Nick

Grimaldi A, Fearon A. **Gluteal tendinopathy: integrating pathomechanics and clinical features in its management**. *J Ortho Sport Phys Ther*. 2015; 45:910-922.

A great article by great authors in a great journal. This article was part of the journal published last November in JOSPT devoted entirely to tendinopathy. I do not feel that I was adequately educated regarding the development, evaluation, and management of this condition in physical therapy school, and knew that I would be benefited by reading this article. I was not disappointed, and I highly commend it to all who are seeking a more thorough understanding of the subject.

The article is a clinical commentary integrating the limited amount of evidence with the authors experience in evaluating and treating the condition. As a clinical commentary, it covers a large amount of material that I will not attempt to repeat here; rather, I will comment myself on a few particularly helpful components in the publication.

The assert that, in contrast to previously held and taught beliefs, gluteal tendinopathy is now believed to be the primary source of lateral hip pain (vs. trochanteric bursitis). Risk factors for developing the condition include female gender, age over 40, concurrent low back pain, trochanteric offset, and gynoid adiposity.

The authors comment on some of the objective tests utilized to diagnose the condition, including single leg stance, resisted external de-rotation, resisted medial rotation, FABER, and Ober testing. As with many of our orthopedic tests, the studies reporting the test metrics have methodological flaws, however the authors repeatedly emphasize that identifying *where* the patient feels their pain during (i.e., near the lateral gluteal tendons/greater trochanter vs. elsewhere) the performance of the test can help to greatly improve the validity of the test.

The authors spend a considerable portion of time discussing the pathomechanics of the condition. They suggest the gluteal tendons become compressed against the greater trochanter through increased superficial abductor and subsequent ITB activation as well as functional movements being performed in greater degrees of hip adduction. Pictures are employed that help to beautifully demonstrate the pathological tendon compression as well as postures to avoid that involve greater degrees of hip adduction. These could be very beneficial for patient education purposes.

Finally, the authors comment on treatment of gluteal tendinopathy, for which research is greatly lacking. The authors recommend load management through activity and positioning modification, avoidance of stretches that increase compression to the gluteal tendons, movement re-education, and exercise therapy including the early use of isometric exercise and progressive tendon loading/gluteal strengthening in healthy positions.

Laura

Cervical and thoracic manipulations: Acute effects upon pain pressure threshold and self-reported pain in experimentally induced shoulder pain

Craig A. Wassinger, Dustin Rich, Nicholas Cameron, Shelley Clark, Scott Davenport, Maranda Lingelbach, Albert Smith, G. David Baxter, Joshua Davidson

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As a product of the ever-challenging and complex enigma that is shoulder pain, authors Wassinger et. al. have used an inductive experimental approach to directly analyze the analgesic effects of cervical and thoracic manipulations on stimulated pain in the shoulder. Previous literature has examined effects of thoracic manipulation in patients with subacromial impingement syndrome. This article is the first of its kind looking at the effect of thrust manipulations of not only thoracic, but of cervical and cervicothoracic regions on exercise-induced delayed onset muscle soreness in shoulders of healthy individuals.

The participants in the study completed a concentric-eccentric exercise protocol on the non-dominant shoulder using an isokinetic dynamometer designed to induce delayed onset muscle soreness of the external rotators of the shoulder. The participants then received high velocity low amplitude thrust manipulations to cervical spine (target levels were C5, C6, C7 using lateral thrust technique), cervicothoracic region (using distraction technique), and thoracic spine (using prone extension PA to apex of kyphosis, then proximal and distal 2 segments for a total of 5 thrusts to thoracic spine). HVLA was performed up to two attempts at each cervical segment until cavitation was noted by participant or investigator. Nine to twelve thrusts total were possible for each participant. Outcomes were measured by SPADI pain scale and pain pressure threshold using a digital algometer to the infraspinatus muscle bellies on both shoulders.

The authors found that the exercise protocol produced a significant increase in reported shoulder pain by the SPADI pain scale (by a mean of 14.1), then subsequently the manipulation protocol produced a significant decrease in reported pain (by a mean of 5.60). The authors also found a 27.7% and 22.5% increase in their pain pressure threshold to the non-dominant and dominant shoulders, respectively post-manipulation. This change was not significantly higher than baseline values and effect sizes were 0.44 and 0.38 (moderate strength) for non-dominant and dominant shoulders, respectively.

The results of this study conclude that thrust manipulation, although location not specified, can have a local and systemic hypoalgesic effect on shoulder pain. The mechanism is unknown, however the authors describe several theories that could explain the positive effects including vertebral joint stimulation, vibration of large diameter low-threshold mechanoreceptors, and descending inhibitory pain systems stimulus. The vertebral levels between C5 and T2 were specifically

selected for their association with the infraspinatus nerve root innervation, more so than regional movement interdependence.

Immediate applicability is limited to due to many factors. The authors state that the use of young participants in the study was to decrease the likelihood of age related degeneration of the shoulder muscles. We usually do not directly treat healthy, not injured shoulders in the clinic, therefore the interventions that we ideally seek are supported in the literature by studies that use similar conditions. The number of thrust manipulations performed on each patient was between nine and twelve in three distinct regions. Personally, this is over the amount of manipulations that I would feel comfortable in performing in a single session and clouds the isolated effect of each region. I am not familiar with any other studies that support using cervical thrust manipulations for decreasing shoulder pain, only preliminary studies on non-thrust cervical mobilizations on subacromial pain and cervical manipulation on upper trapezius latent trigger points. There were no control or comparative groups included in this study to determine isolated effects of cervical, cervicothoracic, and thoracic manipulations. Therefore, there can be no conclusion on the actual effects of cervical, cervicothoracic, and thoracic manipulations in a cluster vs. thoracic manipulations alone.

Vertebrobasiliar insufficiency (VBI) screening was performed twice in the protocol, once to determine eligibility criteria and once to comply with published guidelines on premanipulative screening. Each participant was also screened for precautions and contraindications of each manipulation technique prior to application, however the participants were not screened for any segmental mobility restrictions or impairments in these regions to justify treatment. With the exception of VBI screening, this further separates the representation of clinical practice within this study and narrows the possible effects to only neurophysiological, missing any outcome measure that would report on motor function, movement, and task performance.

In conclusion, this study does not resemble everyday clinical practice. It is, however, a promising study in the neurophysiologic effect of thrust manipulation on systemic pain threshold and subjective report. Even acute, mild pain directly correlated to the exercise protocol can be altered by a series of thrust manipulations. Non-randomized controlled trials are not invulnerable to confounding factors and bias, but it is safe to say that further study is warranted to analyze the effects in all conditions and locations of the human body.

Sean

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Basic science behind the cardiovascular benefits of exercise

Mathew G Wilson[1,2,3], Georgina M Ellison[4], N Tim Cable[2,5,6]

We all know that improving cardiovascular fitness improves overall health and decreases the risk of cardiovascular disease. Beyond that, I realize that's as far as my patient educational session goes regarding this topic. I enjoyed this article because it's helped me develop a better understanding of the effects of exercise on our cardiovascular system and arms me with more specific information I can relay to my patient's to motivate them to make better health changes. Simply telling a patient that exercise is good for them often isn't enough. People need know what to expect and how long it will take before they see a benefit.

Information I found noteworthy:

- "6 months of regular intensive exercise typically decreases resting and submaximal exercising HR by 5-20 bpm"

- "In 2008, the US Department of Health and Human Services released 'physical activity guidelines for Americans'; being 150-300 min per week of moderate intensity aerobic exercise or 75 min per week of vigorous intensity exercise. While all exercise programs must consider intensity, duration and frequency, it is the total volume of exercise that appears to be the most consistently related to the size of reduction in CV disease or functional improvement. "

- When we think of exercise prescription, we tend to attempt to design a program to affect a single system; anaerobic or aerobic. We tend to think a low intensity, long duration activity improves aerobic capacity and short interval, high-intensity exercise only affects anaerobic capacity and not aerobic. Nick and I briefly touched on this topic this week while discussing the effects of CrossFit. I feel the following quote is applicable and could be helpful to all of us:

    - "A meta-analysis of patients with cardiometabolic diseases (ie, coronary artery disease, heart failure, hypertension, metabolic syndrome and obesity) observed significantly greater increases in VO2max following HIIT (>85% VO2max or >90% HR_{peak}, followed by 2-3 minute active recovery) compared with moderate-intensity continuous training (MICT), equivalent to 9%, meaning that HIIT improved cardiorespiratory fitness by almost double"

Oksana

The Effect of Knee Braces on Quadriceps Strength and Inhibition in Subjects With Patellofemoral Osteoarthritis.

MICHAEL J. CALLAGHAN, PhD, MPhil, MCSP1 • MATTHEW J. PARKES, MSc1 DAVID T. FELSON, MD, MPH. 2016. JOSPT

In patients who have patellofemoral osteoarthritis, flexible braces are recommended to enhance proprioception and create a feeling of stability. However, there have been multiple concerns with wearing knee flexible braces due to a perception that they might cause weakness in the surrounding muscles. There is a study that looks at quadriceps muscle activation and inhibition that actually found the opposite. There was a statistical significance and minimal clinical impact on increased quad activation with decreased quad inhibition for those wearing a flexible knee brace. Braces are important; it all depends on how you use them. I don't recommend using them as a crutch, but they are helpful when indicated.

This study includes 108 participants who had at least 3 months of patellofemoral pain and osteoarthritis. Patients were randomily allocated to either a brace for 6 weeks or no brace and then within- subject change was analyzed following 12 weeks of brace use. Patients were assessed with PFJ symptoms such as pain produced with stair climbing or rising from a chair. Participants were excluded if they had a previous patellar realignment surgery, fracture, predominant knee symptpms emanating from tibiofemoral joint, a history of meniscal or ligament injury, RA or other forms of systemic diseases. Patients in the brace group were given a brace that allows free knee motion. These participants did not receive any physical therapy. Outcome measure were quadriceps muscle strength via an isometric dynamometer. Quadricep inhibition was measured using the same dynamometer. The inhibition was determined by the activation deficit at 100% maximum voluntary contraction (interpolated twitch/ resting twitch) x 100. The smaller the activation deficit the less the inhibition. It was found that there was no decreased in quad torque or inhibition after wearing a knee support daily. However, wearing the knee support increased quadriceps strength and reduced quadriceps inhibition. Even though these findings were statistically significant, its impact was minimal for a clinical effect. Some limitations include: these results may not be generalized to other supports or braces and a lack of a no-brace control group at the 12-week time point.

Alex

Plastaras C, Mccormick Z, Nguyen C, et al**. Is Hip Abduction Strength Asymmetry Present in Female Runners in the Early Stages of Patellofemoral Pain Syndrome?**. Am J Sports Med. 2016;44(1):105-12.

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Resident article review

Purpose:

Determine whether hip abduction strength asymmetry is present in female runners in the early stages of PFPS – before symptoms are severe enough to seek medical care.

Methods:

21 female runners (18-45 yrs; mean=30.5; mean running distance = 18.5 mi/wk) vs. 36 controls matched for age, height, weight and mi/wk.

Hip abduction strength was measured with a handheld dynamometer in neutral and 15° of hip extension.

Results:

- No significant hip ABD strength difference side-to-side in neutral and EXT compared to control group
- In neutral - hip ABD strength of affected limb (PFPS) was significantly stronger compared to weaker limb of control group when tested
- In EXT – no significant difference in hip ABD strength between groups
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Discussion:

The authors conclude that early PFPS does not appear to be significantly associated with hip abduction strength asymmetry (side-to-side). The authors hypothesize about possible progressions of hip strength differences or loss, as symptoms of PFPS progress and discuss possible relevance of tensor fascia latae changes.

The authors discuss some limitations in regards to statistical analysis; however, I believe that the primary limitation of the study is the design. The authors attempt to associate development of a multifactorial condition during high impact functional movement with open kinematic chain strength testing. While it would make for a quick and easy screening tool in the clinic, this test alone gives little information about possible functional impairments – present or developing – that could lead to progression of symptoms. Furthermore, there is little information about further possible contributing factors that could lead to PFPS, such as running mechanics,

compensatory mechanisms during functional testing (single leg squat), foot mechanics/structure, and passive accessory motions of hip, knee and foot/ankle joints.

Clinical pearl:

- Hip abduction strength measured in neutral was greater in the affected limb of the PFPS group, when compared to the weaker limb of the control group.
- Possible indication of increased TFL activation in PFPS group.