Median nerve compression is a well-known cause of carpal tunnel syndrome (CTS). Yet, reasons why the most common idiopathic form of CTS develops in certain individuals are not well understood. To further understand the compressive mechanisms at work in CTS development, the authors used ultrasonographic imaging of the median nerve to evaluate 2 patients with CTS. Findings were compared to those of 2 control subjects who did not have CTS. In the patients who had CTS, the transverse carpal ligament was pulled taut by thenar muscle contraction as the flexor tendons tightened, compressing the median nerve between the ligament and tendons. No such compression was observed with the control subjects. Thus, a pathologic mechanism of median nerve compression was confirmed in the patients with CTS. Demonstration of such pathologic mechanisms during prehensile hand movement may improve understanding of how to treat patients with CTS and prevent nerve injury.

Carpal tunnel syndrome (CTS) is generally considered to be caused by median nerve compression at the wrist.\(^1\) The carpal tunnel is a narrow and relatively unyielding space that readily entraps the median nerve between the tough fibrous transverse carpal ligament ventrally and carpal bones dorsally. Because the median nerve is the softest structure within the carpal tunnel, it usually sustains injury first. Repetitive finger activity is believed to contribute to CTS—either because pressure from the flexor tendons irritates the adjacent median nerve or because inflammatory swelling of the tendon sheaths increases compartment pressure within the carpal tunnel.\(^1,2\)

Previous studies have measured intracarpal pressure in patients with CTS and in subjects without CTS, demonstrating higher pressures in those with CTS.\(^1,2\) However, no known previous studies have directly imaged the median nerve during activity to identify direct mechanical compression. High-resolution diagnostic ultrasonography is now available to readily observe the response to hand maneuvers that might challenge the median nerve.

The present case report demonstrates how the median nerve responds to prehensile hand activity in patients with CTS and in individuals without CTS, providing improved understanding of the pathologic mechanisms responsible for nerve compression.

**Reports of Cases**

**Carpal Tunnel Syndrome Case 1**

The patient in CTS case 1 was a man aged 24 years with a several-week history of pain, numbness, tingling, and weakness in his right hand, primarily involving the first three digits. Physical examination revealed positive results in Tinel’s test and Phalen’s test over the right carpal tunnel, as well as palpatory restriction over the right carpal tunnel.

Electrodiagnostic (EDX) studies confirmed median neuropathy at the right wrist. The median distal motor latency was 6.0 milliseconds, and the median distal sensory latency to the index finger was 5.3 milliseconds. Results of comparative radial and ulnar studies were normal, with ulnar distal motor latency at 3.0 milliseconds and ulnar distal sensory latency at 3.7 milliseconds and with all response amplitudes normal. Motor distances were 8 cm, and sensory distances were 14 cm. Needle electromyographic examination yielded normal results except for increased membrane irritability within the right thenar muscles—consistent with mild denervation. These findings were compatible with a diagnosis of moderate-to-severe CTS.

Immediately after EDX, the median nerve in the patient’s right wrist was imaged transversely with high-resolution ultrasound (13 MHz M-Turbo system; SonoSite Inc, Bothell, Washington) to measure the cross-sectional area in the proximal carpal tunnel at the level of the pisiform (Figure 1). Images revealed median nerve enlargement, with a cross-sectional measurement of 14 mm\(^2\) (compared with the normal upper limit of 11 mm\(^2\)).\(^3,4\)

The median nerve was then imaged longitudinally and
transversely at the mid-distal tunnel in a neutral relaxed position. Imaging was repeated during dynamic stress testing (DST), which involves a sustained isometric contraction of the thumb tip against the tips of the second and third digits using a hard rubber ball for resistance (Figure 2). Similar findings were noted in the transverse plane, with median nerve elongation and flattening during DST (Figure 4B-4C).

These findings indicated that as the thenar muscle mass contracted and tightened the transverse carpal ligament, a portion of the muscle bulged dorsally into the carpal tunnel (Figure 4B-4C). Simultaneously, the contracting flexor muscles tightened the flexor tendons ventrally, adding to the compressive effect by creating a more rigid and unyielding tunnel floor—essentially “sandwiching” the median nerve between the transverse carpal ligament and the flexor tendons.

**Carpal Tunnel Syndrome Case 2**

The patient in CTS case 2 was a woman aged 56 years with a 4-month history of numbness and tingling in her right upper extremity. Physical examination was unremarkable except for revealing positive results in Tinel’s test and Phalen’s test over the right carpal tunnel, as well as palpatory restriction over the right carpal tunnel. Electrodiagnostic studies confirmed median neuropathy at the right wrist. The median distal motor latency was 4.0 milliseconds (with 2.7 milliseconds to the ulnar distal motor latency), and the median distal sensory latency to the thumb, at 10 cm, was 3.2 milliseconds. The radial distal sensory latency to the thumb was 2.5 milliseconds (at 10 cm), and all response amplitudes were normal. Results of the needle electromyographic examination were normal.

As with the patient in case 1, ultrasonographic imaging was obtained with the patient in case 2 after EDX. This imaging revealed an enlarged right median nerve, with a cross-sectional measurement of 16 mm², at the level of the pisiform. Longitudinal imaging (Figure 5) revealed right median nerve compression during DST. This compression was documented with prestress and stress diameter measurements, showing that the median nerve diameter decreased from a prestress measurement of 0.26 cm to a measurement of 0.19 cm during stress—representing a 27% compressive narrowing.
nerve size of 10 mm² at the pisiform. Imaging during DST (Figure 6C-6D) revealed no nerve compression, with the right median nerve diameter increasing from a prestress measurement of 0.21 cm to a stress measurement of 0.23 cm—representing a 9.5% increase.

Discussion
Static ultrasonographic imaging to measure median nerve enlargement at the level of the pisiform has been described as a pathologic finding in CTS. Other findings, such as median nerve flattening or “notching” in the distal carpal tunnel, have also been observed in CTS—though less consistently than proximal nerve enlargement.

Some previous studies have used ultrasonographic imaging to identify a decrease in nerve “sliding” within the carpal tunnel during passive index finger motion. For example, Nakamichi and Tachibana observed that in normal control subjects, the median nerve slides transversely to a position in the carpal tunnel that is “freer” (ie, has reduced pressure), but in subjects with CTS, the median nerve has restricted motion (ie, decreased sliding) and increased exposure to compression. In another study, active contraction with fingertip loading was used to demonstrate an increase in pressure within the carpal tunnel during index finger pinch gripping. However, none of these previous studies challenged or imaged the median nerve directly during active muscle contraction and tendon tightening to observe nerve compression dynamically within the carpal tunnel in patients with CTS.

Prehensile hand movement requires fixation of the primary thumb movers (ie, abductor pollicis brevis and opponens pollicis) at their base of attachment, where they anchor to the transverse carpal ligament. Such fixation allows muscle contraction to pull the thumb toward the other digits for controlled grasping functions. A solid immobile base of attachment prepares the muscles to freely move the thumb.

When more powerful and sustained grasping or pinching...
functions are required, such as firmly holding a tool or pencil (the most common form of prehension), the thumb tip becomes immobile and the anchor becomes the mobile segment. As a result, thenar muscle contraction pulls the transverse carpal ligament taut—instead of moving the thumb—and the muscle bulges dorsally into the carpal tunnel. At the same time, the flexor tendons to the thumb and digits are pulled taut, and the tendons “bowstring” toward the underside of the transverse carpal ligament, “sandwiching” the median nerve between the tendons and transverse carpal ligament (Figure 4B-4C). Ultrasonographic images also demonstrate apparent flattening or compression of the median nerve during DST maneuvers, as seen in Figure 3A, Figure 4A, and Figure 5A-5B.

A classic “squeeze play” appears to be at work in this mechanism, with the roof (ie, transverse carpal ligament) of the carpal tunnel tightening and lowering and the floor (ie, flexor tendons) of the carpal tunnel tightening and rising—thereby compressing the median nerve.

In dynamic studies using digital video recording of several patients with CTS, the author demonstrated that the median nerve is actively compressed during pinch activity. This compression is particularly obvious with transverse ultrasonographic imaging in the mid-tunnel region. How-
treatment of patients as well as potential prevention of median nerve injury. Reasons why CTS develops in some individuals who perform repetitive or vigorous hand activities but not in others are not clearly understood. Some researchers have observed contractile cells in the transverse carpal ligaments of patients with CTS, suggesting that the ligaments in these patients were in a constant state of contraction.11 This author previously observed relative mechanical restriction over the carpal tunnel in patients with CTS, as measured by quantitative palpation—a finding that could correlate with tightness in the transverse carpal ligament.8 It is unknown if these patients had preexisting abnormalities that contributed to the development of CTS or if the specific patterns of movement in these patients created the abnormalities that subsequently led to median nerve injury.

However, repetitive or sustained contractions of the thenar muscles, combined with possible contraction of myofibroblasts within the transverse carpal ligament, causes relatively increased tightness in the transverse carpal ligament, leading to further foreshortening and pressure on the median nerve. In addition, perpetual contraction of the thenar muscles contributes to their hypertrophy, leading these muscles to protrude into the carpal tunnel during activity.8,11

These observations suggest a multifactorial causation in CTS, including increased intracarpal pressure2,5; decreased median nerve mobility (from fibrous fixation)5,10; median nerve deformation (ie, compression, stretching, traction)9; increased stiffness of the synovium and flexor retinaculum (ie, transverse carpal ligament)5; relative thenar muscle hypertrophy or increased thenar muscle mass with intrusion into the carpal tunnel; and flexor tendon thickening and tightening during activity. The latter two processes would substantially contribute to compression by tightening and lowering the transverse carpal ligament at the same time that the floor (ie, flexor tendons) is tightened and raised during prehensile activity (ie, thenar flexion and opposition to the first two digits).
These effects of OMT could prepare a normal carpal tunnel for improved activity tolerance, thereby making OMT a valuable component of a program of CTS prevention.

Conclusion

Diagnostic ultrasonographic imaging of the carpal tunnel adds a new dimension to understanding the pathologic mechanisms involved in the development of CTS. It is now possible to directly image median nerve compression during prehensile hand activity—heretofore unconfirmed as a contributory cause of nerve injury. In addition, observation of thenar muscle intrusion into the carpal tunnel indicates that this intrusion may be a factor previously unsuspected in CTS causation.

The present case study using high-resolution diagnostic ultrasonography suggests that the mechanism of carpal tunnel release with OMT impacts several of the suspected factors causing CTS. Stretching of the transverse carpal ligament reduces tension in that structure and leads to increased space within the carpal tunnel, decreasing pressure on the median nerve. At the same time, release of thenar muscle tightness leads to decreased muscle intrusion into the carpal tunnel. Elongation of the flexor tendons should decrease thickening and tightening on the other side of the carpal tunnel and also decrease pressure on the median nerve.
stretching approaches to CTS management. Furthermore, the new findings indicate that application of ultrasonography during DST can open a window to prevention of CTS.

Acknowledgments
I give special thanks to Christine Quinn, RDMS, and Lois Ferguson, RT, RDMS, RVT, of Sonosite Inc in Bothell, Washington, for generous use of the M-Turbo system to acquire the ultrasonographic images for CTS case 1 of this report.

References

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**Figure 6 (continued).** Ultrasonographic images of the right wrists of two control subjects without carpal tunnel syndrome. Longitudinal images of the first control subject’s median nerve at prestress (A) and during stress (B) depict nerve diameter measurements between the “A” markers. These measurements show the initial nerve diameter of 0.24 cm increasing to 0.25 cm during stress—revealing a lack of compression. Longitudinal images of the second control subject’s median nerve at prestress (C) and during stress (D) also depict nerve diameter measurements between the “A” markers. These measurements show the initial nerve diameter of 0.21 cm increasing to 0.23 cm during stress—revealing a lack of compression.

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Editor’s Note: A video clip of the live dynamic stress test described in the present study has been posted online to the JAOA’s Web site at http://www.jaoa.org/cgi/content/full/109/12/641.