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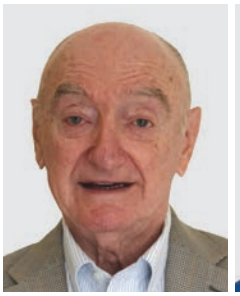
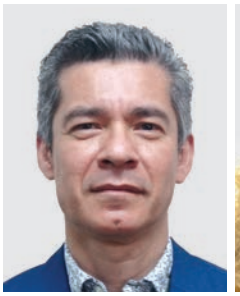
Editorial

To Diagnose Enthesitis Clinically, Should the Entheses Be Put to Work?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Clinicians use pain and tenderness elicited from local pressure at tendon, ligament, and capsular insertions to bone to identify enthesitis.[1,2,3,4,5] In addition, certain entheses have an adjacent bursa or synovial space that, when distended by synovial fluid, adds certainty to a clinical diagnosis of enthesitis.[6,7,8] On the other hand, tenderness at an enthesis site per se is nonspecific, as it may originate in bone, periosteum, joint, muscle, ligament, fascia, or subcutaneous tissue, or reveal fibromyalgia. Pioneers in the field considered that pain caused by traction of the inserting tendon, ligament, or joint capsule was integral to diagnosing enthesitis.[1,2] Eliciting pain by pressing at the enthesis helps determine the actual pain source in the muscle-tendon-bone chain.

Physical examination performs poorly compared with ultrasound (US),[9] and many rheumatologists do not have access to expert US at the point of care. However, there should be no barrier to providing excellent physical examination. Thus, it may be time to retool the physical diagnosis of enthesitis. To this end, we suggest using resisted muscle contraction for tendon entheses and passive joint motion for tendon, ligament, or joint capsule entheses, and applying a gaged force at entheseal sites.

Enthesitis

Giuseppe La Cava, a pioneer in sports medicine from Rome, Italy, first used the term *enthesitis* to name the inflammatory changes at tendon attachments caused by repetitive microtrauma.[1] In 1971, John Ball from Manchester, UK, described the histopathology of ankylosing spondylitis, where inflammation occurred at the enthesis of tendons, ligaments, and joint capsules.[10] There was a notable, farsighted review of enthesopathy by Niepel and Sit'aj in 1979,[2] who stated that "the enthesis includes the cartilaginous insertional tendon, a peritenon in continuity with the perichondrium and the periosteum, bone prominences to allow a firmer tendon grip, and neighboring bursae, fat cushions, and sesamoid bones. In addition, entheses subject to wide angular motion featured an increased cartilage width to prevent sharp angulation and reduce collagen wear." In both these studies,[1,2] subacute or chronic enthesitis features local tenderness and pain upon the resisted action of the involved muscle. Why tendon pain occurs in enthesitis is not fully understood. However, muscle tension is a validated maneuver for tendinopathies of the upper extremity.[11] Stress maneuvers for the greater trochanter pain syndrome have a proven clinical value.[12] Therefore, it stands to reason that pain upon resisted muscle contraction would add clinical validity to a given positive enthesitis tender site. Both traction and attempted contralateral bending would have a similar effect in ligament and capsular entheses, including anterior chest and iliac crest entheses.

In 1998, a study by McGonagle et al[13] based on Ball's findings—plus a broader concept of the spondyloarthropathies (SpA)—hypothesized that enthesitis, defined as an inflammatory process at the bone insertion of tendons, ligaments, and joint capsules, was the central pathophysiologic feature of SpA. In that same year, unaware of the more comprehensive view of the enthesis by Niepel and Sit'aj, an editorial by Canoso[6] based on anatomical, biochemical, and clinical observations, hypothesized on the contributory role of additional structures that were often concurrently involved in SpA in the calcaneal Achilles enthesis and possibly in the tibial patellar tendon enthesis. The additional structures included the retrocalcaneal bursa with its highly viscous bursal fluid, a synovial-layered fat wedge at its top, and insertional, sesamoid, and periosteal fibrocartilage. In addition, a hyaluronate-facilitated movement of the fat wedge in and out of the bursa allowed a more distal, dorsal, and biomechanically advantageous insertion of the Achilles tendon. In 2004, an extensive and detailed study of many articular and extraarticular entheses led to the "enthesis organ" concept,[7] which was an apt term to encompass the complexity of some of these entheses and paved the way for the exponential growth of enthesitis research. In 2007, the enthesis organ concept led to the even more comprehensive idea of the "synovial-entheseal complex," which

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emphasized the presence of synovial membrane in the vicinity of the enthesis, allowing a 2-way origin of inflammation.[8] The cytochemical characteristics of the synovial fluid aspirated from fresh cadaveric retrocalcaneal bursae were identical to fluid aspirated from foot joints.[6] Thus, the blind sampling of small amounts of synovial fluid from these bursae is possible but would be far better performed under US control.

Ultrasonography

Musculoskeletal US provides invaluable support to a diagnosis of enthesitis. Findings include abnormal thickening and hypoechogenicity of the inserting structure on B-mode,[14,15] occurring across various pathologies and even in normal individuals.[9] A positive Doppler signal, except immediately after heavy exercise and repairing tendon tears,[9,16] indicates entheseal inflammation. On the other hand, calcifications, enthesophytes, or erosions reveal structural damage.[9] A cross-sectional single-blinded study of enthesitis with B-mode and Doppler US in SpA, rheumatoid arthritis (RA), and patients with mechanical low back pain showed that entheseal Doppler signal was present only in the first group.[15] In this study, the clinical findings performed poorly compared with the US findings.[9] US-detected enthesitis occurs in SpA,[14,15] psoriasis with and without arthritis,[17] RA, juvenile idiopathic arthritis, systemic lupus erythematosus, systemic sclerosis, familial Mediterranean fever, and possibly in Sjögren syndrome. Enthesopathy can also feature in metabolic syndrome, isotretinoin treatment of acne, and celiac disease, and in patients undergoing dialysis. Interestingly, in healthy subjects, at least 1 US-defined enthesopathy was found in over 50%, albeit with a power Doppler signal > 1 (scale 0–3) in < 2% of studied entheses.[9]

The clinical diagnosis of enthesitis: a proposal

Musculoskeletal US has been considered an extension of the physical examination. If so, in the diagnosis of enthesitis, the

Table 1. Suggested maneuvers for a clinical diagnosis of enthesitis.

Pain[a] and Tenderness to Pressure[b]	Traction Maneuvers[c]	Stressed Tendons/Ligaments/Fasciae
Cervical spinous processes[1]	Sitting or standing, neck flexed, head bending forward	Supraspinous and interspinous ligs. C2–7, nuchal lig. (external occipital protuberance–C7)
Greater humeral tuberosity[1, 5]	Sitting or standing, resisted arm abduction, external rotation, and internal rotation	Supraspinatus m., infraspinatus m., teres major m, subscapularis m. insertions
Distal biceps brachialis[1, 2]	Sitting or standing, resisted forearm supination	Radial insertion of biceps brachii m.
Lateral humeral epicondyle[2, 4, 5, 14]	Sitting or standing, resisted wrist dorsiflexion	Extensor carpi radialis brevis m. origin
Medial humeral epicondyle[1, 5, 14]	Sitting or standing, resisted wrist palmar flexion	Common head of flexors
5th Lumbar spinous process[3]	Sitting or standing, bend forward at the waist	Supra- and interspinous lig.; composite tendon of the thoracolumbar fascia
1st Costochondral joint[3]	Standing, breathe deeply	Costochondral joint
7th Costochondral joint[3]	Standing, breathe deeply	Costochondral joint
Posterosuperior iliac spine[3,18]	Standing, bend forward Lying supine, straight leg raise	Composite tendon of the thoracolumbar fascia
Iliac crest[3]	Sitting or standing, bend to the contralateral side(s); cough	Abdominal muscles insertion
Anterosuperior iliac spine[3]	Lying on the side without pain, extend the painful leg and bring it down behind the examining table	Tensor fascia lata m. origin
Pubis tubercle[1,14]	Lying supine, press extended legs together Sitting, press both knees together	Gracilis m. and adductor longus m. origin
Greater trochanter[5,12,14]	Lying supine, thigh flexed 90° and hip externally rotated, resist derotation Standing single leg stand on painful side for 30 s	Gluteus medius m. or gluteus minimus m. insertion
Medial femoral condyle[4]	Lying supine, (1) hold thigh in place, attempt passive leg abduction with the knee extended and flexed 30°; (2) in either position, resist thigh adduction	(1) Tibial (medial) collateral lig., medial meniscus; (2) Adductor magnus
Patella, upper pole[2,5,14]	Lying supine, passive knee flexion; isometric quadriceps m. contraction	Quadriceps tendon at patellar insertion
Patella, lower pole[2,5,14]	Lying supine, passive knee flexion; isometric quadriceps m. contraction	Patellar tendon origin in patellar pole
Tibial tuberosity[2,5]	Lying supine, passive knee flexion; isometric quadriceps m. contraction	Patellar tendon insertion in tibial tuberosity
Calcaneus, middle posterior facet[1,3,4,5,14]	Standing on tiptoes Lying supine, resisted plantarflexion or passive foot dorsiflexion	Achilles tendon insertion in middle calcaneal facet
Medial process of calcaneal tuberosity[5,14]	Lying supine, passive toe dorsiflexion	Plantar fascia origin
Tibialis anterior insertion at the base of 1st metatarsal[14]	Lying supine or sitting, resisted foot dorsiflexion, resisted foot inversion	Medial base of 1st metatarsal, medial surface of 1st cuneiform

Pain should be at the enthesis. Upon pressure or traction, the patient may express pain spontaneously. If not, ask, "Did it cause pain? If so, show me where." A clinical diagnosis of enthesitis requires meeting all 3 criteria: current or recent (within the past 2 weeks) pain, local tenderness, and pain on resisted motion or traction. [a] Current or within the past 2 weeks at the entheseal site. [b] The pressure applied to enthesis sites, as in spondyloarthritis,[5] should be strong enough to blanch the distal nail. [c] Where there are several available maneuvers, pain caused by 1 maneuver counts as positive. Lig.: ligament; m.: muscle.

principle of equipoise would require that the sequential components of the traditional clinical method—namely the clinical history, the physical examination, and the US—be of the same quality. The rheumatologic history has been, traditionally and at present, detailed and precise. However, the physical examination has not kept pace with either the clinical history or the accuracy of US. A balanced clinical method can only add accuracy to a clinical diagnosis of enthesitis. The proposed improved physical examination of the entheses utilizes and extends resisted tension maneuvers advocated by La Cava and Niepel and Sit'aj[1,2] while attending to the applied pressure at entheseal sites.[5] Pulling from the inserting structure, whether tendon, capsule, or ligament, physiologically addresses the enthesis, which is the site where traction, through linearly arranged structures, reaches bone, its business end. Thus, defining enthesitis by mere tenderness on pressure misses the physiological relevance the test should have.

Table 1 shows the proposed upgraded physical examination. There are 2 overarching principles: (1) as in SpA,[5] the pressure applied to enthesis sites should be strong enough to blanch the distal nail; and (2) the loading maneuvers (resisted muscle pull, stress applied to a ligamentous or capsular enthesis) should resemble everyday loads. There is room for improvement in the loading maneuvers and the description of the anatomical items that cause the pain described herein. For example, a loading test for the posterosuperior iliac spine (PSIS) is hard to come by. PSIS centers the Venus fossa, relates to the interosseous sacroiliac ligament, and provides insertion to the thoracolumbar fascia (TLF).[18] Since the TLF has fine nerve endings and transmits force to the lower extremity directly by the gluteus maximus and indirectly by the hamstring muscles, the straight leg raise test might serve as a provocative maneuver.

The first column of Table 1 lists the sites where tendons, ligaments, and capsules meet bone, cause spontaneous pain in enthesitis, and are tender to pressure. Column 2 lists the proposed loading maneuvers. If ≥ 2 maneuvers are available at a given site, pain caused by one should count as positive. Column 3 names the structures that likely explain a positive loading maneuver. It may be impossible to know the actual structure(s) at fault (tendon, ligament, capsule, insertional bone) in complex areas, but this should not detract from the diagnostic value of the test. A clinical diagnosis of enthesitis should meet all 3 criteria: current or recent (within the past 2 weeks) pain, local tenderness, and pain on resisted motion or traction. A similar method should be applied at each of the chosen entheseal sites. A resisted muscle contraction test, although rarely mentioned,[14,15] is widely used by US researchers in assessing enthesitis and the applied digital pressure force is seldom estimated.[5]

Conclusion

There is a weak link in the clinical diagnosis chain of enthesitis—the physical examination of the entheses. This weakness is barely visible because the wondrous literature on enthesitis principally rests on US. However, rheumatologists may not have immediate access to expert US. Nevertheless, the situation is not hopeless. Pioneers in the enthesitis field considered that applying a tension load to the enthesis was critical for a physiological diagnosis of enthesitis, and their advice still stands today. In addition, a semi-standardized digital pressure applied to the entheses would improve diagnosis. Thus, acknowledging its wholly hypothetical nature, taking a step back to the method proposed by La Cava and Niepel and Sit'aj, and being consistent in the applied pressure to the entheses might mean a big step forward in the clinical diagnosis of enthesitis.

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