in normal foot and ankle mechanics have been described as causes for mechanical shin, leg, and calfpain (24,25,20). Literature describes a correlation between lower leg, shin and calf pain and hyper pronation. In a study that prospectively examined the gait-related risk factors for exerciserelated lower leg pain (ERLLP) in a young, physically active population, subjects showed a significantly increased pronation, accompanied by more pressure underneath the medial side of the foot (20,24,25). These studies describe pronation as increased ankle plantarflexion from mid-stance to toe-off and less ankle inversion at the end of stance and early swing phases. These two features favor the presence of prolonged pronation and the inability to reverse into supination to offer a rigid lever for push off (26). Persistent rearfoot plantarflexion during gait increased demand on the anterior compartment musculature during ambulation, resulting in pain and dysfunction. (20, 27). For much of the diagnoses of lower leg pain described above, the management and reversal of prolonged pronation of the foot during the stance phase of gait has been advocated, with favorable outcomes (28,29). The reversal entailed dorsiflexion of the talocrural and inversion of the subtalar joints, to elevate the medial longitudinal arch (27, 53,54).

Evaluation methods described in the literature to identify pronation have been both qualitative and quantitative. navicular drop, subtalar neutral, windlass test, observing for the presence of a flattened medial longitudinal arch and gastrocnemius tightness associated with limited dorsiflexion and are some of them (30,31, 32). While a single method of assessment may not improve the statistical likelihood of the presence of pronation, a cluster of findings may offer more validity. This case report suggests a novel and easily administered dynamic clinical test that may be added to the cluster, the active supination dorsiflexiontest (ASDT). It is proposed that the ASDT may be sensitive to the patho-mechanical challenges that occur during the stance phase of gait in the presence of prolonged pronation or in the absence of reversal of pronation. The test aims to capture the inability of the foot to be able to reverse into supination in preparation for propulsion (27,53,54).



Figure. 1

The Active supination Dorsiflexion Test

- a. Starting neutral position
- **b.** Intrinsic activation
- c. Inversion and dorsiflexion with knee extended

While pronation has been described to cause lower leg symptoms, studies have suggested that muscle imbalance between the invertors and evertors, in the absence of prolonged pronation, also cause similar symptoms (33). MTSS may occur without pronation indicators like medial longitudinal arch deformation or navicular drop. In such cases, one of the causes described is the strength disbalance of the invertor and evertor muscles in favor of the evertor muscles. It may be of value to know that inversion is a component of supination and a lack thereof can favor dysfunctional pronation (33). Additionally, the ankle invertor muscles are also dorsiflexors of the ankle and lack of dorsiflexion has also been described as a component of dysfunctional pronation (34,35,38). Thus, this case report aims to highlight the ability of contractile structures to be able to initiate the windlass mechanism and the reversal of pronation, namely, the foot intrinsics, invertors and dorsiflexors of the foot and ankle (26). Optimal strength of these muscles is essential for the normal functioning of the foot and ankle.

#### **Case Description**

### **History and Clinical Findings**

A 66-year-old retired male who experienced symptoms of right sided shin and calf pain, is presented. The pain was reported to have started gradually over an 8- month period, following bouts of regular 3-5 mile walks and light running. Symptoms began towards the end of his walking session and lingered for a few hours. He also reported pain and twitching during the nighttime disrupting sleep. He worked as a salesman and has been retired for 3 years. He reported his symptoms to his primary care physician. He had a doppler study (36) and an x-ray which revealed negative findings. His blood profile was unremarkable with normal findings in electrolyte values (11). He was a recovering alcoholic and was on anti- depressants. He had no other relevant medical history.

Activity especially extended periods of standing and walking typically increased his pain intensity. He reported a relative decrease in symptom intensity with rest and occasionally felt no pain. His pain intensity was 7-8/10 on the Numerical Pain Rating Scale (37). at its worse and 2/10 at best. His primary care physician diagnosed his condition as restless leg syndrome and muscle pain and prescribed pain medication. He was also referred for physical rehabilitation.

## Testing

The patient was independently ambulant and did not exhibit an antalgic gait. In standing, he presented with a complete loss of his medial longitudinal arch bilaterally. In the standing position, on observation from the posterior aspect he presented with calcaneal eversion. On observation there was no swelling or discoloration of the lower leg with intact dorsalis pedis and posterior tibial pulsation. He reported comparable discomfort and pain over the anterior and lateral shin area and over the mid-calf region. He also reported comparable pain over the medial tibial region. Passive dorsiflexion was limited at 10 degrees on the right side. Active inversion was also terminally restricted on the right side. Plantarflexion and eversion were full and free.

In the standing position the navicular drop test was performed (30). The distance from the navicular to the ground was measured in a subtalar neutral position and again after the patient was permitted full weight bearing. There was a navicular drop of nearly 2 cm indicating moderate pronation. The windlass test was performed as follows. In the standing position the first metatarsophalangeal joint was extended while allowing the interphalangeal joint to flex. There was no elevation of the medial longitudinal arch with no reproduction of pain in the foot (31). At this time, the ASDT (Fig. 1) was performed with the following operant definition. In the standing position with the knees completely extended, the patient was asked to curl his toes inwards to raise the medial longitudinal arch and invert and dorsiflex the ankle and foot. Knee extension was emphasized to maintain tension of the gastrocnemius which challenged dorsiflexion. He presented with difficulty curling his toes, inverting, and dorsiflexing the foot. Range of motion on active dorsiflexion was 5 degrees on the right and 10 degrees on the left side. Gait assessment revealed the persistence of a flattened medial longitudinal arch throughout stance and into the propulsion phase of gait.

The overall assessment thus was suggestive of a chronically pronated ankle and foot. In the absence of non-mechanical pathology, the presence of a chronically pronated foot has been shown to correlate to lower leg pain (37). Additionally, the presence of reproducible anterior and medial

shin and calf tenderness made the findings more correlative.

#### **Evaluation, Diagnosis, and Prognosis**

The differential screening process for the current pain presentation recommended ruling out the possibility of a stress fracture, blood clot, compartment syndrome and electrolyte imbalance. The patient had undergone a detailed medical evaluation prior to being referred to physical therapy. This included a visceral screen, blood chemistry analysis, x-rays and doppler study. He was assured that the findings were negative and suggested that the presentation was musculoskeletal requiring the expertise of a physical therapist.

Given the above findings it was concluded that the patient presented with medial and anterior tibial stress syndrome with mechanical somatic dysfunction of the talocrural and subtalar articulations. The mechanical dysfunction presented as chronic pronation extending into the late and terminal stance phase of gait. This resulted in overuse of the decelerators of pronation namely gastrocnemius, soleus, anterior tibial group of muscles, and the tibialis posterior, causing leg pain and dysfunction (37,38,39,40,27).

Based on the clinical findings, the patients age, activity level and general health, it was anticipated that the patient would respond well to treatment and regain normal physiological function and return to pain free participation of his work and leisure activities. The clinical information presented in this case report was in full adherence to the case reporting guidelines set forth by the Henry Ford Health System, Detroit. Michigan.

## **Intervention:**

Treatment addressed mechanical dysfunction at the right ankle and lower leg comprising manual therapy in the form of joint mobilization to the talocrural and subtalar joints. The intent was to improve dorsiflexion of the talus as a reversal for its plantarflexed position which was favoring pronation (41,42,43). This was followed by soft tissue mobilization of the lower leg region addressing the muscles in the anterior and posterior tibial compartments. The intent was to improve blood supply, heating and plasticity of the tissues, and facilitate synthesis and realignment of new collagen (41). Exercise prescription began with the patient being instructed in calf stretching to encourage talus dorsiflexion to revert pronation. Care was taken to maintain a neutral supinated position of the foot and ankle to prevent a mid-tarsal over a talocrural stretch (44). This was followed by open kinetic chain exercises done actively and with resistance bands (Fig.4) targeting the anterior tibial group comprising dorsiflexion and inversion and posterior tibial group comprising plantarflexion and inversion. Both concentric and eccentric contractions were performed. This was followed by active exercises for the intrinsic foot muscles comprising toe curls over a towel to encourage elevation of the medial longitudinal arch and reversal of supination (Fig. 2). Treatments were concluded with a cold pack over the lower leg region, mainly as an adjunct for its short term analgesic effect (40).

He was wearing a custom-made foot orthotic which controlled pronation by elevating the medial longitudinal arch (46), prescribed by his physician. He mentioned however, that he did not wear it while at home. He was instructed to wear his orthotic during all upright postures, including at home and was instructed on a home program to continue his exercises on the days he did not attend therapy sessions. The program was progressed in intensity during the third week until the end of the program at 4 weeks. Ankle and foot strengthening exercises were progressed to a weight bearing closed kinetic chain position. A spring-loaded foot exerciser was used for strengthening the intrinsic foot muscles (Fig. 3) (courtesy Elgin). Additionally, he stood over a padded rubber surface

and performed the same intrinsic foot exercises with the resistance offered by the padded rubber surface (47,48) to encourage closed kinetic chain concentric reversal of supination. This was progressed to one legged heel raises with more weight bearing over the lateral border of the forefoot. He also performed hip abduction and extension in standing with elastic bands of a light resistance, for the gluteus medius and maximus. Weak gluteal muscles contribute to the dynamic valgus on one legged stance, further contributing to valgus and pronation of the foot (49).



# Figure. 2

Intrinsic activation done in sitting progressed to closed kinetic chain on a padded surface



Figure. 3

# Spring loaded resisted intrinsic training



Figure. 4

Open chain concentric and eccentric training for anterior tibial group with resistance band

### Outcomes:

At the end of the fourth week, on completion of 8 treatment sessions, the patient reported a decrease in the intensity of pain to 2/10 at its worse and 0/10 at best on the NPRS scale with negligible symptom reproduction on local pressure over the shin and calf area. He exhibited improved and full range of motion in passive and active dorsiflexion in supine lying He reported negligible nighttime pain with twitching on and off. He was functioning with almost no discomfort during routine activities of daily living including his routine walks. He planned to resume running during the springtime. His lower extremity functional scale (LEFS) score was 78/80 and his Global Rating Of Change (GROC) score was 5+ (A good deal better). The LEFS and GROC are considered reliable self-report measures for pain and function (50,51). A retest of the ASDT was performed. In the standing position with the knees completely extended, the patient was asked to curl his toes inwards to raise the medial longitudinal arch and invert and dorsiflex the foot and ankle. He presented with no difficulty curling his toes and inverting and dorsiflexing the foot. Dorsiflexion range exhibited on the right side was 15 degrees. Passive and active inversion was full and free.

# Discussion and defense of the 'description' of the test:

The ankle and foot are made up of 26 bones and divided into 3 regions, the rearfoot, midfoot and forefoot (27). The well described muscles controlling all movements of the ankle are arranged in compartments in the lower leg (27). Of interest is the mechanics of the ankle and foot during the gait cycle and the mostly overlooked role of the four layers of intrinsic muscles in the sole of the foot (52).

While the mechanics of the foot during the normal gait cycle is reviewed the following speculations can be put forth that may have a clinical relevance to this case report. Chronic pronation has been established as a potential contributing factor for lower leg ankle and foot pain (28,29,19,20,24,25). The inability of the foot to reverse into supination by increasing the height of the medial longitudinal arch (Fig.5) may be the key component that needs to be addressed, by

therapeutic intervention. However, a dynamic test assessing this reversal ability as opposed to a static test that just assesses a navicular drop position or a flattened medial longitudinal arch, may be required. In other words, while a static pronated foot can be identified by observation, observing its dynamic ability to be able to raise the medial arch, invert and supinate may also be of value. Pronation is described as a loose packed position that occurs from heel strike to mid and early late stance when there is deformation of the medial longitudinal arch for shock absorption, adaptation of ground terrain changes, and maintenance of equilibrium. In the gait cycle, during heel contact (53,54,27), the talus is described to be in a position of plantarflexion as the foot drops to the ground while being controlled eccentrically by the anterior tibial group. As the foot progresses to mid stance, the eccentrically controlled plantarflexion continues while the talus adducts with calcaneal eversion to drop the medial longitudinal arch. The midtarsal joint, which consists of the talonavicular and the calcaneal cuboid articulations, unlocks with subtalar joint pronation bringing the cuboid and the navicular become more parallel. This drops the medial longitudinal arch further, allowing the forefoot to become a loose packed structure (27,53,54). Additionally, there is an anterior shearing force of the tibia on the talus which is decelerated mainly by the gastrocnemius and soleus muscle groups.

An optimal and timely reversal is hence essential to prevent prolongation of pronation and possible overuse. This is the phase where the lower leg muscles may work in excess if pronation is prolonged, resulting in pain and dysfunction.

At the late phase of stance supination begins as It enables the foot to transform into a rigid lever from which to push off. Two aspects are required in a closed chain capacity for supination to be initiated, one being elevation of the loose packed medial longitudinal arch and a more lateral position of the rear foot comprising the talus and the calcaneus (53,54,27). The literature describes the components of reversal poorly but supports the fact that elevation of the medial arch is assisted and speculated as initiated by the foot intrinsic muscles (55). The relative dorsiflexion, abduction of the talus combined with calcaneal inversion that follows, creates a more lateral position of the rear

foot completing the process of reversal into supination (53,54,55). The ASDT aims to identify the ability of a pronated foot to perform this reversal as an active clinical test.

Figure.5. Reversal of pronation to supination during stance phase of gait.



Pronation at mid stance



Intrinsic activity combined with supination at late stance

The plantar intrinsic foot muscles play a crucial role in supporting the medial longitudinal arch, providing the foot stability and flexibility for shock absorption. These muscles also can eccentrically sustain foot pronation for shock absorption and reverse pronation by increasing the height of the medial longitudinal arch during terminal stance (26). Researchers tested and described that the medial longitudinal arch can deform under increasing load, producing stretch of the plantar intrinsic foot muscles (Abductor Hallucis, Flexor Digitorum Brevis and Quadratus Plantae) and an increase in involuntary activity favoring shock absorption. These muscles were then capable of generating sufficient forces to attenuate medial longitudinal arch deformation produced by the load, and reverse the process by effectively increasing medial longitudinal arch stiffness. Activation of these muscles with load and their ability to generate sufficient force to counter medial longitudinal arch deformation may have important implications for how the foot can first absorb and then generate energy for efficient push off during gait (26, 45).

While this is a normal process the pathomechanical situation is when this reversal of supination does not occur to a point where push off occurs in a pronated loose packed position. This is what is described dysfunctional pronation and a contributor to multiple pain syndromes in the ankle foot and lower leg (20,24,25). This reversal of pronation and lack thereof is what is targeted mostly during rehabilitation intervention (56). Chronic fatigue of the decelerators of pronation in the anterior and posterior compartments and the persistent eversion sustained by the lateral compartment (52) are described as the potential mediators of pain in the lower leg.

In conclusion, a chronically pronated foot that poses difficulty with reversal of supination may exhibit the following mechanical challenges. The intrinsic foot muscles do not have the ability to effectively raise the medial longitudinal arch as an initiation of the reversal process. This results in the talus being in a position of chronic plantarflexion and adduction favoring tightness of the gastrocnemius and soleus with subsequent difficulty to dorsiflex the ankle. The ASDT aims to
capture this dysfunctional situation by making the individual actively supinate the foot by raising the medial longitudinal arch and performing inversion and dorsiflexion of the ankle in an upright standing posture.

### Conclusion

This case report describes a common cause for lower leg pain and the benefits of physical rehabilitation it its management. Prolonged pronation during the stance phase of gait, is described as a causative factor for pain syndromes in the lower leg (20,24,25). There is anatomical evidence to support the role of the intrinsic muscles of the foot and subtalar mobility in their ability to reverse pronation to supination during normal gait (53,54,55,27,26). While a chronically pronated foot has difficulty initiating this reversal process, a novel clinical test to identify this reversal difficulty, has been presented. It may be utilized in a screening cluster to identify the presence of dysfunctional pronation. Additionally, it may also be used to assess treatment outcomes. Future research to investigate its diagnostic utility is recommended.

# **Bibliography:**

1. Brewer RB, Gregory A. Chronic Lower Leg Pain in Athletes. A Guide for the Differential Diagnosis, Evaluation, and Treatment. Sports Health. 2012; 4(2): 121–127.

2. Breno FQ et al. Factors associated with sedentary behavior in patients with intermittent claudication. Eur J Vasc Endovasc Surg. 2016 Dec; 52(6): 809–814.

3. Stone J et al. Deep vein thrombosis: pathogenesis, diagnosis, and medical management. Cardiovasc Diagn Ther. 2017; 7(Suppl 3): 276–284.

4. Rajasekaran S, Finnoff JT. Exertional Leg Pain. Phys Med Rehabil Clin N Am. 2016;27(1):91-119.

5. Hetsroni I et al. The role of foot pronation in the development of femoral and tibial stress fractures: a prospective biomechanical study. Clin J Sport Med. 2008;18(1):18-23.

6. Chai T, Suleiman ZA, Roldan CJ. Unilateral Lower Extremity Pain Due to Malignancy Managed With Cordotomy: A Case Report. PM R. 2018;10(4):442-445.

7. Reinking MF, Austin TM, Richter RR, Krieger MM, Medial Tibial Stress Syndrome in Active Individuals: A Systematic Review and Meta-analysis of Risk Factors. Sports Health. 2017; 9(3): 252–261.

8. Reinking MF. Exercise Related Leg Pain (ERLP): a Review of The Literature. N Am J Sports Phys Ther. 2007; 2(3): 170–180.

9. Benhissen Z, Benaim C. Leg pain : differential diagnosis and treatment. Rev Med Suisse. 2019;15(635):216-220.

10. Mohile N. Chronic Lower Leg Pain in Athletes: Overview of Presentation and Management. HSS J. 2020 Feb;16(1):86-100.

11. Maughan RJ, Shirreffs SM. Muscle Cramping During Exercise: Causes, Solutions, and Questions Remaining. Sports Med. 2019;49(Suppl 2):115-124.

12. Jones A. Hansen K. Recognizing the musculoskeletal manifestations of vitamin D deficiency. J Musculoskelet Med. 2009; 26(10): 389–396.

13. Leishear K. The Relationship of Vitamin B12 and Sensory and Motor Peripheral Nerve Function in Older Adults. J Am Geriatr Soc. 2012; 60(6): 1057–1063.

14. Gardecki J. A case of exercise induced rhabdomyolysis from calf raises. World J Emerg Med. 2017; 8(3): 228–230.

15. Vellieux G, d'Ortho MP. Restless legs syndrome. Rev Med Interne. 2020;41(4):258-264.

16. Rezaie ES, Maas M, van der Horst CMAM. Episodes of extreme lower leg pain caused by intraosseous varicose veins. BMJ Case Rep. 2018: 2017-223986.

17 Goebel JR. Heart Failure: The Hidden Problem of Pain. J Pain Symptom Manage. 2009; 38(5): 698–707.

18. Arnold R. Neurological complications in chronic kidney disease. JRSM Cardiovasc Dis. 2016; 5: 2048004016677687.

19. Werner RA, Gell N, Hartigan A, Wiggerman N, Keyserling WM. Risk factors for plantar fasciitis among assembly plant workers. PM R. 2010;2(2):110-6.

20. Roberts A. Biomechanical differences between cases with chronic exertional compartment syndrome and asymptomatic controls during walking and marching gait. Gait Posture. 2017;58:66-71.

21. Kortebein PM, Kaufman KR, Basford JR, Stuart MJ. Medial tibial stress syndrome. Med Sci Sports Exerc. 2000;32(suppl 3):27-33.

22. Newman P. Risk factors associated with medial tibial stress syndrome in runners: a systematic review and meta-analysis. Open Access J Sports Med. 2013; 4: 229–241.

23. Beeson P. Plantar fasciopathy: revisiting the risk factors. Foot Ankle Surg. 2014;20(3):160-5.

24. Willems TM. A prospective study of gait related risk factors for exercise-related lower leg pain. Gait Posture. 2006;23(1):91-8.

25. Willems TM, Witvrouw E, De Cock A, De Clercq D. Gait-related risk factors for exercise-related lower-leg pain during shod running. Med Sci Sports Exerc. 2007;39(2):330-9.

26. Kelly LA. Intrinsic foot muscles have the capacity to control deformation of the longitudinal arch. J R Soc Interface. 2014; 11(93): 20131188.

27. Brockett C, Chapman GJ. Biomechanics of the ankle. Orthop Trauma. 2016; 30(3): 232–238.

28. Kim EK, Kim JS. J Phys Ther Sci. 2016; 28(11): 3136–3139.

29. Rachmawati MR. Correcting of pronated feet reduce skeletal muscle injury in young women with biomechanical abnormalities. Anat Cell Biol. 2016; 49(1): 15–20.

30. Spörndly-Nees S. The navicular position test- A reliable measure of the navicular bone position during rest and loading. Int J Sports Phys Ther. 2011; 6(3): 199–205.

31. Alshami AM, Babri AS, Souvlis T, Coppieters MW. Biomechanical evaluation of two clinical tests for plantar heel pain: the dorsiflexion-eversion test for tarsal tunnel syndrome and the windlass test for plantar fasciitis. Foot Ankle Int. 2007;28(4):499-505.

32. A M Picciano , M S Rowlands, T Worrell. Reliability of Open and Closed Kinetic Chain Subtalar Joint Neutral Positions and Navicular Drop Test. 1993;18(4):553-8.
33. Yüksel O. Inversion/Eversion Strength Dysbalance in Patients With Medial Tibial Stress Syndrome. J Sports Sci Med. 2011; 10(4): 737–742.

34. T.Shiroshita . Relationship between the medial longitudinal arch, foot dorsiflexion range of motion, and dynamic gait parameters. 2018; 61: 444-445

35. Johanson M. Subtalar Joint Position During Gastrocnemius Stretching and Ankle Dorsiflexion Range of Motion. J Athl Train. 2008; 43(2): 172–178.

36. Swanson E. Doppler ultrasound imaging for detection of deep vein thrombosis in plastic surgery outpatients: a prospective controlled study. Aesthet Surg J. 2015;35(2):204-14.

37. Ferreira-Valente MA, Pais-Ribeiro JL, Jensen MP. Validity of Four Pain Intensity Rating Scales. 2011;152(10):2399-404.

38. Amis J. The Split-Second Effect: The Mechanism of How Equinus Can Damage the Human Foot and Ankle. Front Surg. 2016; 3: 38.

39 Hintermann B, Knupp M. Injuries and dysfunction of the posterior tibial tendon. Orthopade. 2010;39(12):1148-57.

40. Pietrzak M. Diagnosis and management of acute medial tibial stress syndrome in a 15 year old female surf life-saving competitor. Int J Sports Phys Ther. 2014; 9(4): 525–539.

41. Kim J, Sung DJ, Lee J. Therapeutic effectiveness of instrument-assisted soft tissue mobilization for soft tissue injury: mechanisms and practical application. J Exerc Rehabil. 2017; 13(1): 12–22.

42. Schulze C, Finze S, Bader R, Lison A. Treatment of Medial Tibial Stress Syndrome according to the Fascial Distortion Model: A Prospective Case Control Study. Scientific World Journal. 2014; 790626.

43. Hawson ST. Physical Therapy and Rehabilitation of the Foot and Ankle in the Athlete. Clinics in podiatric medicine and surgery. 2011; 28 (1): 189-201.

44. Young R, Nix S, Wholohan A, Bradhurst R, Reed L. Interventions for increasing ankle joint dorsiflexion: a systematic review and meta-analysis. J Foot Ankle Res. 2013; 6: 46. J

45. Tosovic D, Ghebremedhin E, Glen C, Gorelick M, Brown JM. 2012. The architecture and contraction time of intrinsic foot muscles. J. Electromyogr. Kinesiol. 22, 930–938

46. Seo KC, PhD, Park SW, Park KY. Impact of wearing a functional foot orthotic on the ankle joint angle of frontal surface of young adults with flatfoot. J Phys Ther Sci. 2017; 29(5): 819–821.

47. Sulowska I, Mika A, Oleksy L, Stolarczyk A. The Influence of Plantar Short Foot Muscle Exercises on the Lower Extremity Muscle Strength and Power in Proximal Segments of the Kinematic Chain in Long-Distance Runners. Biomed Res Int. 2019; 6947273.

48. Sulowska I et al. The Influence of Plantar Short Foot Muscle Exercises on Foot Posture and Fundamental Movement Patterns in Long-Distance Runners, a Non-Randomized, Non-Blinded Clinical Trial. PLoS One. 2016; 11(6): 0157917.

49. Galbraith MR, Lavallee ME. Medial tibial stress syndrome: conservative treatment options.
Current Reviews in Musculoskeletal Medicine. 2009; 2: 127–133.
49.

50. Kamper SJ, Maher CG, Mackay G. Global Rating of Change Scales: A Review of Strengths and Weaknesses and Considerations for Design. J Man Manip Ther. 2009; 17(3): 163–170.

51. Yeung TSM, Wessel J, Stratford P, Macdermid J. Reliability, Validity, and Responsiveness of the Lower Extremity Functional Scale for Inpatients of an Orthopaedic Rehabilitation Ward. J Orthop Sports Phys Ther. 2009;39(6):468-77.

52. Bavdek R, Zdolšek A, Strojnik V, Dolenec A. Peroneal muscle activity during different types of walking. J Foot Ankle Res. 2018; 11: 50.

53. Donatelli R. Normal Biomechanics of the Foot and Ankle. J Orthop Sports Phys Ther. 1985; 7(3):91-5.

54. Donatelli R. Abnormal Biomechanics of the Foot and Ankle. J Orthop Sports Phys Ther. 1987;9(1):11-16

55. Ferber R, Hreljac A, Kendall KD. Suspected mechanisms in the cause of overuse running injuries: a clinical review. Sports Health. 2009; 1(3):242-6.

56. Chinn L, Hertel J. Rehabilitation of Ankle and Foot Injuries in Athletes. Clin Sports Med. 2010; 29(1): 157–167.

# PHASE 2: REPRODUCIBILITY (RELIABILITY)

# SCAPULA BACKWARD TIPPING TEST

ORIGINAL DESCRIPTION: Sebastian D, et al. The scapula backward tipping test: An inter-rater reliability study. Journal of Bodywork & Movement Therapies. 2017; 21: 69-73.

### **Conceptual basis of the second phase (reliability) of clinical test development:**

The next phase of clinical test development is to investigate if a clinical test is reproducible between blinded examiners. In other words, the test can present consistently in the presence of a clinical problem and identified by multiple observers consistently. As an example, if 5 experienced orthopedists observed an x-ray of a fractured humerus simultaneously without discussion (blinded). They would all agree that there is a fracture of the humerus based on the x-ray finding. Credit may be directed towards the instrument for having made the fracture of the humerus accurately available to several clinicians simultaneously to establish a consistent finding between them of a certain diagnosis. This consistency in methodological research is termed reliability and one can hypothesize then that an x-ray is a reliable test to identify the presence of a fractured humerus. On a similar note the scapula backward tipping test (SBTT) was described as a clinical test to identify the presence of stubborn forward tipping or protraction of the scapula which is a detriment to optimal functional outcomes in the shoulder. The following is an original research example conducted by me (PhD candidate) that describes how reliability is investigated. The relevant description of the hypothesis, design, methods, analysis and conclusion of reproducibility or reliability is as follows:

### THE SCAPULA BACKWARD TIPPING TEST

# Abstract

**Background & Purpose**: The purpose of this study was to determine the reliability and diagnostic utility of the scapula backward tipping test (SBTT) in detecting the presence of pectoralis minor (PM) tightness and subsequently scapula forward tipping, in a symptomatic population. PM tightness with scapula forward tipping has been described to cause pain and dysfunction in the shoulder region.

**Methods:** 30 patients with a diagnosis of shoulder pain were randomly assigned and examined by 2 musculoskeletal physical therapists at a time. The procedure consisted of having the individual lay on the stomach in a neutral head position with palms in the anatomical position. The examiner placed one hand on the inferior angle of the scapula and the fingers of the other hand hooked the under surface of the coracoid process. A gentle yet firm pull was imparted in an upward direction to sense tightness and to observe movement of the acromion up to the tragus of the ear. An allowance was made up to an inch above the tragus. A comparison was made with the asymptomatic side. Interrater reliability was determined using the kappa statistic. Sensitivity, specificity and likelihood ratios were calculated using a contingency table.

**Results:** The SBTT was found to be reproducible between examiners and sensitive to the symptomatic side being tested (Kappa=0.66, SE of kappa = 0.096, Sn= 92.5, Sp=75.5, +LR=3.8, -LR=0.10, 95% CI).

**Conclusion:** The SBTT may be incorporated as a simple yet effective test to determine the presence of PM tightness and subsequently scapula forward tipping. Additionally, it is postulated that a sustained test position may be a localized and effective stretch for the PM.

**Key Words:** special test, scapula, forward tipping, shoulder pain, reliability, diagnostic utility

# Introduction:

Mechanical dysfunction of the scapula has been described in patients presenting with shoulder, neck and thoracic pain and dysfunction (Cools AM et al, 2014). Authors have previously described the scapula to excessively protract, downwardly rotate, wing and forward tip during dysfunctional states (Ludewig PM and Reynolds JF, 2009). One structure contributing to this phenomenon, when tight, is the pectoralis minor (PM). It has been described to increase scapula protraction, forward tilting and downward rotation (Ludewig PM and Reynolds JF, 2009). Additionally, studies have demonstrated lengthening of the PM during scapula backward tilting and upward rotation (Muraki et al, 2009 ; Borstad and Ludewig, 2006). While the fact remains clear that PM tightness contributes to pain and dysfunction, a practically feasible method to dynamically assess its length, and presence of tightness, continues to remain a challenge. Few studies have attempted to demonstrate methods to assess PM length, with good reproducibility, however, either lacking specificity or requiring instrumentation (Struyf F et al, 2014; Lewis JS and Valentine RE, 2007). This study proposes a novel and practical method to assess PM length, the scapula backward tipping test (SBTT), which has demonstrated good reproducibility between experienced raters. Additionally, it has also demonstrated acceptable levels of sensitivity, reinforcing it's diagnostic utility.

While sensing the presence of tightness of the PM appears to be the objective, the challenge remains in establishing the presence of it's 'normal' length. The neutral head posture seems to provide a basis for a hypothetical 'normal length' of the pectoralis minor. A deviation from the neutral head posture, as in a forward head posture, has been described to contribute to upper quarter pain and dysfunction (Lewis JS, Green A and Wright C, 2005).

Studies report the forward head posture to co-exist with scapula downward rotation and forward tipping, secondary to a tight PM (Won-gyu Yoo, 2013; Weon JH et al, 2010). A neutral head posture may hence be postulated to be the optimal length of the PM.

A neutral head posture also requires an operant definition as the literature substantiating it is scarce. Studies describe the neutral head posture in reference to the forward head angle equal to or greater than 46° relative to the vertical line extending from C7 to the line connecting C7 to the tragus. Rounded shoulder position was described as having a forward shoulder angle of equal to or greater than 46° relative to the vertical line extending from C7 to the line connecting C7 to the acromion (Thigpen CA et al, 2010; Cole AK). Hence, for practical purposes, a neutral head posture, while performing a visual inspection, requires the tragus of the ear to line up directly over the acromion with the head aligned over a normal lordosis, directly over the thorax. Clinicians (Sahrman S, 2001 ; Lewis JS and Valentine RE, 2007) may have used this as a precedent to assess normal PM length. They have described a PM rest length normal when the acromion is an inch above the table in the supine lying position. This however does not make allowance dynamic PM length testing as the results were based on positional observational data. The SBTT describes a starting position analogous to a neutral head posture, and proposes normal PM length to be a movement of the acromion to be in line with or up to one inch above the tragus of the ear, in the prone lying position. It claims a dynamic and observational method of assessing PM length in addition to a qualitative end feel of tightness in comparison to the other side.

### Methods:

Three orthopaedic board certified and orthopaedic manual therapy fellowship trained physical therapists, with 23 years of experience in orthopaedic physical therapy, conducted a pilot training on patients with shoulder pain. The intention was to first familiarize the methodology of performing the SBTT and subsequently establish an operant definition. The procedure consists of having the individual lay on the stomach in a neutral head position with palms in the anatomical position. The clinician placed one hand on the inferior angle of the scapula and the fingers of the other hand hooked the under surface of the coracoid process. With a firm inferiorly directed stabilizing force on the inferior angle of the scapula, a gentle pull is imparted in an upward direction (Figs 1&2) to sense tightness and to observe movement of the tragus of the ear for up to, and an inch above the acromion. Care was taken to not hook the fingers under the clavicle as this would place a stretch on the acromioclavicular joint and not the pectoralis minor. A statistical analysis was not done for the pilot training as the intent was to simply understand the methodology for consistency of performing the test, prior to conducting the study. Once the 'operant definition' for consistency of performance was clearly understood by the participating clinicians, the study proceeded.





Fig 2. Hand placements and direction to perform the scapula backward tipping test



Hand placements to perform the scapula backward tipping test

Upon the approval of the 5 member Institutional Review Board of the Institute of Therapeutic Sciences, 30 patients with a diagnosis of shoulder pain were randomly assigned and independently examined by 2 musculoskeletal physical therapists at a time, in order to determine the presence PM tightness. All patients that were referred to the practice with a shoulder diagnosis or complained of shoulder pain, who signed the informed consent, were tested. Both symptomatic and asymptomatic sides were tested, to determine sensitivity and specificity, hence technically 60 shoulders (30 symptomatic and 30 asymptomatic) were examined by a blinded clinician. Three orthopaedic board certified and orthopaedic manual therapy fellowship trained physical therapists, with 23 years of experience in orthopaedic physical therapy, participated in the study. The test was performed and recorded simultaneously by two clinicians at a time. The clinicians were considered to be in agreement when they agreed individually on the side in which the tightness was elicited. Additionally, if the tightness was elicited on the symptomatic side as opposed to the asymptomatic side, it was considered sensitive. The number of false positives were also recorded, to determine specificity.

#### **Results:**

Inter-rater reliability was determined using the kappa statistic. Since the scale for measurement was nominal, with no levels of seriousness of agreement or disagreement, the Cohen's un-weighted kappa statistic was used (Hallgren, 2012). The software used was the GraphPad Software, Inc., La Jolla, CA. For the SBTT. Inter-rater reliability was 'good' (k=0.667, SE of kappa = 0.096, 95% confidence interval), with a percentage agreement of 83.3%. (Table 1).

# **Reliability evaluation:**

- AA: Both examiners identified positive
- BB: Both examiners identified negative
- AB: A identified positive and B identified negative
- BA: B identified positive and A identified negative

### Table 1

	A	В	
A	25	8	33
В	2	25	27
Total	27	33	60

Number of observed agreements: 50 (83.33% of the observations)

Number of agreements expected by chance: 29.7 (49.50% of the observations)

Kappa= 0.670

SE of kappa = 0.093

95% confidence interval: From 0.487 to 0.853

The strength of agreement is considered to be 'good'.

### Discussion and defense of the 'reliability' of the test:

Studies reporting the prevalence of PM tightness are scarce, however, the prevalence of a forward head posture, which is documented to co-exist with a forward tipped and a downwardly rotated scapula has been documented (Thigpen CA et al, 2010). Studies have reported greater forward head posture in patients with shoulder conditions than in healthy control participants (Greenfield et al, 1995). Patients with preexisting forward head and rounded shoulder postures exhibited greater anterior tilt and upward rotation of the scapula during flexion motions at the shoulder (Thigpen CA et al, 2010). Acutely, adopting a forward head rounded shoulder posture also creates increased scapular anterior tilt and upward rotation (Finley MA and Lee RY, 2003). An association has been reported between forward head and rounded shoulder postures and reports of shoulder or scapular pain (Griegel-Morris et al, 1992). The need for testing altered scapula mechanics, mediated by a tight PM, is hence obvious. To establish the diagnostic utility of a clinical test, a gold standard reference is the norm. Investigators (Sahrmann S, 2001; Lewis JS and Valentine RE, 2007; Struyf et al, 2014) have previously described methods to determine PM tightness and coincidingly all descriptions have been in the supine position. At present there is no gold standard reference test for the measurement of PM length in the prone position. The operant definition was determined by 3 orthopaedic board certified and orthopaedic manual therapy fellowship trained physical therapists, with 23 years of experience in orthopaedic physical therapy. With substantial experience in treating shoulder disorders and a detail review of literature of previously described testing procedures for PM length, a movement of the acromion up to and one inch past the tragus of the ear was considered normal excursion. It was also noted that in the symptomatic subjects who exhibited tightness on both sides, the 'symptomatic' side was to be considered as a positive finding.

This risk of asymptomatic individuals with PM tightness developing shoulder dysfunction at a later date requires more longitudinal studies, which is currently lacking in literature. As the prevalence of PM tightness has been documented in asymptomatic individuals (Struyf et al, 2014), the premise of testing the asymptomatic side of symptomatic patients rather than asymptomatic patients, is justified. The reason being, asymptomatic individuals may still test positive for the presence of PM tightness, challenging specificity. In any case, the benefits of lengthening the PM in the 'presence' of shoulder dysfunction (Kuhn JE, 2009) still offers positive substantiation as to the need to test it for the presence of tightness.

The results of this study offer positive insight as to the utility of the SBTT. The strength of agreement between examiners was found to be 'good', favoring the reproducibility and

reliability of the test. The diagnostic utility evaluation revealed high 'Sensitivity' and 'negative predictive values' and low 'negative likelihood ratio' values suggesting the ability of the SBTT to 'rule out' the presence of a dysfunction when tested negative. The therapeutic value of the SBTT as a localized stretching maneuver for the PM is indicated, however not validated. Further studies demonstrating its therapeutic ability to lengthen the PM, is warranted. Clinicians incorporating PM stretching for scapula forward tipping and downward rotation should understand that the optimal outcome occurs when the stretching maneuver is followed up with strengthening of the lower fibers of the trapezius and the serratus anterior (Huang TS et al, 2015; Park SY and Yoo WG, 2014).

The limitations of this study is twofold. The first lies in the defining a normal length of the PM as there are no validated studies describing its normal excursion. The assumption of a movement of the acromion up to and one inch past the tragus of the ear is based on empirical clinical observation made by experienced shoulder practitioners. Hence further studies determining an operant 'normal PM length', is warranted.

The determination of the presence of PM tightness lacks a gold standard, therefore the second limitation would be the assumption of a true positive. Owing to the association of PM tightness to shoulder pathology, the symptomatic side was considered the side positive, for the presence of PM tightness.

### **Conclusion:**

The SBTT may be incorporated to determine the presence of PM tightness in a symptomatic and asymptomatic population. The test position is novel and easily administered once the operant definition is fully understood. Additionally, it may be

indicated as a localized stretching procedure for the PM, for which further studies are warranted. The presence of a forward head and rounded shoulder posture has been correlated to PM tightness. Stretching the pectoralis minor has been suggested as an important treatment intervention for conditions caused secondary to forward head and rounded shoulder postures, which includes mechanical shoulder, neck and thoracic dysfunction. Clinicians treating shoulder, neck and thoracic dysfunction may hence benefit from the utility of the SBTT as a diagnostic as well as therapeutic modality.

### **Bibliography:**

- Ashley K Cole, Melanie L McGrath , Shana E Harrington, Darin A Padua, Terri J Rucinski, William E Prentice. Scapular Bracing and Alteration of Posture and Muscle Activity in Overhead Athletes With Poor Posture. J Athl Train. 2013 Jan-Feb;48(1):12-24
- Borstad JD, Ludewig PM. Comparison of three stretches for the pectoralis minor muscle. J Shoulder Elbow Surg. 2006 May-Jun;15(3):324-30.
- Cools AM, Struyf F, De Mey K, Maenhout A, Castelein B, Cagnie B. Rehabilitation of scapular dyskinesis: from the office worker to the elite overhead athlete. Br J Sports Med. 2014 Apr;48(8):692-7.
- Finley MA, Lee RY. Effect of sitting posture on 3-dimensional scapular kinematics measured by skin-mounted electromagnetic tracking sensors. Arch Phys Med Rehabil. 2003;84(4):563–568
- 5. Greenfield B, Catlin PA, Coats PW, et al. Posture in patients with shoulder overuse injuries and healthy individuals. JOSPT 1995;21:287-95.
- Griegel-Morris P, Larson K, Mueller-Klaus K, Oatis CA. Incidence of common postural abnormalities in the cervical shoulder, and thoracic regions and their association with pain in two age groups of healthy subjects. Phys Ther 1992;72:425-31.
- Huang TS, Ou HL, Huang CY, Lin JJ. Specific kinematics and associated muscle activation in individuals with scapular dyskinesis. J Shoulder Elbow Surg. 2015: 1058-2746(15)00008-7.

- Hallgren, K. A. (2012). Computing Inter-Rater Reliability for Observational Data: An Overview and Tutorial. Tutorials in Quantitative Methods for Psychology, 8(1), 23–34.
- John E. Kuhn . Exercise in the treatment of rotator cuff impingement: A systematic review and a synthesized evidence-based rehabilitation protocol. J Shoulder Elbow Surg. 2009 Jan-Feb;18(1):138-60.
- Lee JH, Cynn HS, Yoon TL, Ko CH, Choi WJ, Choi SA, Choi BS. The effect of scapular posterior tilt exercise, pectoralis minor stretching, and shoulder brace on scapular alignment and muscles activity in subjects with round-shoulder posture. J Electromyogr Kinesiol. 2015;25(1):107-14.
- 11. Lewis JS, Valentine RE. The pectoralis minor length test: a study of the intra-rater reliability and diagnostic accuracy in subjects with and without shoulder symptoms. BMC Musculoskeletal Disorders. 2007; 8:64
- Lewis JS, Green A, Wright C. Subacromial impingement syndrome: the role of posture and muscle imbalance. J Shoulder Elbow Surg. 2005 Jul-Aug;14(4):385-92.
- Ludewig PM, Reynolds JF. The Association of Scapular Kinematics and Glenohumeral Joint Pathologies. J Orthop Sports Phys Ther. 2009 Feb; 39(2): 90– 104.
- 14. Muraki T, Aoki M, Izumi T, Fujii M, Hidaka E, Miyamoto S. Lengthening of the pectoralis minor muscle during passive shoulder motions and stretching techniques: a

cadaveric biomechanical study. Phys Ther. 2009;89(4):333-41.

- 15. Park SY, Yoo WG. Activation of the serratus anterior and upper trapezius in a population with winged and tipped scapulae during push-up-plus and diagonal shoulder-elevation. J Back Musculoskelet Rehabil. 2014; Feb 20. [Epub ahead of print]
- Sahrman S. Diagnosis and treatment of movement impairment syndromes. 2001; Mosby, St Louis, MO.
- 17. Struyf F, Meeus M, Fransen E, Roussel N, Jansen N, Truijen S, Nijs J. Interrater and intrarater reliability of the pectoralis minor muscle length measurement in subjects with and without shoulder impingement symptoms. Man Ther. 2014 Aug;19(4):294-8.
- Thigpen CA, Padua DA, Michener LA. et al. Head and shoulder posture affect scapular mechanics and muscle activity in overhead tasks. J Electromyogr Kinesiol.2010; 20 (4):701–709.
- Van Stralen KJ, Stel VS, Reitsma JB, Dekker FW, Zoccali C, Jager KJ. Diagnostic methods I: sensitivity, specificity, and other measures of accuracy. Kidney Int. 2009 Jun; 75(12):1257-63.
- 20. Weon JH, Oh JS, Cynn HS, Kim YW, Kwon OY, Yi CH. Influence of forward head posture on scapular upward rotators during isometric shoulder flexion. J Bodyw MovTher. 2010;14(4):367–374.
- 21. Won-gyu Yoo. Comparison of Shoulder Muscles Activation for Shoulder Abduction between Forward Shoulder Posture and Asymptomatic Persons. J Phys Ther Sci. 2013 Jul; 25(7): 815–816.

# PHASE 3: VALIDATION FOR TRUTHFULLNESS (VALIDITY)

# THE SITTING ACTIVE AND PRONE PASSIVE LAG TEST: A VALIDITY STUDY IN A SYMPTOMATIC KNEE POPULATION

ORIGINAL DESCRIPTION: Sebastian D, et al. The sitting active and prone passive lag test: An inter-rater reliability study. Journal of Bodywork and Movement Therapies. 2014 Apr;18(2):204-9.

#### **Conceptual basis of the third phase (Validity) of clinical test development:**

The best analogy to test validity is an employee of an organization reporting to work regularly and on time, however, performs poorly within the expectations of his job description. In other words, he is a reliable employee but not valid as he does not do what he is supposed to do, efficiently. Reverting to the example in the reliability section where an x-ray identified a fractured humerus which was consistently observed by 5 different orthopedists who agreed it was a fracture humerus. Imagine a situation where the x-ray reveals a varus angulation instead of a fracture (when there truly is one), and this was consistently agreed upon by 5 different clinicians. This simply means that the x-ray is reliable (reproducible) but does not reveal the true problem, which is a matter of concern. Hence, to summarize the final phase, a test is created out of an experienced clinicians need to identify a disease or dysfunction. With adequate clinical support and justification, the test may be described (description) as a hypothetical benefit to identifying that particular disease or dysfunction. The test is then assessed for consistency or reproducibility between examiners (reliability), Once deemed consistent or reliable, it is assessed whether it does what it is supposed to do, which is accurately identifying the 'true' problem (validity). A useful test is thus born and when it performs to perfection over time can elevate itself to a reference standard or 'gold standard'. The following is an original research example conducted by me (PhD candidate) that describes how validity is investigated. The relevant description of the hypothesis, design, methods, analysis and conclusion of truthfulness or validity is as follows:

### SITTING ACTIVE AND PRONE PASSIVE LAG TEST

# Abstract

**Purpose:** To determine the diagnostic utility of the sitting active and prone passive lag test (SAPLT) in identifying terminal extension lag in symptomatic knees. The lack of full extension at the knee results in greater force of quadriceps activation, abnormal gait mechanics, overloading of the ipsilateral and contra-lateral joints, resulting in pain and dysfunction.

**Participants & Methods:** 54 knees (27 symptomatic and 27 asymptomatic) were randomly assigned and independently examined by 2 blinded examiners at a time, to determine the presence of an active or a passive extension lag at the knee using the SAPLT. Reproducibility of results between the 2 examiners was determined, for reliability. In addition, the presence of an extension lag in the symptomatic knee and its absence in the asymptomatic knee were determined for validity.

**Results:** For the SAPLT, the inter-rater reliability was 'almost perfect' (Kappa 0.92, SE of kappa 0.072, 95% confidence interval). Test sensitivity (Sn) was 1.0 and specificity (Sp) was 0.82. Positive and negative likelihood ratios (LR) were 5.5 and 0.00 respectively. Positive and negative predictive values (PPV) were 0.83, and 1.0 respectively with an overall accuracy of 90.74%.

**Conclusion:** The SAPLT may be incorporated as a reliable and valid test to determine the presence of terminal extension lag in a unilaterally symptomatic knee population.

Keywords: Diagnostic Utility, Active Lag, Passive Lag

### Introduction:

The knee is a mobile joint and the available mobility or range of motion (ROM) can determine its ability to engage in activities of daily living. ROM in the knee is a composite parameter comprising extension and flexion. With both combined the total knee ROM ranges from 0° extension to 140° flexion. Since limitations of knee ROM are observed during dysfunctional states its measurement and management has been an integral component of physical therapy practice (Hancock GH, Hepworth T and Wembridge K, 2018). Knee flexion has procured more attention than its counterpart extension, owing to its functional capabilities (Koc TA, 2019). More recently, the need for normal knee extension has been gaining importance (Kappetijn O, Trijffel EV and Lucas C, 2014) . The mechanical challenges and unfavorable consequences of its presence has made rehabilitation professionals target its restoration during routine clinical practice.

Rehabilitation professionals, having comprehended the importance of normal knee ROM, and have devised methods of evaluating its presence and quality. Although knee flexion has procured more attention, the lack of terminal knee extension and its consequences have not been completely ignored (Rice et al., 2011; Stillman, 2004; Shea et al., 2012; Adernito., 2005; Temelli and Akalan, 2009). Clinically they have been recognized as flexion deformity or extension lag with causes ranging from trauma, surgery, arthrogenic muscle inhibition (AMI), and neurological weakness (Runner RP., et al), (Koh et al., 2012; Onodera et al., 2012), (Rice and McNair, 2009), (Nyland et al., 2005; Chimera et al., 2012; Gupta et al., 2012), (Jefferson et al., 1990). Clinicians have recognized that a lack of terminal knee extension can contribute to overuse, increased energy expenditure, gait abnormalities, pain in the involved and adjacent joints and poor functional scores

((Goudie et al., 2011; Su 2012), (Hotfiel et al., 2012), (Rice et al., 2011; Vince et al., 2005; Gurney, 2002 ; Zalata, 2008). The presence and consequences of lack of terminal knee extension makes its identification and appropriate management a matter of extreme importance. The need to identify its presence warrants the need for a reliable and valid clinical test.

Despite knee extension lag being a common occurrence the description of assessment methods in the literature to identify its presence is remote. One study recommended assessing knee extension lag in the supine position with the ankle supported on a roll and the knee left unsupported. They then visually observed for a lack of terminal knee extension. Additionally, open kinetic chain terminal knee extension was performed in the supine position to visually observe for a lack in terminal knee extension (Stillman, 2004). Limitations of this description was that the influence of the hamstrings and gastrocnemius were excluded and a methodology to determine the diagnostic utility of the testing method was lacking. More recently the sitting active and prone passive lag test (SAPLT) was described and appears to address these limitations (Sebastian, Chovvath and Malladi, 2015). While the operant definition and reproducibility appeared convincing, the validity of this clinical test was not established. In other words, when a knee extension lag is presented the SAPLT should ideally be able to identify its presence on the symptomatic side and its absence on the asymptomatic side, consistently, establishing its truthfulness or validity.

The hallmark of clinical practice is to be able to diagnose a clinical ailment which then aids in its appropriate treatment. Multiple methods are incorporated in the diagnostic process, beginning with clinical signs and symptoms, followed by clinical testing in the form of hands-on procedures, biochemical lab analysis and imaging. While imaging and lab analysis are preferred methods, hands on clinical tests have continued to prevail in clinical practice alerting the clinician of issues that may not be identified by imaging or lab analysis. This especially applies to musculoskeletal movement disorders and are termed impairment-based tests to identify movement dysfunction (Rossi, 2011).

Clinical tests are required to be reliable or reproducible between examiners prior to the establishment of their diagnostic utility (Tweed and Wilkinson, 2012). A consistent and reproducible clinical test is still considered to be lacking in utility or usefulness if it does not solve a problem or address a purpose. This usefulness is termed validity and when this concept is applied to a clinical test, it simply means that the test should detect a clinical issue when present and not be able to do so when absent. The variables of interest used in the evaluation of clinical test validity are sensitivity (Sn), specificity (Sp), predictive values (PPV/NPV) and likelihood ratios (LR).

### **Participants and Methods:**

This study was a methodological design to develop test validity estimating parameters of relevance such as test reliability, sensitivity, specificity, predictive values, likelihood ratios, and receiver operating characteristic (ROC) curves of an individual impairment based diagnostic test. While the larger purpose of this study was to establish the validity and subsequently the diagnostic utility of the SAPLT, its ease of administration across different experience levels was also being brought to light. Hence, two orthopaedic board certified and orthopaedic manual therapy fellowship trained physical therapists, with 20+ years of experience in orthopaedic physical therapy, along with one general physical

therapist with 3 years of experience in orthopedic physical therapy participated in the testing process. They initially conducted a pilot training on unilaterally symptomatic knees. The intention was to first familiarize the methodology of performing the SAPLT and subsequently understand its operant definition. An active lag was determined by the inability of the erectly seated subject to actively extend the involved knee, with the ankle in maximum dorsiflexion, to the same level as the normal knee held in a full knee extension and ankle dorsiflexion. This was determined by the low position of the toes on the involved side. A passive lag was determined by placing the subject prone with the knees just past the edge of the table (Fig 1). With both legs fully extended and resting the high position of the heel compared to the heel on the normal side determined the presence of a passive lag (Fig.2).



Fig 1 .TEST POSITION FOR 'ACTIVE LAG' SHOWING POSITIVE 'ACTIVE LAG' ON THE RIGHT



Fig 2. TEST POSITION FOR 'PASSIVE LAG' SHOWING POSITIVE 'PASSIVE LAG' ON THE RIGHT

The differentiation between an active and a passive lag was considered mandatory as, when appropriately identified, the most appropriate management could be instituted. An active lag may indicate the need to address the contractile structures as in muscle strengthening, provided there are no passive restraints, while a passive lag may indicate the need to address tight restraints as in tightness of the posterior knee capsule, gastrocnemius, hamstrings, iliopsoas, followed by muscle strengthening. Although the intent of the pilot study was to simply understand the methodology for consistency of performing the test prior to conducting the study, a statistical analysis was also done for the pilot training. A total of 6 knees were assessed by the 3 testers. Results revealed a kappa score of 1.0 indicating the ability of the SAPLT to identify a positive test on the symptomatic side and a negative test on the asymptomatic side for all 6 knees. The pilot training, instituted among physical therapists with different levels of experience was to establish the user-friendly nature of

the test. Once the 'operant definition' for consistency of performance was clearly understood by all participating clinicians, the study proceeded.

Upon the approval of the Institutional Review Board of an accredited university, and a large health system, 27 symptomatic and 27 asymptomatic knees (n=54) were randomly assigned and independently examined by 2 blinded musculoskeletal physical therapists at a time, to determine the presence of an active or a passive, extension lag at the knee, or both. A power analysis was conducted using 2 software to determine sample size for proportions one sample, two-sided binomial testing for sensitivity and specificity. The results suggested a sample size of 25 on Statistical Package for Social Sciences (SPSS) and 34 on Power Analysis Sample Size (PASS). The researchers decided to maintain a sample size of 50. All patients that were referred to a large teaching health system, and an outpatient physical therapy clinic, with a diagnosis of unilateral knee pain or complained of unilateral knee pain, stiffness, or weakness, who signed the informed consent, were tested.

### **Measurement / Instrumentation**

The primary variable of interest was to see if the test appeared positive on the symptomatic knee and negative on the asymptomatic knee. The end point hence was the truthfulness or validity, in other words does the test measure what it is intended to measure. The SAPLT has been studied earlier for consistency and has been described as having 'good' reproducibility between experienced raters. While the operant definition and reproducibility appear convincing, the validity of this clinical test had not been established, which is the endpoint.

The measurement technique and the instrument were the informed clinicians with trained observation skills. The testers observed the positions of the toes and heels of the subject in the sitting and prone lying position and mark the 'high' position on the heels in the prone position and 'low' position on the toes in the sitting position, respectively, as a positive finding. The primary variable of interest and subsequently leading to the endpoint was to see if the 'high' and 'low' positions are present in the symptomatic knee and absent in the asymptomatic knee.

### **Testing procedure:**

The testing was carried out in the exact same manner as the pilot study. The diagnoses encountered in this study were knee osteoarthritis, patello-femoral dysfunction, ligament strains, meniscus tears, and total knee arthroplasty. All patients had a unilateral dysfunction, and the normal knee was used as a reference for normal knee extension range. Both active and passive lag were tested. As mentioned earlier, two orthopaedic board certified and orthopaedic manual therapy fellowship trained physical therapists, with 20+ years of experience in orthopaedic physical therapy, and one physical therapist with 3 years of experience in orthopaedic physical therapy, participated in the study. However, at a given time, only two raters tested. The 'assigner' was the physical therapist who positioned the subject in the test positions for the 2 blinded testers. The responsibility of the assigner was to position the subjects in the sitting and prone test positions and summon the raters for testing. The knees were draped, or the subject was allowed to have their exercise pants with shoes on, to blind the testers from signs of obvious swelling, redness or a surgical scar that may indicate the symptomatic side. The testers were not permitted into the testing area when the positions were changed. The test was performed once, and both clinicians

observed the results simultaneously mainly by the positions of the heels, and toes. They independently recorded their findings for both parts of the test on the recording sheet. The testers were blinded to the symptomatic knee and upon completion of the testing, the assigner marked the symptomatic side on the recording sheet for future reference and statistical analysis. The clinicians were considered to be in agreement when they agreed individually on the active and the passive components of the test, identified as positive or negative. The test was considered valid if the positive test that was identified consistently, was on the symptomatic side and if the negative test that was identified consistently, was on the asymptomatic side.

### **Data Analysis:**

Analysis were performed on IBM statistical package for social sciences (SPSS) (Pallant, 2020), version 28.0.0.0 (190), GraphPad kappa calculator. MedCalc statistical software and by utilizing mathematical formulas described in the next section to calculate values obtained from the contingency table.

### **Test Validity:**

The data collected consisted of True positives (TP), False positives (FP), False negatives (FN) and True negatives (TN), and were represented and calculated on a 2x2 contingency table (Hassanzadeh J and Rezaianzadeh A, 2012) (Table.1).

I able T		bl	е	1
----------	--	----	---	---

Test	Present	Absent	Total
+VE	ТР	FP	TP+FP
-VE	FN	TN	FN+TN
Total	TP+FN	FP+TN	Total

The parameters of relevance were Sensitivity, Specificity, Predictive Values and Likelihood Ratios. Sensitivity (Sn) is the percentage with a knee dysfunction who test positive on the dysfunctional side and is calculated as Sn=TP/(TP+FN). A test that is highly sensitive is good at ruling out disease or an impairment and are good for screening purposes. Specificity (Sp) is the percentage without a knee dysfunction who test negative on the non-dysfunctional side and is calculated as Sp=TN/(FP+TN). A test that is highly specific is good at ruling in disease or an impairment and are good confirmatory tests. Likelihood Ratio (LR) represents the probability of a patient with a disease or dysfunction having a positive or negative test result in comparison to the probability of a patient without the disease having a positive or negative test result. Positive LR (+ve LR) suggests the number of times more likely a positive test result observed in the symptomatic knee versus the asymptomatic knee. It suggests how well disease is ruled in. Positive likelihood ration is calculated as +ve LR = sensitivity/1-specificity. Negative LR suggests the number of times more likely a negative test result is observed in the symptomatic knee versus asymptomatic knee. It suggests how well disease is ruled out. Negative likelihood ratio is calculated as -LR = 1-sensitivity/specificity. Positive predictive value (PPV) is the percentage of positive test results that are true positives and is calculated as PPV=TP/(TP+FP). Negative predictive value (NPV): It is the percentage of negative test results that are true negatives and is calculated as NPV=TN/(FN+TN).

### **Test Reliability:**

The reliability of a clinical test is the extent to which results are consistent between different and blinded examiners across different occasions of testing. The weighted kappa

was used to measure association as the scale of measurement was ordinal. Thus, interrater reliability was determined using the kappa statistic (Raghavan, Fosler and Lai, 2012).

# **Receiver operating characteristic (ROC)**

ROC curves are a graphical depiction of a test's overall diagnostic performance (Karimollah Hajian-Tilaki, 2013). The Y axis represents Sensitivity, and the X axis represents 1-Specificity. The closer the curve fills out the top left corner, the better the test is performance is quantified by the area under the curve (AUC). An AUC of 0.5 states that the test performs no better than chance (not useful). An AUC of 0.9 would suggest a better-performing test.

# **Results:**

For the SAPLT, the inter-rater reliability was k=0.926 (SE of kappa = 0.072 95% confidence interval) indicating almost perfect agreement. (Table 2).

### Table 2

	А	В	Total
A	26	1	27
В	1	26	27
Total	27	27	54

*Number of observed agreements: 26 ( 96.30% of the observations)* 

Number of agreements expected by chance: 13.5 (49.93% of the observations)

*Kappa*= 0.926

*SE of kappa* = 0.072

95% confidence interval: From 0.784 to 1.000

25 of the 54 knees tested resulted in a positive test elicited on the symptomatic side and 24 of the 54 knees tested resulted in a negative test on the asymptomatic side. 5 of the 54 knees resulted as a false positive test (Table 3). Calculation for test validity was performed with basic calculations utilizing the formulas to calculate Sn, Sp, positive and negative LR and PPV respectively.
#### Table 3: Contingency Table

TEST	PRESENT	ABSENT	TOTAL
+VE	TP <b>25</b>	FP 5	TP+FP= <b>30</b>
-VE	FN 0	TN 2 <b>4</b>	FN+TN=2 <b>4</b>
Total	TP+FN= <b>25</b>	FP+TN=2 <b>9</b>	54

Sensitivity (Sn): Sn=TP/(TP+FN) 25 / (25+0) = 1.0

Specificity (Sp): Sp=TN/(FP+TN) 24 / 5+24) = 0.82

Positive LR (+ve LR): sensitivity/1-specificity100 / 0.18) = 5.5 Negative LR (-ve LR):1-sensitivity/specificity 0/0.82 = 0.0 Positive predictive value (PPV): PPV=TP/(TP+FP) 25 / (25+5) = 0.83 Negative predictive value (NPV): NPV=TN/(FN+TN) 24/ (24+0) = 1.0 Overall accuracy 100x(TP+FP) / (TP+FN+FP+TN) 4900 / 54 = 90.74

Test validity calculation revealed Sn=1.0, Sp=0.82, +ve LR=5.0, -ve LR=0.0, PPV of 0.83, NPV of 1.0 and an overall accuracy of 90.74% (Table 4). A highly sensitive test means that there are few false positive results, and thus fewer cases 'with' the disease are missed. A highly specific test means that there are few false negative results, thus fewer cases 'without' the disease are missed. A test with a sensitivity and specificity of around 0.9 (90%) is generally considered to have clinically meaningful and acceptable diagnostic performance. The results suggest the SAPLT to

be a sensitive test, however lacking specificity. A positive LR of 10 or greater results in a large and significant increase in the probability of a disease, and a negative LR of 1 or lesser suggests less likely the disease or outcome (Ranganathan P and Aggarwal R, 2018). The results of this study indicated a high negative likelihood ratio suggesting that if the SAPLT is negative, the less likely the individual will 'have' the impairment or disease (Fig 3).

## Table 4: Validity Results

Statistic	Value	95% CI
Sensitivity	100.00%	86.28% to 100.00%
Specificity	82.76%	64.23% to 94.15%
Positive Likelihood Ratio	5.80 /5.5	2.61 to 12.87
Negative Likelihood Ratio	0.00	
Positive Predictive Value (*)	83.33%	69.25% to 91.73%
Negative Predictive Value (*)	100.00%	
Accuracy (*)	90.74%	79.70% to 96.92%

#### Fig. 3. Nomogram for LR



The greater the positive predictive value, the more specific the test and less likely an individual with a positive test will 'not have' the disease or impairment (Trevethan R, 2017). The results of this study revealed a greater negative predictive value indicating the SAPLT to be a more sensitive test and that an individual with a negative test less is likely to 'have' the impairment or disease. ROC analysis revealed the curve filling out the top left corner with an AU of 0.964, suggesting a better-performing test (Fig.4).

### Fig 4. ROC analysis



Diagonal segments are produced by ties.

# Discussion and defense of the 'validity' of the test:

The purpose of this study was to investigate if the SAPLT is a reliable and valid test to evaluate the presence of extension lag at the knee. Variables of interest relevant to assessing diagnostic utility of a test like Sn, Sp, LR and PV, were evaluated. The results of this study suggest the SAPLT to be a reliable and valid test to evaluate the presence of extension lag at the knee. While diagnostic tests assess the presence of a disease or disorder, the SAPLT was specifically used to evaluate impairment and not disease, which requires further description.

### The impairment-based test and its relevance to a gold or reference standard:

Impairment-based tests are tests that attempt to specifically identify impairments in the sensory or motor systems in the body. Such tests are predictors of pain, imminent dysfunction, or functional challenges such as gait disorders or falls (Jiandani and Mhatre, 2018). In diagnosis literature the term 'gold standard' or 'reference standard' is often encountered (Ruties et al, 2007). The term "gold standard" is used to describe a diagnostic test that is regarded as definitive for a particular disease, and thereby becomes the ultimate measure for comparison. While they may be expected to have a 100% accuracy, it may not always be the case. In methodological designs where the diagnostic utility of a particular test is studied, the blinded examiner applies the test on a symptomatic and asymptomatic population where the symptomatic subjects are evaluated based on a gold standard. Then the validity of the test studied may be tested by its ability to identify a positive test on the symptomatic and the negative test on the asymptomatic subjects. An impairment-based test, however, may often lack a gold standard as the impairment is visually or clinically obvious. The deep neck flexor endurance test (DNF) that is most preferred by clinicians to assess the strength of the deep flexors of the neck (Bobos et al, 2016) is an example as there is no gold standard described to assess weakness of the deep neck flexors. The SAPLT may also be considered an impairment-based test that may operate without a reference standard as there is no gold standard described to assess terminal extension lag at the knee. The lack of a reference standard however may mean that the SAPLT may be inadequate to test individuals with bilaterally symptomatic knees.

The single largest advantage of an impairment-based test is that while it correlates current or predicts imminent dysfunction it also suggests the most appropriate and directly relevant treatment intervention. As an example, while weakness of the deep neck flexors has been adequately

described to cause neck dysfunction (Bobos et al, 2016), a positive DNF test will also suggest the need for deep neck flexor strengthening. Similarly, the SAPLT firstly correlates current or predicts imminent knee dysfunction. In addition, it suggests that if the active component appears positive, the possibilities are weak quadriceps, tight hamstrings and gastrocnemius, leading to their appropriate management of flexibility and strength training. If the passive component appears positive, the appropriate intervention may be to first lengthen the posterior capsule, hamstrings, and gastrocnemius prior to strengthening the quadriceps and the gastrocnemius for an effective plantarflexion knee extension force couple.

### Clinical implications of extension lag at the knee:

An active knee extension lag is described as the inability to extend the knee actively into full range of extension, in the absence of a passive restraint. The quadriceps has been described as the primary mechanism that performs this activity. The quadriceps may be directly challenged by trauma or surgery, by inhibition secondary to trauma and by a neurological compromise of the femoral nerve (Runner RP et al, 2018; Koh et al, 2012; Onodera et al, 2012; Rice and McNair, 2009; Nyland et al, 2005; Chimera et al, 2012; Gupta et al, 2012; Jefferson et al, 1990).

While a direct injury to the extensor mechanism disrupts the ability of the knee to extend fully, another mechanism that occurs following trauma, surgery, progressive wear and tear or immobilization is a process known as arthrogenic muscle inhibition (AMI). While AMI is ubiquitous across knee joint pathologies, its severity may vary according to the degree of joint damage, time since injury, and knee joint angle (Rice, McNair and Lewis, 2011). AMI is caused by a change in the discharge of articular sensory receptors, and challenges to the spinal reflex pathways. Additionally, a poor cortical representation can also contribute to inhibitory states of the quadriceps (Rice and McNair, 2010), (Iglesias, Lourenco and Marchand-Pauvert, 2012).

The quadriceps receives its nerve supply from the femoral nerve which arises from rootlets of the L2, L3, and L4 nerve roots. It also receives minor contributions from the L1 and L5 spinal nerves. Injury of the femoral nerve, therefore, may result in weakness or paralysis of hip flexion and knee extension, atrophy of the quadriceps muscle, and numbness of the anterior thigh and medial aspects of the leg and foot. The femoral nerve can be compressed along its course due to penetrating, compressive, and iatrogenic injuries, mostly over the anterior aspect of the hip and inguinal area. Mechanical compression may range from prolonged postures of hip flexion as in sitting on a low chair for extended periods or gynecologic procedures in the lithotomy position (Sebastian D and Krishnan. S) (Naroji S., et al, 2009). Thus, clinicians should be aware of the possibility of potential femoral nerve compression as a causative factor for extension lag at the knee

Passive restraints to knee extension are relatively rigid and sometimes termed as flexion deformity. The description of a passive restraint is the inability to extend the knee fully both passively and actively with the continued ability to flex the knee. The primary structures described in the literature to cause a passive restraint to terminal knee extension are the capsuloligamentous structures followed by adaptive shortening of the relevant musculature. The posterior capsule of the knee is described as the primary passive restraint to terminal knee extension with the hamstrings, popliteus, and gastrocnemius as adaptive contributors. (Rutherford , Hubley-Kozey and Stanish , 2012 ; Nyland et al., 2005 ; Gupta et al., 2012). While all causes described for an active lag may cause a passive lag, the assumed position of comfort of knee flexion in a painful knee (Johnson, 2000) and the avoidance of terminal knee extension in individuals who present with ACL deficient knees (Dandy and Edwards, 1994), are other causes (Fuentes et al., 2011).

The presence of an extension lag at the knee might indicate a compromise of the normal functioning of the lower kinetic chain during functional activities. The implication is higher when this compromise is persistent for an extended period. In closed kinetic chain, the quadriceps and the plantarflexion knee extension couple have been described to work eccentrically during the early stance phase of gait and to assist in knee extension during terminal stance, respectively. (Rice, McNair and Lewis, 2011; Schweizer Romkes and Brunner, 2013). This collectively is described to sufficiently absorb shock, reducing impulsive loading at the knee. Quadriceps weakness has also been associated with an increased rate of loading at the knee joint, contributing to persistent knee pain, patellofemoral cartilage loss and tibiofemoral joint space narrowing (Petterson et al., 2008; Amin et al., 2009; Segal et al., 2010).

### Tibial internal rotation and relevance to knee extension lag and knee dysfunction:

The knee presents as a hinge joint with two degrees of motion flexion and extension, with accompanying rotation (Chen h, 2014 and Vianti E, 2017). Extension of the knee is accompanied by tibial external rotation, also termed 'screw home' (Kim H, 2015) and flexion is accompanied by tibial internal rotation (Chen, 2014 and Vianti E, 2017). Researchers have described that the tibia rotated internally  $(11.55^{\circ} \pm 3.20^{\circ})$  during knee flexion and rotated externally  $(11.40^{\circ} \pm 3.0^{\circ})$  during knee extension (Chen H, 2014). During daily activities, many muscles are used to achieve tibial internal rotation, such as the popliteal muscle, semitendinosus, semimembranosus, sartorius, and gracilis (Sebastian D, Chovvath R and Malladi R 2015), and in external rotation, such as the biceps femoris and vastus lateralis ( Lee TQ, 2003). The clinical measurement of knee range of motion of flexion and extension has been standardized (Hancock GH, Hepworth T, Wembridge K 2018), however there is currently no clinical method to establish the presence of tibial external and internal rotation. While the literature supports tibial internal and external rotation to be occurring with knee flexion and extension respectively (Chen, 2014 and Vianti E, 2017), the assumption that a lack of terminal knee extension has rendered the tibia to be in a position of relative internal rotation may be justified. In other words, the presence of a positive SAPLT may indicate the presence of

tibial internal rotation (lacking screw home), however, not specified in degrees. This clinical finding, as described introductory section, has strong clinical and functional implications warranting resolution, to prevent undue stress of the vulnerable structures.

Uncontrolled or persistent internal rotation of the tibia, especially in closed kinetic chain has been described to unduly stress the tibiofemoral articular cartilage (Yazdi H, 2016), the menisci (Kim JG), the supporting ligaments principally the anterior cruciate ligament (ACL) (Mokhtarzadeh H, 2015) and the patellofemoral joint (Sisk and Fredericson, 2019). Mechanical factors can favor the progression of knee osteoarthritis (OA) and is determined in part by mechanical effects on local structures (Omori G). Although mechanical influences of excessive loading or malalignment can cause cartilage loss, it has also been described that tibial internal rotation can increase tibiofemoral cartilage surface contact pressure (Yazdi H, 2016). It can be argued that in the early stages of knee joint degeneration, if tibial internal rotation is not identified with an appropriate test and resolved, cartilage wear might result. This may be especially relevant when the persistence of tibial internal rotation is prolonged or left untreated, predisposing to a progression in the degenerative process.

The menisci play an important role in load distribution, load bearing, joint stability, lubrication, and proprioception. The meniscus is embedded between the femoral condyle and the tibial condyle, which improves congruence and stability of the knee joint. During the process of flexion, the meniscus decreases in diameter as it moves backward. The movement is small and much lesser compared to the lateral meniscus. The tibiofemoral contact area gradually decreases during flexion with its accompanying internal rotation because of the large curvature radius at the top of the femoral condyle and the reduced radius posteriorly. This arrangement helps the loading to be uniformly distributed, preventing damage to the meniscus. This movement is reversed during extension and tibial external rotation (Kim JG, 2013). While the maintenance of

this small movement is important for both maintenance and damage prevention of the meniscus, it may be sufficiently challenged during prolonged knee flexion and lack of screw home. This may in turn render the medial meniscus vulnerable suggesting another structure that can potentially benefit from the identification and resolution of an extension lag at the knee.

Internal tibial rotation is a risk factor for anterior cruciate ligament (ACL) injury. Researchers investigated the effect of impact loads and restraining tibial rotation on ACL injury with respect to flexion angle (Mokhtarzadeh H, 2015). They hypothesized that restraining tibial rotation could protect the knee from ACL injury compared to free tibial rotation regardless of flexion angle to create a safety zone to protect the ACL. The findings revealed that the largest difference between peak axial compressive forces causing ACL injury was reported at a knee flexion angle of 22°. Their findings recommended neuromuscular training programs or brace designs that can avoid excessive internal tibial rotation.

Pain from patellofemoral compression continues to remain a diagnosis of exclusion, with several variables being described as potential contributors (Sisk and Fredericson, 2019; Petersen W, 2014). The variable of relevance to the SAPLT would be hamstring tightness and knee flexion which is described as a cause for knee extension lag. Researchers have demonstrated a relationship between hamstring tightness and increased patellofemoral joint (PFJ) stress during squatting due to increased PFJ reaction force and reduced medial PFJ contact area (White EF, 2010). Other studies have described the influence of both femoral and tibial rotation as a potential source of increased PFJ stress and subsequent dysfunction (Sisk and Fredericson, 2019; Lee TQ, 2003).

#### Disclosures

No funding was obtained for this study and the authors of this study do not report any conflict of interest.

### **Conclusion:**

The SAPLT has demonstrated excellent reproducibility between examiners and has shown to be sensitive in identifying the presence of an extension lag in unilaterally symptomatic knees. Additionally, the test has demonstrated the ability to differentiate between the presence of an active versus a passive lag or identify the presence of both, enabling the clinician to institute the most appropriate management. There is ample evidence to suggest that the resolution of a persistent knee extension lag can decrease undue stresses over multiple structures in and around the knee, decreasing the risk for dysfunction. This warrants the prompt identification of the presence of a lack of knee extension and the SAPLT may hence serve as an easily administered reliable and valid method. The test however poses limitations as tightness of the iliopsoas in causing a lag is ignored (Lee et al, 2001). Additionally, the maintenance of an erect and normal lordosis and maximum ankle dorsiflexion may be missed during the testing process rendering results to be possibly inaccurate (Sebastian D, Chovvath R, Malladi R). The SAPLT is an impairment-based test. While a reference standard may be appropriate for testing diagnostic utility, establishing one for an impairment-based test might be a difficult task. While the gold standard for measuring ROM is electro goniometry, many 'normal' knees may hyperextend past zero degrees of extension, questioning 'normal 'even if accurate ROM is established. Clinically however, the reference standard limitation suggests that the SAPLT might be best suited for identifying the presence of a knee extension lag in unilaterally symptomatic knees. The above stated limitations, however, may be suggestions for future research.

### **Bibliography:**

- 1. Aderinto J, Brenkel IJ, Chan P, 2005 Natural history of fixed flexion deformity following total knee replacement: a prospective five-year study. J Bone Joint Surg Br. 87(7):934-6.
- Amin S, Baker K, Niu J, Clancy M, Goggins J, Guermazi A, Grigoryan M, Hunter DJ, Felson DT 2009 Quadriceps strength and the risk of cartilage loss and symptom progression in knee osteoarthritis. Arthritis Rheum 60:189–198.
- Bédard M, Vince KG, Redfern J, Collen SR 2011 Internal Rotation of the Tibial Component is Frequent in Stiff Total Knee Arthroplasty. Clin Orthop Relat Res 469(8): 2346–2355.
- Bobos P, Billis E, Papanikolaou DT, Koutsojannis C, MacDermid JC 2016 Does Deep Cervical Flexor Muscle Training Affect Pain Pressure Thresholds of Myofascial Trigger Points in Patients with Chronic Neck Pain? A Prospective Randomized Controlled Trial. Rehabil Res Pract 6480826.
- 5. Brooks P 2009 Extensor mechanism ruptures. Orthopedics 32(9): 728-31.
- Bylski-Austrow DI, et al 1994 Displacements of the menisci under joint load: an in vitro study in human knees. J Biomench 27: 421-431.
- Chen H, et al 2014 Assessment of tibial rotation and meniscal movement using kinematic magnetic resonance imaging. Journal of Orthopaedic Surgery and Research 9 : 65.
- Chimera NJ, Castro M, Davis I, Manal K 2012 The effect of isolated gastrocnemius contracture and gastrocnemius recession on lower extremity kinematics and kinetics during stance. Clin Biomech 27(9):917-23.

- Dandy DJ, Edwards DJ 1994 Problems in regaining full extension of the knee after anterior cruciate ligament reconstruction: does arthrofibrosis exist? Knee Surg Sports Traumatol Arthrosc 2(2):76-9.
- Enrico Vaienti, Giacomo Scita, Francesco Ceccarelli, and Francesco Pogliacomi 2017 Understanding the human knee and its relationship to total knee replacement. Acta Biomed 88(Suppl 2): 6–16.
- 11. Feng GZ, Dai H, He Y 2006 Clinical observation on extensor weakness after total knee arthroplasty for severe flexion deformity knees. Zhonghua Wai Ke Za Zhi 44(8):519-22.
- Fuentes A, Hagemeister N, Ranger P, Heron T, de Guise JA 2011 Gait adaptation in chronic anterior cruciate ligament-deficient patients: Pivot-shift avoidance gait. Clin Biomech 26(2):181-7.
- Goudie ST, Deakin AH, Ahmad A, Maheshwari R, Picard F 2011 Flexion contracture following primary total knee arthroplasty: risk factors and outcomes. Orthopedics 34(12):855-9.
- 14. Gupta S, Mahmud T, Davy A, Mitchell AW, Williams A 2012 Isolated ischaemic contracture of medial head of gastrocnemius. Knee Surg Sports Traumatol Arthrosc 20(11):2353-5.
- 15. Gurney B 2002 Leg length discrepancy. Gait Posture 15(2):195-206.
- 16. Hancock GH, Hepworth T, Wembridge K 2018 Accuracy and reliability of knee goniometry methods. J Exp Orthop 19;5(1):46.
- Hassanzadeh J, Rezaianzadeh A 2012 Assessing the Validity of Diagnostic Tests. Iran J Med Sci 37(1): 2.

- Ha Yong Kim, et al, 2015 Screw-Home Movement of the Tibiofemoral Joint during Normal Gait: Three-Dimensional Analysis. Clin Orthop Surg 7(3): 303–309.
- 19. Hotfiel T, Carl HD, Swoboda B, Jendrissek A, Pauser J 2012 Foot load with different restrictions of knee joint extension. Z Orthop Unfall 150(3):257-61.
- 20. Husson JL, Mallet JF, Huten D, Odri GA, Morin C, Parent HF 2010 Applications in hip pathology. Orthop Traumatol Surg Res 4.
- 21. Iglesias C, Lourenco G, Marchand-Pauvert V 2012 Weak motor cortex contribution to the quadriceps activity during human walking. Gait Posture 35(3):360-6.
- 22. Jefferson RJ, Collins JJ, Whittle MW, Radin EL, O'Connor JJ 1990 The role of the quadriceps in controlling impulsive forces around heel strike. Proc Inst Mech Eng H 204:21–28.
- Jiandani MP. Mhatre BS 2018 Physical therapy diagnosis: How is it different? J Postgrad Med 64(2):69-72.
- Johnson MW 2000 Acute Knee Effusions: A Systematic Approach to Diagnosis, Am Fam Physician 61(8):2391-2400.
- 25. Kappetijn O, Trijffel EV and Lucas C 2014 Efficacy of passive extension mobilization in addition to exercise in the osteoarthritic knee: an observational parallel-group study. Knee 21(3):703-9.
- 26. Karimollah Hajian-Tilaki 2013 Receiver Operating Characteristic (ROC) Curve Analysis for Medical Diagnostic Test Evaluation. Caspian J Intern Med 4(2): 627–635.
- 27. Kim JG et al, 2013 Tibiofemoral contact mechanics following posterior root of medial meniscus tear, repair, meniscectomy, and allograft transplantation. Knee Surg Sports Traumatol Arthrosc 21: 2121-2125.

- 28. Koc TA et al, 2019 The immediate effects of knee flexion range of motion following manual therapy or self-stretching/active range of motion following a total knee arthroplasty: a case report. J Phys Ther Sci 31(12): 1002–1005.
- 29. Koh IJ, Chang CB, Kang YG, Seong SC, Kim TK 2012 Incidence, Predictors, and Effects of Residual Flexion Contracture on Clinical Outcomes of Total KneeArthroplasty. J Arthroplasty 5403(12): 541-4.
- 30. Lee KM et al, 2011 Reliability of physical examination in the measurement of hip flexion contracture and correlation with gait parameters in cerebral palsy. J Bone Joint Surg Am 93(2):150-8.
- 31. Lee TQ, Morris G, Csintalan RP 2003 The influence of tibial and femoral rotation on patellofemoral contact area and pressure. J Orthop Sports Phys Ther 33(11):686-93.
- 32. Michel De Maeseneer, Peter Van Roy, Maryam Shahabpour, Robert Gosselin 2004 Normal Anatomy and Pathology of the Posterior Capsular Area of the Knee: Findings in Cadaveric Specimens and in Patients. American Journal of Roentgenology 182(4):955-62
- 33. Mokhtarzadeh H et al, 2015 Restrained tibial rotation may prevent ACL injury during landing at different flexion angles. Knee 22(1):24-9.
- Naroji S 2009 Vulnerability of the Femoral Nerve During Complex Anterior and Posterior Spinal Surgery. J Spinal Cord Med. 32(4): 432–434.
- 35. Nyland J et al, 2005 Anatomy, function, and rehabilitation of the popliteus musculotendinous complex. J Orthop Sports Phys Ther 35(3):165-79.
- 36. Omori G 2016 Association of mechanical factors with medial knee osteoarthritis: A cross-sectional study from Matsudai Knee Osteoarthritis Survey. J Orthop Sci 21(4):463-468.

- 37. Onodera T, Majima T, Nishiike O, Kasahara Y, Takahashi D 2012 Posterior Femoral Condylar Offset After Total Knee Replacement in The Risk of Knee Flexion Contracture. J Arthroplasty 5403(12): 557-8.
- 38. Pallant J 2020 SPSS survival manual: A step by step guide to data analysis using IBM SPSS 7<sup>th</sup> ed. McGraw Hill, London.
- Petersen W et al 2014 Patellofemoral pain syndrome. Knee Surg Sports Traumatol Arthrosc 22(10): 2264–2274.
- 40. Petterson SC, Barrance P, Buchanan T, Binder-Macleod S, Snyder-Mackler L 2008 Mechanisms underlying quadriceps weakness in knee osteoarthritis. Med Sci Sports Exerc 40:422–427.
- 41. Ranganathan P, Aggarwal R 2018 Understanding the properties of diagnostic tests Part
  2: Likelihood ratios. Perspect Clin Res 9(2): 99–102.
- 42. Raghavan P, Fosler-Lussier E, Lai AM 2012 Inter-annotator reliability of medical events, coreferences and temporal relations in clinical narratives by annotators with varying levels of clinical expertise. AMIA Annu Symp Proc 3:1366-74.
- 43. Rice DA, McNair PJ 2010 Quadriceps arthrogenic muscle inhibition: neural mechanisms and treatment perspectives. Semin Arthritis Rheum 40(3):250-66.
- 44. Rice DA, McNair PJ, Lewis GN 2011 Mechanisms of quadriceps muscle weakness in knee joint osteoarthritis: the effects of prolonged vibration on torque and muscle activation in osteoarthritic and healthy control subjects. Arthritis Res Ther 13(5):151.
- 45. Rossi R et al, 2011 Clinical examination of the knee: know your tools for diagnosis of knee injuries. Sports Med Arthrosc Rehabil Ther Technol 28(3):25.

- 46. Runner RP et al, 2018 Quadriceps Strength Deficits After a Femoral Nerve Block Versus Adductor Canal Block for Anterior Cruciate Ligament Reconstruction: A Prospective, Single-Blinded, Randomized Trial. Orthop J Sports Med. 6(9): 2325967118797990.
- 47. Rutherford DJ, Hubley-Kozey CL, Stanish WD 2012 Knee effusion affects knee mechanics and muscle activity during gait in individuals with knee osteoarthritis. Osteoarthritis Cartilage 20 (9):974-81.
- 48. Rutjes AWS, Reitsma JB, Coomarasamy Khan AKS, Bossuyt PMM 2007 Evaluation of diagnostic tests when there is no gold standard. A review of methods. Health Technol Assess 11(50)3: 9-51.
- 49. Schache AG, Blanch PD, Murphy AT 2000 Relation of anterior pelvic tilt during running to clinical and kinematic measures of hip extension. Br J Sports Med 34(4):279-83.
- 50. Schweizer K, Romkes J, Brunner R 2013 The association between premature plantarflexor muscle activity, muscle strength, and equinus gait in patients with various pathologies. Res Dev Disabil 34(9):2676-83.
- 51. Sebastian D, Chovvath R. Malladi M 2015 The sitting active and prone passive lag test: An inter-rater reliability study. Journal of Bodywork and Movement Therapies 18(2):204-9.
- 52. Sebastian D, Krishnan S 2019 Principles of Diagnostic Sonography in Iliopsoas Tendon Pathology. Journal of Bodywork & Movement Therapies. 23(2): 352-58.
- 53. Segal NA et al, 2010 Quadriceps weakness predicts risk for knee joint space narrowing in women in the MOST cohort. Osteoarthritis Cartilage 18:769–775.
- 54. Shea KG, Archibald-Seiffer N, Kim KM, Grimm NL 2012 Bucket-handle meniscal tear in a 5-year-old child. Knee Surg Sports Traumatol Arthrosc 20(11):2291-3.

- 55. Sisk D, Fredericson M 2019 Update of Risk Factors, Diagnosis, and Management of Patellofemoral Pain. Curr Rev Musculoskelet Med 12(4): 534–541.
- 56. Stillman BC 2004 Physiological quadriceps lag: its nature and clinical significance. Aust J Physiother 50(4):237-41.
- 57. Temelli Y, Akalan NE 2009 Treatment approaches to flexion contractures of the knee. Acta Orthop Traumatol Turc 43(2):113-20.
- Thompson WO, Thaete FL, Fu FH, Dye SF 1991 Tibial meniscal dynamics using threedimensional reconstruction of magnetic resonance images. Am J Sports Med 19: 210-215.
- Trevethan R 2017 Sensitivity, Specificity, and Predictive Values: Foundations,
   Pliabilities, and Pitfalls in Research and Practice. Front Public Health 5: 307.
- 60. Tweed M, Wilkinson T 2012 Diagnostic testing and educational assessment. Clin Teach 9(5):299-303.
- 61. Vince KG, Cameron HU, Hungerford DS, Laskin RS, Ranawat CS, Scuderi GR 2005 What would you do? Case challenges in knee surgery. J Arthroplasty 20(4 Suppl 2):44-50.
- 62. Whyte EF 2010 The influence of reduced hamstring length on patellofemoral joint stress during squatting in healthy male adults. Gait Posture 31(1):47-51.
- 63. Yazdi et al, 2016 The effect of tibial rotation on knee medial and lateral compartment contact pressure. Knee Surg Sports Traumatol Arthrosc 24(1):79-83.
- 64. Zalta J 2008 Massage therapy protocol for post-anterior cruciate ligament reconstruction patellofemoral pain syndrome: a case report. Int J Ther Massage Bodywork 1(2):11-21.

# ACNOWLEDGEMENTS

I wish to thank my patients, teachers, and students for reminding me of my purpose for existence. I sincerely thank my parents, almighty God, my wife, son, family and friends, for just about everything and anything that I do not wish to take for granted. I wish to thank my supervisor and relevant faculty at the Selinus University of Sciences and Literature for the opportunity and the scrutiny offered to validate and proclaim my work.

Sincerely

Deepak Sebastian