Intrinsic Muscle Spasm of the Hand; Bunnell's Sign

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Patients with full bodied hands are difficult to assess in terms of active synovitis of the metacarpophalangeal (MCP) joint. Traditional examination for soft tissue swelling can be difficult to discern. Lateral compression for tenderness may cause pain in normal hands, and is subjective. An understanding of Bunnell's sign, a test for intrinsic muscle spasm induced by MCP joint inflammation, is a valuable test for early MCP joint synovitis[1]. When applying this test, it is important to distinguish intrinsic muscle spasm due to MCP arthritis from proximal interphalangeal (PIP) joint tenderness, and from intrinsic muscle tightness due to fibrosis, shortening, deformity, or other causes of an intrinsic-plus hand.

Intrinsic muscles of the hand consist of the 4 *interossei dorsales*, 3 *interossei palmares*, and 4 *lumbricales* muscles, which originate in the hand, cross the MCP joints, and insert into the extensor hoods of the PIP and distal interphalangeal (DIP) joints (Figure 1). These intrinsic muscles are responsible for flexion of the MCP joint and extension of the PIP and DIP joints. The 4 *lumbricales* originate from the 4 profundus flexor tendons, and insert into the radial side of the PIP dorsal extensor expansion, where they serve to strengthen finger opposition to the thumb. The dorsal *interossei* originate from the shafts of adjacent metacarpal bones and have dual insertions into the base of the proximal phalanx and extensor hood, which contributes to flexion of the MCP joint and extension of the PIP and DIP joints. They are innervated by branches of the ulnar nerve. Ulnar nerve paralysis results in extension of the MCP joints and flexion of the PIP and DIP joints, which gives rise to a claw-hand deformity (Figure 2). This is sometimes referred to as intrinsic-minus position. Conversely, spasm or contracture of the intrinsic muscles causes flexion of the MCP joints and extension of the DIP and PIP joints, thus folding the extended fingers across the palm, which gives rise to a contracted hand, seen in carpal spasm, as in hyperventilation syndrome, or in advanced rheumatoid arthritis (RA) (Figure 3). This is referred to as intrinsic-plus position[2,3].

It is possible to take advantage of intrinsic muscle spasm, as a sign of MCP inflammation in diagnosing early small joint arthritis, by testing for Bunnell's sign. Because the intrinsic muscles span the MCP joints, any inflammation of adjacent MCP joints will cause varying degrees of intrinsic muscle spasm. Passive extension of the MCP joint will stretch already tight intrinsic muscles, and thus cause pain and spring-like tightness when the examiner passively flexes the PIP joint: Bunnell's sign (Figures 4A, 4B). Conversely, active extension of the MCP joint, by dorsal extensor tendons, and 90° flexion of the PIP and DIP joints, should place the fingertips on the palm's volar pad, and signifies lack of intrinsic spasm, or intrinsic-minus position[4].

PIP joint inflammation may confound this test for intrinsic muscle spasm secondary to MCP synovitis by also contributing to pain and resistance on passive PIP flexion. To control for PIP inflammation, the examiner should place the MCP joint in flexion, which relaxes the intrinsic muscles, thus eliminating intrinsic spasm as a cause of pain. The PIP joint may now be flexed without pain and resistance from intrinsic tightness (Figures 5A, 5B). Any resistance or pain caused by passive PIP flexion is now due to PIP joint inflammation[5]. Since PIP joint pain originates from stretching the joint capsule and compression of the PIP volar plate, this maneuver is often referred to as the volar plate test[3].

Bunnell's sign and the term intrinsic-plus hand were originally used by surgeons to describe intrinsic tightness due to fibrosis and tendon shortening[1,2]. Evolution of rheumatoid hand deformities results in contracture of intrinsic mechanism, but more commonly laxity of collateral and sling liga-

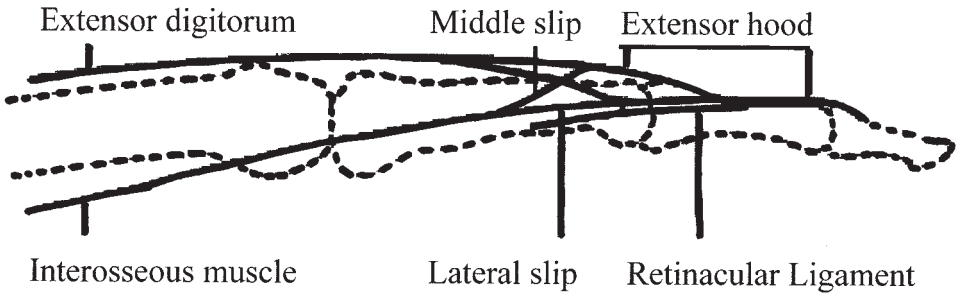


Figure 1. The interosseous muscle traverses the MCP joint, and inserts in the PIP and DIP extensor hood.

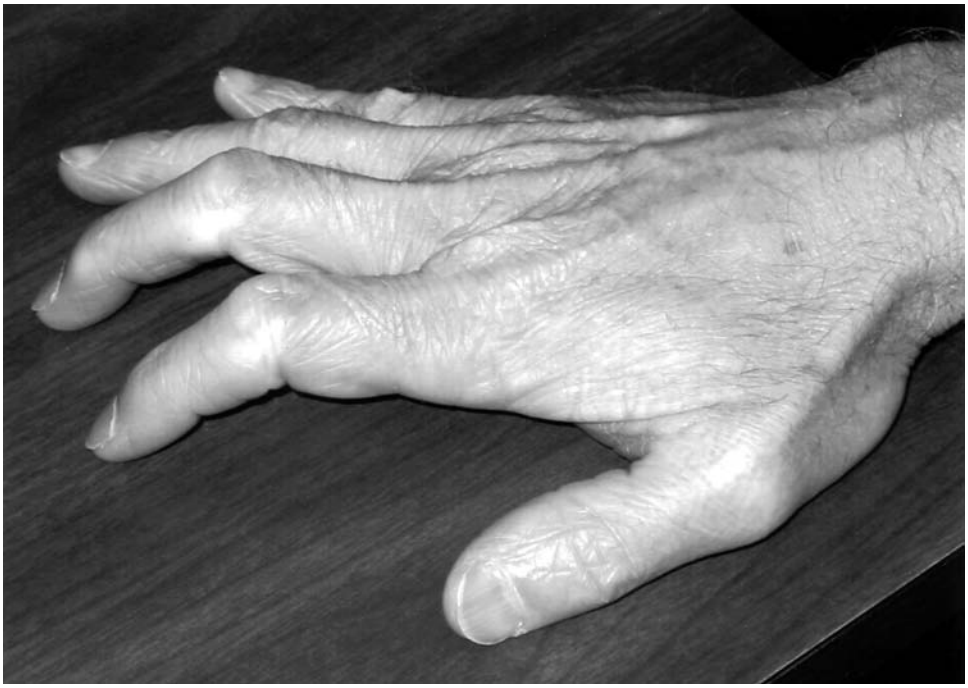


Figure 2. Claw hand (simulated): intrinsic muscle weakness with unopposed long extensor action on MCP joints, and clawing of the fingers due to unopposed long flexors, exemplified by ulnar nerve paresis. This is the intrinsic-minus position. Normally, intact intrinsic muscles would flex the MCP joint and extend the PIP and DIP joints.

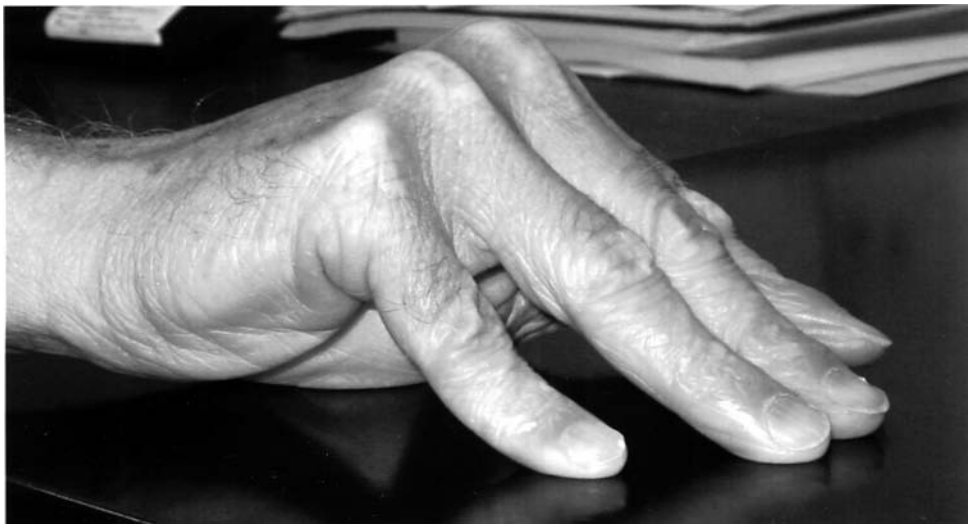
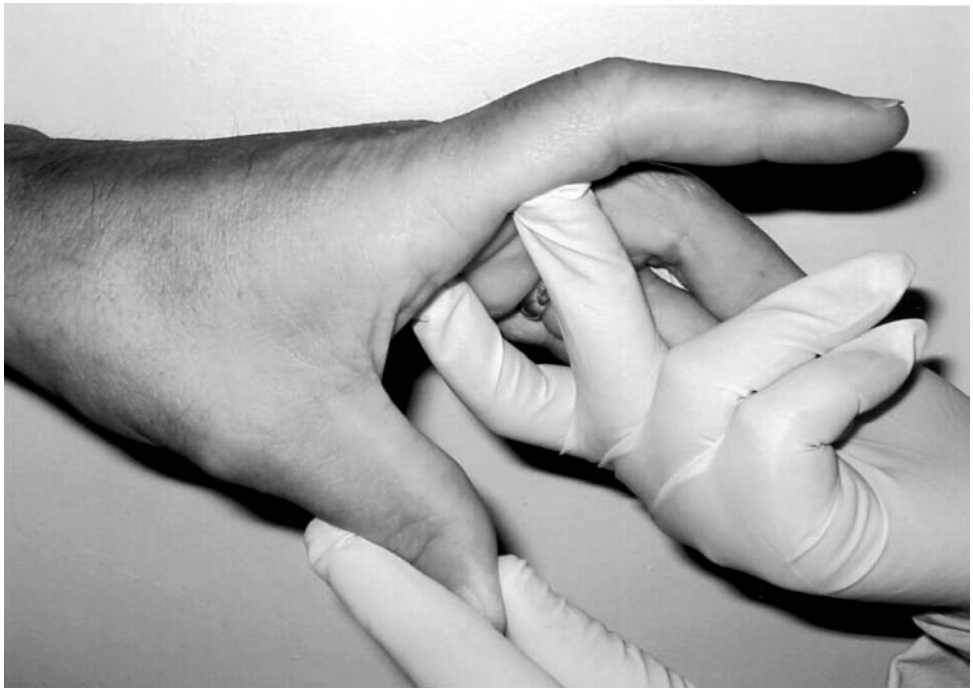


Figure 3. Intrinsic muscle spasm causing flexion of the MCP joints and extension of the phalanges. Simulated here, but exemplified by carpal spasm in patients with hyperventilation syndrome. This is the intrinsic-plus position.

ments, thickening of flexor tendon sheaths, volar plate dislocation, MCP subluxation, and ulnar deviation, all of which serve to confound Bunnell's sign as an indicator of MCP joint inflammation. Lax and misaligned intrinsic-plus mechanisms no longer respond to passive MCP extension and PIP flexion, as a test of intrinsic muscle spasm, seen best in early joint inflammation, i.e., Bunnell's sign[6,7]. Exclusion of these patients should not interfere with the value of Bunnell's sign as a test for subtle MCP arthritis in early small-joint arthritis, when deformity is not yet likely. The clinical usefulness of Bunnell's sign for clinicians today is detection of intrinsic muscle spasm as a subtle and early sign of MCP inflammatory arthritis.

Reference to Bunnell's sign has been missing in recent textbooks and from reviews of physical diagnosis, orthopedics, and rheumatology. If we are to resurrect this clinically useful sign, it might better be referred to as intrinsic muscle spasm in the setting of early synovitis, due to contracture of



A

B

Figure 4. A. Bunnell's sign: extend the MCP joint to stretch the intrinsic muscles. B. While holding MCP extension, flex the PIP joint to further stretch the intrinsic muscles, which will be tight and painful if there is spasm of the intrinsic muscles due to MCP joint inflammation.

intrinsic muscles as they span an inflamed MCP joint, and to intrinsic muscle tightness in later stages of RA due to fibrosis and deformity. As eponym antagonists, we suggest emphasis on the clinical aspect of Bunnell's sign by referring to it as a test of the intrinsic-plus hand. The differential diagnosis of intrinsic muscle tightness includes MCP joint inflammation, local infection, fibrosis, acute muscle injury, chronic muscle or tendon scarring, joint subluxation, trauma, overuse, carpal-pedal spasm, and Parkinson's disease. In the setting of morning stiffness and polycyclic, symmetric, small-joint arthritis, Bunnell's sign becomes more specific for MCP inflammatory arthritis, as seen in early RA.

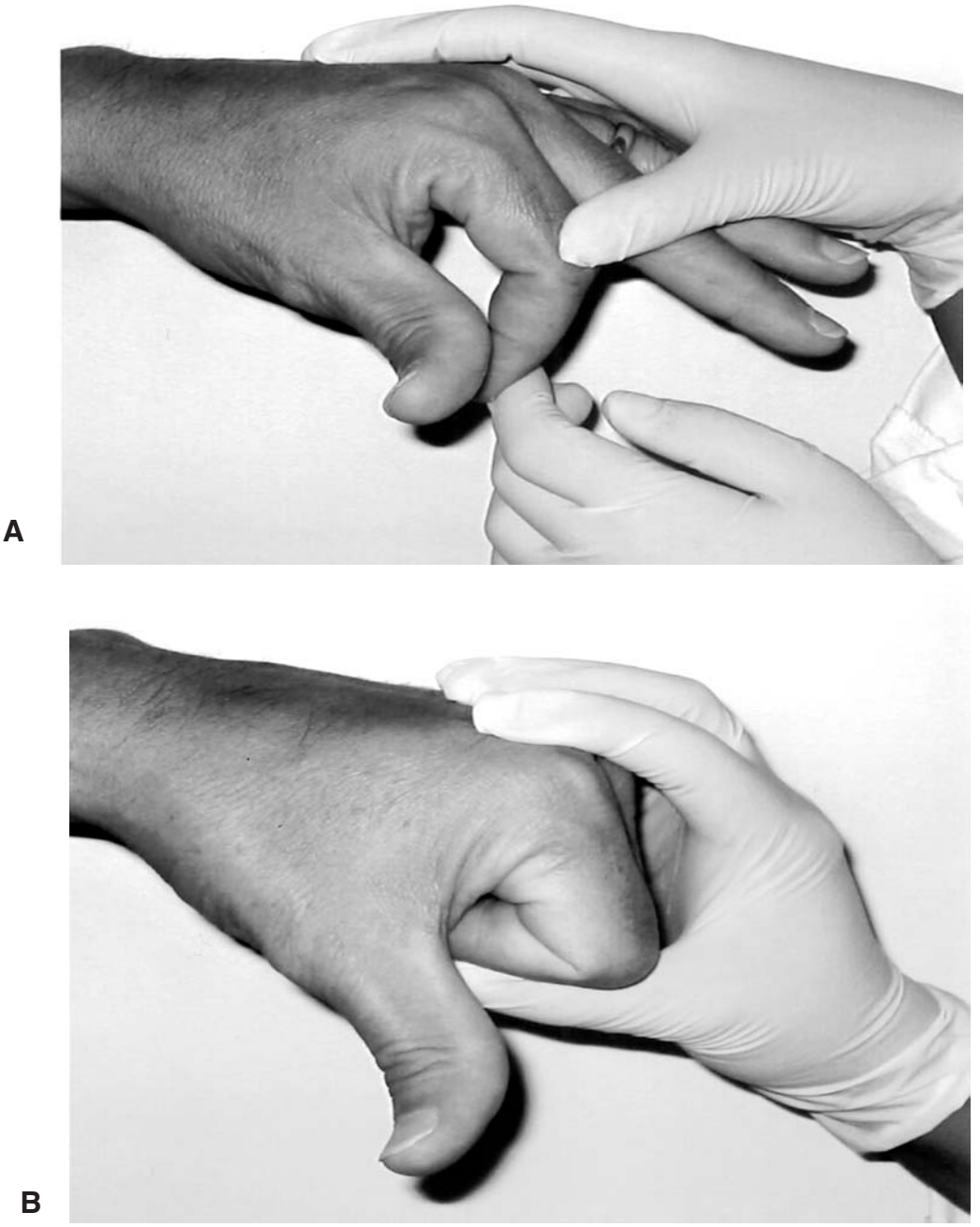


Figure 5. A. Bunnell's sign control: flex the MCP joint to relax the intrinsic muscles. B. While holding MCP flexion, flex the PIP joint, which will now be lax and painful only if the PIP joint is inflamed. This is referred to as the volar plate test.

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