Understanding Midcarpal Instability

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This article outlines the historical development of midcarpal instability observations and terminology and places them in the broader context of currently accepted theories of wrist pathomechanics. Such an understanding may help resolve the following questions: Are there 1 or more entities under the current designation of midcarpal instability? What are the underlying pathogenesis and pathomechanics of the disorder(s)? What are the recommended treatment options? What further research needs to be done to better answer these questions?

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The concept of midcarpal joint instability (MCI) has evolved slowly since it was first described by Mouchet and Belot in 1934. Subsequent researchers have not agreed about whether the differing personal observations of MCI represent distinct clinical entities or rather different manifestations of the same (or very similar) conditions. Lacking unanimity in regard to the etiology and pathomechanics of this disorder it is easy to understand why there is also a lack of agreement on terminology and treatment for instability of the midcarpal joint. Even the name “midcarpal instability” is not accepted universally as the proper designation for this condition.

Wrist Mechanics

Navarro introduced the concept of the columnar carpus in 1921. In 1943 Gilford et al. popularized Lambrinudi’s concept of the wrist as a link mechanism in which the radius, proximal carpal row, and distal carpal row comprise the individual links. Fisk expanded on Gilford’s link theory noting the importance of the volar ligaments. He first discussed pathologic carpal instability in 1970, noting carpal collapse occurring after trauma or as a late sequela of Kienböck’s disease.

Linscheid and associates further developed the concept of carpal instability by describing 2 distinct patterns of intercarpal collapse: volar intercalated segmental instability (VISI) and dorsal intercalated segmental instability (DISI). Their designation is based on the longitudinal axis of the lunate relative to the capitate on the lateral radiograph. In DISI the lunate is displaced volarly and extended. In VISI the lunate is displaced dorsally and flexed. In addition they introduced the slider-crank analogy to explain the scaphoid’s role in preventing intercarpal collapse while controlling relative intercarpal motion.

In 1976 Taleisnik modified Navarro’s columnar theory of wrist kinematics based on established patterns of carpal instability. In Taleisnik’s model the distal row along with the lunate becomes the central flexion and extension column. The scaphoid forms the mobile lateral column and the triquetrum represents the rotary medial column. Based on this concept carpal instabilities presumably would occur in columnar or longitudinal patterns. The ring concept was developed to accommodate for the pathogenesis of perilunate and transverse carpal instabilities and to explain the complex kinetic relationship between the proximal and distal rows. The 2 rows of carpal bones form a ring with 2 physiologic links: the mobile scaphotrapeziotrapezoid (STT) joint (radial link) and the rotary triquetrohamate joint (ulnar link). These links, assisted by dynamic joint reactive forces across the midcarpal joint, create reciprocal motion between the proximal and distal carpal rows. Radial deviation creates an unbalanced flexion moment at the radial (STT) link inducing proximal row flexion while the capitate and hamate move palmarly (physiologic VISI). The opposite occurs with ulnar deviation. An unbalanced extension moment at the ulnar link causes the triquetrum to extend against the hamate with the proximal row following and the hamate and capitate moving dorsally (physiologic DISI). In neutral deviation the opposing moments at
the radial and ulnar links are balanced and the proximal row remains stationary (in neutral flexion/extension).

More recently Moritomo et al have shown that the midcarpal joint allows radiodorsal/ulnopalmar motion that coincides with the so-called dart-throwing motion. This motion is one of the most frequently used wrist motions in activities of daily living.

In response to the growing number of described carpal instability patterns Dobyns and coworkers divided them into 2 groups: carpal instability dissociative and carpal instability nondissociative (CIND). Carpal instability dissociative was defined as a true disruption of intrinsic intercarpal ligaments, most commonly within the proximal row, resulting in instability between adjacent carpal bones (eg, lunotriquetral or scapholunate). In CIND there is laxity or overstretching of the extrinsic ligaments with intermittent carpal subluxation. The term CIND has been used most often in conjunction with transverse instabilities at the midcarpal joint. Radiocarpal instability also can occur and this is usually secondary to trauma.

Lichtman classified carpal instabilities by using a combination of these various concepts (Table 1). He divided carpal instabilities into perilunate (scapholunate and lunotriquetral) instabilities, MCIs, and radiocarpal instabilities. Although perilunate and radiocarpal injuries are usually dissociative (loss of ligament integrity) and midcarpal injuries are usually nondissociative (ligament laxity) theoretically all 3 categories may represent dissociative or nondissociative lesions from a pathologic standpoint.

### History of Midcarpal Observations and Terminology

Laxity of the midcarpal joint first was described by Mouchet and Belot in 1934 using the phrase “snapping wrist.” A decade later Suturo described 2 patients with painful palmar subluxation of the distal carpal row with palmarward-directed pressure over the distal carpus. It was not until the 1980s, however, after the presentation of a series of patients, that the concept of MCI was recognized as a true clinical disorder. Since then there has been increasing interest and awareness of instabilities of the midcarpal joint. Along with this interest has come an abundance of innovative nomenclature.

Ulcer and Palmar MCI

In 1981 Lichtman et al described a small series of patients presenting with a volar sag at the midcarpal joint and a history of a painful and spontaneous clunk occurring with ulnar deviation and pronation of the wrist (Fig. 1). Tenderness to palpation was noted over the ulnar carpus especially at the triquetrohamate joint. Lichtman and colleagues initially referred to this clinical entity as ulnar MCI. In 1993 they renamed it palmar MCI (PMCI) to distinguish it from this clinical entity as ulnar MCI. In 1993 they renamed it palmar MCI (PMCI) to distinguish it from an entity described in several reports where the midcarpal subluxation appeared to be occurring in the dorsal direction.

Routine x-rays generally showed a mild VISI pattern in the neutral position but were otherwise normal (Fig. 2). Videofluoroscopy was diagnostic in every case. Careful scrutiny of the videos indicated that the proximal row was not rotating smoothly from flexion to extension as the wrist moved from radial to ulnar deviation. Instead the proximal row maintained a position where the radius was in varus and the midcarpal joint allowed radiodorsal/ulnopalmar motion. Videofluoroscopy was diagnostic in every case. Careful scrutiny of the videos indicated that the proximal row was not rotating smoothly from flexion to extension as the wrist moved from radial to ulnar deviation. Instead the proximal row maintained a position where the radius was in varus and the midcarpal joint allowed radiodorsal/ulnopalmar motion. Instead the proximal row maintained a position where the radius was in varus and the midcarpal joint allowed radiodorsal/ulnopalmar motion.

As described this test is performed by placing the patient’s wrist in neutral with the forearm pronated. A palmar force then is applied to the hand at the level of the distal capitate. The wrist simultaneously is loaded axially and deviated ulnarly. The test result is
positive if a painful clunk occurs that reproduces the patient’s symptoms.

In an attempt to define the etiology and pathomechanics of MCI the researchers tried to reproduce it \textit{in vitro} in the fresh cadaveric wrist. Sectioning of the dorsal and ulnar triquetrohamate ligament (capsule) produced only slight midcarpal laxity and no clunk; however, division of the stout ulnar limb of the palmar arcuate (triquetrohamate-capitate) ligament resulted in a clunk similar—but not identical—to that observed in their patients.\textsuperscript{7} The researchers acknowledged that precise laboratory reproduction of the clinical entity was difficult, leaving some doubt as to the exact etiology and pathogenesis.

In 1988 Trumble et al\textsuperscript{18} reported a series of patients with symptoms similar to those described by Lichtman.\textsuperscript{7} Subsequent studies by Trumble et al\textsuperscript{19} and Viegas et al\textsuperscript{20} have shown that sectioning either the ulnar arm of the palmar arcuate ligament or the dorsal radiotriquetral ligament can produce a VISI deformity and wrist pathomechanics characteristic of PMCI (Figs. 3, 4). More recently Lichtman\textsuperscript{21} showed \textit{in vivo} that tightening the dorsal radiotriquetral ligament alone can stabilize the proximal row and eliminate the clunk of PMCI, thus emphasizing the potential importance of dorsal extrinsic ligament laxity in the pathogenesis of this disorder.

Based on these anatomic studies Lichtman et al\textsuperscript{15} now believe that PMCI is caused by laxity of both the dorsal radiotriquetral and palmar ulnar arcuate ligaments, allowing the head of the capitate and hamate to sag in the midcarpal joint. They believe that this volar translation induces or occurs in conjunction with a VISI pattern across the entire proximal row. In this subluxated position the normal joint reactive forces cannot develop between the proximal and distal rows. As a result the smooth transition from proximal row flexion to extension does not occur with ulnar wrist deviation. The VISI pattern (proximal row flexion) remains constant until the extreme of ulnar deviation has been reached and the forces across the triquetrohamate joint are re-engaged. At that time a rapid catch-up clunk occurs as the proximal row jumps from excessive flexion into physiologic extension and the head of the capitate and hamate are relocated completely.

As initial treatment Lichtman et al\textsuperscript{22} described nonsurgical therapy consisting of activity modification, nonsteroidal anti-inflammatory drugs, and splinting. Dorsally directed pressure under the pis
form reduced the carpal sag along with the VISI position of the proximal row. Applying this principle a 3-point dynamic splint maintaining reduction was used successfully to permit wrist motion in milder cases. Surgical reconstruction was recommended when nonsurgical treatment failed.

The early surgical reconstruction for PMCI by Lichtman and colleagues consisted of stabilizing the triquetrohamate joint by rerouting the extensor carpi ulnaris tendon. Later, based on preliminary anatomic studies they advanced the ulnar arm of the volar arcuate ligament across the midcarpal joint. After the failure of some of these soft-tissue reconstructions they began to perform limited midcarpal arthrodesis (triquetrohamate or capitolunate-triquetrohamate). In 1993 Lichtman et al compared the results of their midcarpal fusions with soft-tissue reconstruction in 13 patients who had 15 surgical procedures. The results of this study confirmed the advantage of midcarpal arthrodesis; however, in milder cases Lichtman still performs reefing of the dorsal radiotriquetral ligament. The long-term results of dorsal capsule reefing have not been reported.

Rao and Culver have noted less success treating MCI with limited midcarpal fusion. Based on their scoring system triquetrohamate arthrodesis was successful in only 6 of 11 cases. Pain, however, usually was relieved and clunking was eliminated in all patients. Consequently the surgeons still prefer limited arthrodesis to ligamentous reconstruction for treatment of symptomatic MCI.

Goldfarb et al reported results similar to those of Lichtman: 7 of 8 patients with PMCI were satisfied with four-corner arthrodesis and 6 of 8 had no or mild pain.

Capitolunate Instability Pattern
In 1984 Louis et al described a series of 11 patients with a chief complaint of pain and clicking in the wrist. Tight grasping, particularly in supination, aggravated their symptoms. The authors described a dynamic dorsal displacement test that was helpful in establishing the diagnosis. To perform the test they applied dorsally directed pressure to the scaphoid tuberosity while longitudinal traction and flexion were applied to the wrist. Videofluoroscopy of these maneuvers showed dorsal subluxation of the proximal row and almost complete dorsal subluxation of the capitate from the lunate. This examination reproduced the patients’ symptoms. Plain radiographs and arthrography were not helpful.

Louis et al termed this presentation capitolunate instability pattern (CLIP) wrist. They believed that dynamic laxity of the radiolunate ligaments and extrinsic stabilizers of the scaphoid were likely responsible for the instability. In addition they postulated that laxity of the dorsal capitolunate ligament complex could allow displacement of the capitate from the lunate. They recommended avoidance or modification of specific activities that exacerbate the symptoms. Ten of the 11 patients became asymptomatic.
with nonsurgical treatment. Ono et al.\textsuperscript{26} however, have reported that nonsurgical treatment did not provide consistently long-term pain relief in their patients with CLIP wrist.

Chronic Capitohamate Instability
Johnson and Carrera\textsuperscript{14} used the term chronic capitohamate instability (CCI) to describe the clinical presentation of a series of 12 patients with complaints of posttraumatic chronic pain, weakness, and wrist clicking. All patients had a prior extension injury to the wrist.

Standard orthopedic examination and plain radiographs were unremarkable except in 1 patient whose radiographs showed a DISI pattern. A dorsal capitohamate-displacement stress test under fluoroscopy showed dorsal subluxation of the capitate on the lunate, associated with patients’ becoming apprehensive when the test was performed. There was an associated painful click as the lunate abruptly shifted dorsal and ulnar. They noted that the instability was located primarily at the level of the capitohamate joint. They believed the condition was attributable to attenuation of the palmar radiocapitate ligament caused by trauma.

Johnson and Carrera\textsuperscript{14} performed surgery on 11 of 12 patients. Although they could not identify the site of injury, the surgery consisted of suturing the volar radiocapitate ligament to the radiotriquetral ligament, partially or completely closing the space of Poirier. After surgery there was some loss of wrist extension; however, both the lunate and capitate were stabilized effectively. At an average follow-up time of 52 months results were excellent in 6 patients, good in 3, fair in 1, and poor in 1.

In 1996 Apergis\textsuperscript{27} reported a series of 14 patients with chronic wrist pain, numbness, and reduction of grip strength. Half of the patients reported an extension injury to the symptomatic wrist. Physical examination showed palmar sagging of the wrists and a positive dorsal capitohamate-displacement stress test result as described by Johnson and Carrera.\textsuperscript{14} Almost all of the patients were noted to have generalized ligamentous laxity. Radiographs in all cases showed a VISI deformity. Videofluoroscopy performed during the dorsal displacement test showed dorsal subluxation of the capitohamate or the capitohamate and radiolunate joints. None of the patients responded to nonsurgical treatment. The wrists were stabilized by suturing the volar radioscaphocapitate to the long radiolunate ligament on the radial side and tethering the capitohamate and lunotriquetral ligaments on the ulnar side. Results were excellent in 8 cases; good in 5; and fair in 1.

Carpal Instability Nondissociative
In 1994 Wright et al.\textsuperscript{28} described a series of patients with similar clinical characteristics and radiographic findings to those reported by Lichtman et al.\textsuperscript{7,15} and possibly also by Louis et al.\textsuperscript{13} and Johnson and Carrera.\textsuperscript{14} On physical examination their patients had a painful clunk with ulnar deviation of the wrist.

Most patients had normal plain radiographs; occasionally radiographs showed a VISI pattern at rest or, rarely, a DISI pattern. They noted that videofluoroscopy was extremely helpful in making the diagnosis.

In addition Wright et al.\textsuperscript{28} observed similar symptoms in several patients with an ulna-minus variance, usually accompanied by a lunate fossa that had an increased radioulnar slope on anteroposterior radiographs. They noted that exerting pressure directed radially on the triquetrum during radial ulnar deviation in some patients prevented the clunk from occurring. They suggested that the lunate tended to “hang up” on the ulnar edge of the lunate fossa going from radial to ulnar deviation. They also suggested that ulnar translation of the proximal carpal row would increase susceptibility to instability.

Nakamura et al.\textsuperscript{29} since have shown that the kinematics of a type I lunate are different from those of a type II lunate during radial to ulnar deviation of the wrist. It is possible that the morphology of the lunate also may contribute to the pathology noted in the patients in Wright’s group with an ulna-minus variant.

Wright et al.\textsuperscript{28} commented that the pathology of MCI was understood poorly. They found the mechanism of injury to be inconsistent: many of the patients were noted to have ligament laxity with half of the patients reporting trivial or no history of trauma. The term CIND was suggested by these researchers rather than MCI because they believed that the pathology responsible for the instability patterns could involve ligament insufficiency or joint malalignment found at the midcarpal level, radiocarpal level, or both. In addition they believed that extrinsic factors could contribute to this condition as well.

Wright et al.\textsuperscript{28} recommended direct repair of the injury site in the rare case in which it could be discovered. Otherwise various dorsal and volar approaches were used to imbricate selected ligaments for both the CIND-VISI and CIND-DISI varieties. In those individuals with CIND and ulna-minus wrists radial shortening was recommended. Radiocarpal or midcarpal fusion was suggested for those with an unknown source of pathology, for those who required additional release to correct the deformity, for those who required excessive force to maintain reduction, and for those with severe joint destruction.
The authors recommended surgery only after a patient failed nonsurgical treatment. In their series 38 of the 45 patients diagnosed with CIND were treated surgically according to the above guidelines. Fifty-seven percent of the patients had good to excellent results determined by the Mayo modified wrist scores. Patients with an ulna-minus variant may have benefited the most, with 83% of patients having good to excellent results.

As in the previously described series this study again failed to identify a consistent anatomic source for the instability pattern; however, this study reinforces the previously noted dorsal and volar directional vectors for the subluxations (CIND-VISI and CIND-DISI) and, additionally, introduces a new anatomic association: ulnar-minus variance.

Radial Midcarpal Instability
Caputo et al classified MCI into 4 types, depending on whether the pathology involved the ulnar or radial side of the wrist. Types I and II involved the ulnar side and were believed to be similar to the patients in Lichtman’s series. Types III and IV were applied to patients with rotary subluxation of the scaphoid. The researchers believed that these cases represented radial-sided periscaphoid ligament laxity. In type III MCI the scapholunate ligament was intact, with ligament laxity at the STT joint. Type IV was the most severe form, associated with scapholunate ligament disruption.

The researchers believed that many of the previously reported MCI cases for which ulnar-sided soft-tissue repair failed were, in fact, type III or type IV lesions. Their patients with radial MCI presented with pain over the dorsal aspect of the wrist, scaphoid tenderness, a catch-up clunk, a positive resisted finger extension test result, and/or a positive scaphoid shift test result with scaphoid subluxation. They suggested that treatment of radial MCI should be directed toward midcarpal stabilization. Their suggested technique was STT fusion.

Midcarpal Instability Caused by Malunited Fractures of the Distal Radius
Linscheid and colleagues first recognized carpal instability of the wrist occurring as a result of distal radius malunion in 1972. Subsequently Taleisnik and Watson reported 13 patients with symptoms of midcarpal pain and instability after sustaining malunited fractures of the distal radius. Clinically their patients had tenderness at the lunocapitate and triquetrohamatate joints. Approximately half of them had intermittent midcarpal subluxation, producing a painful audible snap at the triquetrohamate joint when ulnarly deviating the wrist with the forearm pronated.

The remaining patients had a symptomatic midcarpal synovitis without demonstrable subluxation. Lateral radiographs of the distal radius showed an average dorsal tilt of 23° whereas the normal palmar tilt is 11°. The lunate had migrated dorsally and was angled palmarly to compensate for the dorsal radial displacement. Finally the capitate was collinear with the lunate but dorsal to a longitudinal loadline of the proximal radius.

The instability was attributed by the researchers to repetitive overload of the midcarpal joint as a result of reversal of the normal palmar tilt of the distal radius. Under these circumstances they reasoned that the otherwise intact carpal ligaments were incapable of preventing excessive dorsal translation of the capitate. Ulnar deviation therefore produced a clunk with dorsal translocation.

Taleisnik and Watson reported good results with corrective osteotomy of the distal radius. Currently Watson no longer considers this condition MCI but uses the term dorsal radius angulation carpal overload to describe the clinical entity.

Discussion
Based on a compilation of the above studies and the contained data there are some common threads that can be of assistance in conceptualizing the pathomechanics of MCI. It appears that MCI represents several distinct clinical entities differing in the cause and direction of subluxation but sharing the common characteristic of abnormal force transmission at the midcarpal joint. In each published clinical study the researchers describe either a palmar, dorsal, or both palmar and dorsal directional vector for the subluxation.

The mechanism of the clunk in the palmar pattern has been studied and described by Lichtman and colleagues. Loose dorsal radiotriquetral and volar ulnar arcuate ligament support permits an excessive volar sag (VISI) of the proximal row in the nonstressed wrist. This sag results in a loss of joint contact across the midcarpal joint, which manifests clinically as a loss of the smooth transition of the proximal row from flexion into extension as the wrist deviates ulnarward. Complete ulnar deviation causes a sudden reversal of the VISI as the helicoidal triquetrohamate joint is re-engaged and physiologic joint contact forces are brought into play. The sudden proximal row extension (DSI) causes the painful clunk. As the wrist moves back to neutral the ulnar forces are disengaged and the proximal row drops back into VISI while the distal row again settles palmarly into its slightly subluxated starting point.

The dorsal pattern subluxations have not been described or visualized as extensively. From the
cases of Johnson and Carrera\textsuperscript{14} and Apergis\textsuperscript{27} it appears that volar ligament laxity, especially on the radial side, permits the capitate and lunate to translate dorsally to an excessive degree, especially with ulnar deviation of the wrist. In both of those series volar radial reefing corrected the dorsal translation of the distal row.

Taleisnik and Watson’s\textsuperscript{31} study is very enlightening because the findings of dorsally directed MCI (similar to CLIP, CCI, and CIND-DISI) occurred in patients with presumably normal wrist ligaments. Here a deformity extrinsic to the wrist created an abnormal alignment of carpal bones, altering the normal transmission of forces across the midcarpal joint. Another possible explanation for the dorsal subluxation, in addition to that noted by the researchers, is that reversal of the normal volar tilt of the distal radius effectively slackened the volar extrinsic wrist ligaments (decreased the distance from their origin to insertion), making them less effective in restraining dorsal subluxation of the distal carpal row. This would create a similar situation to the CCI wrist described by Johnson and Carrera\textsuperscript{14} CCI the CLIP wrist described by Louis et al.\textsuperscript{13} The fact that corrective osteotomy eliminated the clunk reinforces the theory that re-establishing correct tension of the volar ligaments also corrects the tendency for dorsal migration of the lunate and capitate.

It should be noted that in both the palmar and dorsal patterns the proximal row always moves into extension and the distal row translates dorsally with ulnar deviation. Where this transition is smooth it represents normal wrist kinematics. It is the timing and force of this movement that differentiates the 2 patterns and creates the pathologic clinical patterns of MCI. In the VISI pattern it is the initial volar translation (subluxation) in neutral that reduces in ulnar deviation whereas in the DISI pattern the wrist is reduced in neutral and the dorsal subluxation occurs in ulnar deviation. In either case the instability is caused primarily by true or relative laxity of selected ligaments supporting the proximal row and their failure to control the complex kinematic relationships between the articular surfaces across the midcarpal joint.

### Consolidated Classification of Midcarpal Instability

Midcarpal instability also can be classified into extrinsic and intrinsic forms. Intrinsic MCI is characterized by laxity of wrist ligaments whereas extrinsic forms are secondary to bone abnormalities outside the carpus. The extrinsic form is exemplified by cases that occur after distal radius malunion and those associated with ulnar-minus variance. As noted the intrinsic form is subdivided further into dorsal, palmar, and dorsal/palmar types. The intrinsic dorsal type is best represented by cases from the CLIP wrist series of Louis et al\textsuperscript{13} and the CCI series of Johnson and Carrera.\textsuperscript{14} The intrinsic palmar type is found in the series of Lichtman et al.\textsuperscript{7,15} and Caputo et al.\textsuperscript{30} The cases presented by Apergis\textsuperscript{27} have characteristics of both types and may represent a more universal ligament laxity pattern. Cases from the series of Wright et al\textsuperscript{28} fall into all of the above categories, including extrinsic forms. This classification scheme may be useful for treatment recommendations and is presented in Table 2.

### Treatment Recommendations

If a specific case of MCI can be placed into 1 of the categories outlined above treatment recommendations then can be matched appropriately. Clearly if ligament laxity can be traced to a direct site specific ligament augmentation is indicated. For extrinsic forms of MCI radial osteotomy or ulnar lengthening, depending on the pathology, have been reported to be curative. For palmar intrinsic forms milder cases can respond to dorsal radiotriquetral ligament reefing. More severe PMCI has been shown to respond best to midcarpal fusion, either ulnar (four-corner) or perhaps radial (STT). Dorsal intrinsic MCI (CLIP wrist, CCI) may respond to nonsurgical treatment as in the series of Louis et al\textsuperscript{13}; however, if surgery is indicated palmar ligament reefing (radiotriquetral to radiolunate) has been effective in most cases.

Although we have come a long way since Mouchet and Belot’s article\textsuperscript{1} was published in 1934 much of our current understanding of MCI is still based on theory, conjecture, and insufficient scientific evidence. Available data heretofore have not been synthesized into an acceptable systematic overview of the various components of MCI. To move forward the next step will be to agree on a common terminology and classification system for these interrelated clinical entities that concurs with what each investigator is seeing. Concurrently each entity should be reproduced anatomically in the laboratory and biomechanical markers should be established to ascertain that the in vitro reproduction is a true model of the clinical condition being studied. Finally with
the above steps accomplished various ligament and bony reconstructions can be devised and tested to determine the best surgical options for each clinical component of MCI.

It is hoped that this review has provided a sufficient overview of the subject to enable the reader to see some common integral threads in the published material and to encourage investigators to accelerate the process of clinical and biomechanical verification of the various published observations regarding MCI. Unless this process begins soon and leads to a more scientific approach we will continue to rely for many years on a very hazy picture of MCI when trying to determine the appropriate diagnosis and treatment options in the clinical setting.

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References