REVIEW

Midcarpal Instability: Unlocking the Secrets of the Wrist

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ABSTRACT

Our attempt to understand the pathology leading to midcarpal instability (MCI) over the previous 40 years has allowed us to not only better describe the pathomechanics of the condition, but also guide treatment decisions, and even propose a theory of carpal kinematics which seems to describe multiple carpal instability patterns. Pathology of the dorsal radiotriquetral ligament (DRT) has been identified as an etiology for MCI, and reefing of this ligament is now the recommended surgical treatment. Furthermore, nonsurgical proprioceptive training allows for the avoidance of surgical intervention for many patients with MCI. We describe our experience with MCI, from our earliest patient encounters, from which we gained insight into the importance of the DRT, to the observation that nonsurgical symptomatic management with functional splinting and proprioceptive training could alleviate many patient complaints and allow for a return to full activity.

Level of Evidence: V; Descriptive review/Expert opinions.

Keywords: Midcarpal instability; Carpal kinematics; Dorsal radiotriquetral ligament; Wrist.

INTRODUCTION

Instability of the midcarpal joint, like many conditions in medicine, had been observed and was called many different names before anyone realized they were probably describing the same entity. Mouchet & Belot [1] in 1934 were the first to document asymptomatic subluxation at the midcarpal joint, which they called the "snapping wrist." In 1984, White & Louis [2] described a patient with wrist instability that they termed capitolunate instability pattern, or CLIP wrist. In 1986, Johnson & Carrera [3] published a series on patients with chronic capitolunate instability (CCI). These authors attributed this condition to laxity of intrinsic wrist ligaments, but did not attempt to explain the pathomechanics. In retrospect, all these reports probably represented variants of midcarpal instability (MCI).

A major reason for the delayed recognition of MCI is that almost all wrist clicks and clunks were originally thought to be due to scapholunate (SL) or lunotriquetral (LT) instability. This overlap, along with the historically poor understanding of carpal

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pathomechanics, made the identification and description of MCI challenging. Our experience with MCI over the years, however, has allowed us to gain a better understanding of this unique entity. Along the way, it has revealed several other "secrets" of the wrist as well.

Our first encounter with MCI was in 1976 at the Bethesda Naval hospital. A female surgical technician had undergone operative treatment elsewhere for dorsal dislocation of the ulna. Intraoperatively, no pathology had been identified and, as such, no repair was performed. The patient presented to us with a painful wrist clunk with ulnar deviation that interfered with her job activities. Examination revealed a palmar sag on the ulnar aspect of the wrist in neutral deviation, giving the clinical appearance of a dorsal dislocation of the distal radio ulnar joint (DRUJ). Examination of the DRUJ was unremarkable and stable to stress. Passive and active ulnar deviation with the forearm pronated reproduced the painful clunk at the wrist. Radiographs revealed a mild volar intercalated segment instability (VISI) pattern. These findings were reported in 1981 as part of a small case series [4].

The initial thought for this patient was SL instability, as this was the cause of wrist noises in the 1970s. Repeated examinations of the wrist revealed some distinctions, however. First, the palmar sag disappeared (with a clunk) as the wrist moved into ulnar deviation. As the wrist moved back to neutral, the palmar sag reappeared. Second, this was not really a click, as heard with SL dissociation, but a distinct clunk, which implied joint subluxation or dislocation (as in the hip). The final distinction was that the location of pain accompanying the clunk was at the ulnar corner of the midcarpal joint, not at the DRUJ or at the anatomic snuffbox.

The patient agreed to a second wrist exploration. Through a dorsal ulnar incision, the proximal carpal row was noted to be normal in appearance. The ulnar stabilizers of the midcarpal joint demonstrated laxity, enough to permit excessive proximal row rotation and dorsal-palmar translation between the proximal and distal rows. This laxity was most pronounced with the wrist in neutral. As the wrist was brought into ulnar deviation, the proximal carpal row was visualized flipping into extension, accompanied by an audible clunk.

There was insufficient capsular tissue to stabilize the triquetrohamate (TH) and capitolunate (CL) joints in this patient, so a formal ligament reconstruction was performed using the distal extensor carpi ulnaris tendon woven in a dorsal to palmar direction to stabilize the ulnar corner of the midcarpal joint. As there was no other term to describe the findings, we named this entity "ulnar midcarpal instability." The patient returned to full duty several months postoperatively. We were pleased to have solved the patient's problem, but were puzzled by the unique findings and lack of specific information available in the literature.

Soon after, a second patient presented with similar complaints and findings (Figure 1A). This patient, an academic radiologist at our hospital, had difficulty playing tennis due to the painful wrist clunk. A tri-compartmental wrist arthrogram was performed, which was within normal limits. Fluoroscopic videos were taken as he actively recreated his wrist clunk in an attempt to identify any areas of dissociation or abnormal carpal motion. As the wrist moved into ulnar deviation, the entire proximal row flipped from flexion to extension.

This sudden rotation was simultaneous with the painful wrist clunk. As the wrist was moved back into neutral, a VISI deformity reappeared. These fluoroscopic findings correlated perfectly with the surgical findings of our previous patient.

Clinically, when a dorsally directed force was placed on the pisiform in this patient, not only was the palmar sag corrected (Figure 1B), but the wrist would no longer clunk as it moved from neutral to ulnar deviation. A palmar splint was created to maintain this position with constant 3-point fixation while still permitting active range of motion of the wrist. The patient was able to return to playing tennis using this splint and never required surgical intervention.
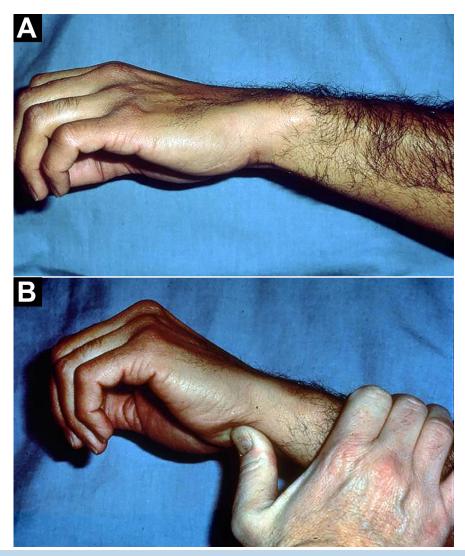

Figure 1. Clinical photograph representing the ulnar sag present in midcarpal instability (**A**). Ulnar volar sag corrects with dorsally applied force on the pisiform (**B**).

Based on our findings in these and subsequent patients, we described a midcarpal shift test, which we found diagnostic for midcarpal instability [5-8]. The test is performed with the forearm stabilized in pronation and the wrist in neutral (Figure 2A). A palmarly directed force is applied to the distal capitate, and then the wrist is axially loaded and ulnarly deviated (Figure 2B). A painful clunk that reproduces

the patient's symptoms is a positive test for MCI.

After encountering our first 2 patients, we began to recognize the clinical pattern of MCI more frequently. These initial encounters encouraged us to better define the pathoanatomy and pathomechanics of the process. With this in mind, we performed selective ligament sectioning on multiple fresh wrist specimens [4]. The goal was to identify the pathology and then to develop a way to repair or reinforce the compromised structure(s). Although the volar arcuate ligament was our initial focus, we eventually realized that the dorsal radiotriquetral ligament (DRT) had the greatest effect in stabilizing the proximal row. In actuality, however, we were never able to reproduce the in vivo clinical findings of MCI in the laboratory to our complete satisfaction.
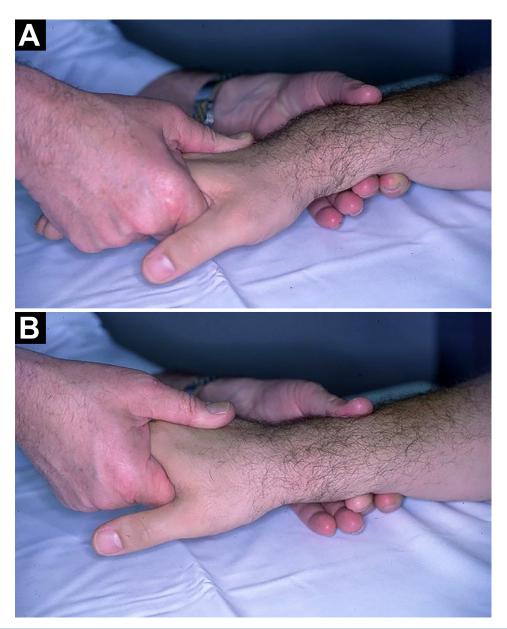

Figure 2. The midcarpal shift test. A force is applied from a dorsal direction with the wrist pronated. An axial load is applied (**A**). As the wrist moves into ulnar deviation, a painful clunk is identified in a positive midcarpal shift test (**B**). A positive midcarpal shift test is diagnostic of midcarpal instability.

Although we did not define the exact pathologic anatomy, our study of MCI caused us to reassess the standard theories of wrist kinematics. Because of inconsistencies in the longitudinal "columnar theory" of carpal instabilities, we began to visualize the wrist as a dynamic ring ("ring theory"), with 2 transverse rows rather than 3 rigid longitudinal columns of bones (Figure 3). In the ring theory, the proximal carpal row serves as a transverse intercalary link permitting reciprocal motion between the 2 carpal rows in response to external dynamic forces. Bones within the 2 rows are stabilized by their unique anatomy and a series of short intercarpal ligaments. In the older columnar theory, the rigid longitudinal connections were thought to provide stability, but this arrangement could not possibly permit controlled, physiologic motion between the 2 carpal rows.

The key to understanding the ring theory of carpal kinematics is the fact that the loading forces on the wrist created by routine activities of daily living, combined with the unique anatomy of the carpus itself, create a constantly changing torque
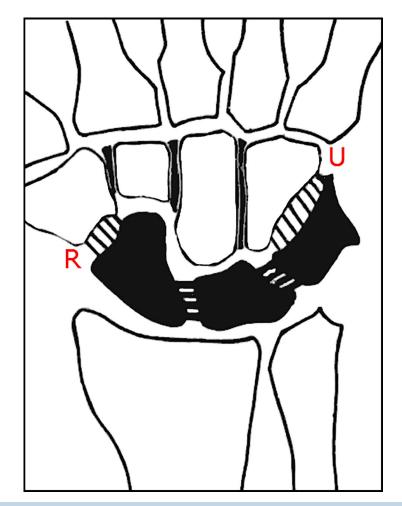

Figure 3. The ring theory of carpal dynamics. The "R" and "U" represent the radial and ulnar links between the proximal and distal carpal rows, respectively. In a wrist at neutral, a flexion moment is present at the radial link, and an extension moment is at the ulnar link. This results in a balance of forces (no VISI or DISI) on the mobile proximal row, and a stable wrist. A break in the "balanced ring" results in an instability pattern, with rotation of carpal bones based on the location of the disruption.

force on the mobile proximal row. The force moment is flexion on the radial side created by compression on the distal scaphoid at the scaphotrapeziotrapezoid (STT) joint, and extension on the ulnar side created by compression on the helicoid slope of the triquterum at the triquetrohamate articulation. Excessive proximal row extension is resisted by the shape and anatomy of the bones themselves, but excessive proximal row flexion is resisted by the series of intact ligaments that interconnect the proximal row. With radial deviation, the flexion forces predominate at the STT joint and the entire proximal carpal row follows into physiologic flexion; with ulnar deviation, extension forces predominate at the TH joint, and the entire proximal row follows into extension. In neutral, the moments are balanced and the proximal row is stable and immobile.

By visualizing the wrist according to the ring theory, one can visualize much more easily how various instability patterns arise. With disruptions of the LT ligament, the lunate follows the still-linked scaphoid into flexion (VISI) as the previously balanced forces are released across the proximal row. Likewise, with disruption at the SL joint, the lunate rotates into DISI as it is pulled by the triquetrum into extension. In MCI, there is insufficient restraint of rotation across the entire proximal row, so the midcarpal joint sags into gravity-induced flexion (VISI). With ulnar deviation the extension forces are reengaged at the TH joint and the entire proximal row snaps into sudden extension with an audible clunk.

While the ring theory made it possible to understand the pathomechanics of carpal instabilities, we were still unsure of the exact pathologic anatomy of MCI. In the early 1990s, however, a soldier who had previously undergone a successful intercarpal fusion for midcarpal instability in his right wrist presented again with a painful clunk, this time in his left wrist. He requested a midcarpal fusion for the second wrist as well. Because our cadaver studies had shown that selective ablation of the DRT was one cause of proximal row hypermobility, he consented to let us temporarily clamp and shorten this ligament as a test prior to the fusion. Once the DRT was clamped, the proximal carpal row was stabilized in neutral rotation (no VISI) and we could no longer recreate the audible clunk by performing the midcarpal shift test. The clunk and proximal carpal row hypermobility (with VISI) returned immediately upon releasing the clamp. This in-vivo experiment confirmed the importance of DRT laxity as a major etiologic factor in midcarpal instability and as a potential focus of surgical reconstruction. Since then, we have successfully utilized DRC ligament reefing for treatment of MCI in a limited number of cases.

Understanding the role of the DRT ligament in stabilizing the proximal row makes it possible to visualize the pathomechanics of MCI in the same light as the other common carpal instabilities. The DRT ligament, along with the entire dorsal ligament-bone complex acts as a single, spiral stabilizer of the proximal carpal row starting proximally at the radius. It then runs ulnarly and distally through the DRT ligament, then changes course to run radially and distally through the triquetrum, the TL ligament, the lunate, the SL ligament, and finally the scaphoid where it is anchored distally by the palmar STT ligaments. Traumatic discontinuity to any of the ligamentous or boney components at any upstream (proximal) point will result in a characteristic flexion deformity (VISI) of all downstream components due

to the ever present flexion moment at the STT joint. As a consequence, when the SL ligament is torn (SL instability), the scaphoid flexes alone; when the LT ligament is torn (LT instability), the lunate and scaphoid flex together; and when the DRT ligament is torn or incompetent (MCI), the entire proximal row flexes as a unit. In most instances, the distal flexion is accompanied by a reciprocal extension deformity.

The ring theory was the first important secret that was unlocked while studying the pathomechanics of MCI. It was instrumental in visualizing normal carpal mechanics as well as the pathomechanics of the other common instabilities. The second secret was the concept of a dorsal spiral ligament complex, which helped our understanding of the pathomechanics of MCI as well as establishing a conceptual bond between all the commonly named carpal instabilities. When the third secret came to light more, it revealed important implications for the future treatment of carpal instabilities. This has to do with the role of proprioception in counteracting wrist instability, secondary to injury or inherent ligament insufficiency.

Ever since we built our first splint to stabilize the wrist by pushing dorsally on the pisiform (thereby rotating the proximal carpal row out of flexion), we noticed that many patients could actively maintain that position through voluntary contraction of selected forearm and hypothenar muscles. More recently, the wrist ligaments have been shown to contain a rich network of proprioceptive fibers [9-11] similar to those of the shoulder, knee, ankle, etc. [12]. Proprioceptive retraining has been successful in many instances in allowing patients with major joint injuries to self-stabilize these joints and return to moderately stressful activities. This principle has also been applied

to stabilization of the scapholunate joint with mixed results [13, 14]. MCI seems to be the best example of how this principle can be applied to the wrist.

In clinical practice, in association with our hand therapists, we teach MCI patients to "set" the wrist prior to active ulnar deviation by isometrically contracting their periarticular wrist muscles. This reduces and stabilizes the proximal row in neutral, thereby eliminating the possibility of a catch-up reduction clunk in ulnar deviation. Through repetition, this active contraction becomes ingrained and patients will do it without conscious recognition. We have all witnessed proprioceptive retraining work following shoulder and knee injuries. It is exciting to think that carpal instabilities have the potential to be treated in the same way.

In conclusion, the secrets unlocked by studying MCI have enabled us to better visualize the normal kinematics of the wrist, which has led to a greater understanding of the pathomechanics of all carpal instabilities. More specifically, it has led to a stepwise, patient-specific treatment protocol for MCI (Table 1) [15]. For all initial encounters, and for patients with mild symptoms, activity modification, NSAIDS, and splinting are indicated. The next step is to train patients to "set" the wrist prior to active ulnar deviation as outlined above. In our experience, the majority of patients, especially teenagers and those with mild symptoms, will respond favorably to nonoperative measures.

For patients who have failed conservative treatment, we recommend reefing of the dorsal intercarpal ligament. Thus far, the procedure has successfully eliminated the clunk, with minimal loss of wrist flexion, in most instances. Midcarpal arthrodesis was once our first line of surgical treatment, but the ring theory taught us

Table 1. Treatment Recommendations for Midcarpal Instability.

Nonoperative Management

NSAIDs, Customized splint	Age less than 18; adults with mild instability
Proprioceptive training	Age less than 18; adults with mild instability; failed splinting and NSAIDs

Soft-Tissue Reconstruction

Dorsal capsular reefing (DRT)	Mild to moderate instability; failed nonoperative treatment
Thermal capsulorraphy (experimental)	Mild to moderate instability; failed nonoperative treatment

Osseous Procedures

Triquetro-hamate-capitate-lunate (4-Corner) fusion	Moderate to severe instability; failed nonsurgical and soft-tissue procedures
RSL or RL fusion (experimental)	Moderate to severe instability; failed nonsurgical and soft-tissue procedures

that midcarpal function is essential for well controlled motion between the 2 carpal rows. Four-corner fusion is therefore reserved for highly symptomatic patients, or for those who have failed prior surgical treatment.

Current and future research into finite vector analysis and four-dimensional computed tomography will inevitably provide more information about the pathologic anatomy of midcarpal instability. This information should allow for the development of even more precise recommendations for ligament reinforcement or reconstruction. Likewise, additional study is needed on the proprioceptive properties of carpal ligaments, the identification of muscles that act as secondary stabilizers of the wrist, and the retraining of these secondary stabilizers to compensate for loss of the inherent

stabilizing mechanism.

Research into one area often sheds light on related topics and provides unanticipated solutions to others. In this sense, our experience investigating the pathomechanics of MCI has been rewarding but is certainly not unique [16]. It is these unexpected discoveries that make the study of carpal instabilities especially rewarding and we hope will stimulate future investigations into the mysteries that remain.

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