Complex Foot Deformities: A Case Study

Complex structural deformities of the foot and ankle, especially those caused by neurological disorders or trauma, present some of the most time-intensive and technically challenging cases for orthopedic surgeons and involve some of the most difficult recoveries for patients. Many of these deformities result from a combination of muscular, anatomical and neurological dysfunction.

For example, in the hereditary sensorimotor peripheral neuropathy Charcot-Marie-Touche (CMT) disease, some muscles become weaker than others. This leads to an imbalance of forces across the foot and ankle, which in turn leads to deformity. Over time, the deformity progresses causing pain, difficulty wearing shoes, and sometimes skin breakdown, nonhealing ulcers and infections.

The goal in treating deformity in the foot and ankle, including CMT, is to prevent complications and achieve a plantigrade, stable foot that allows reasonable function. This can be accomplished surgically by realigning the foot using osteotomies or fusions and balancing the forces across the foot by way of soft tissue releases or tendon transfers.

A common approach for a symptomatic, relatively moderate deformity in CMT, for example, is first to osteotomize the calcaneus and first metatarsal to bring about a more plantigrade foot. In a more severe deformity, the shape of the foot is corrected through a hindfoot fusion or triple arthrodesis. The foot is then balanced using soft tissue releases and tendon transfers, such as an Achilles tendon lengthening, plantar fascia release, peroneus longus to peroneus brevis tendon transfer and transfer of the tibialis posterior tendon to the dorsal foot. In this way, the foot is made plantigrade with osteotomies or fusions, and it is balanced through the soft tissues.

“Processes such as Charcot-Marie-Tooth have classic presentations — a cavus, high-arched foot, clawed toes and ankle laxity,” says Daniel B. Ryssman, M.D., an orthopedic surgeon at Mayo Clinic’s campus in Rochester, Minnesota. “But ones are unique. It’s important to examine patients thoroughly to see what is working and what isn’t. Surgeons must rely on X-rays, a careful physical exam and sometimes nerve conduction studies to determine what is causing the deformity. Then they can start planning what to do.”

Dr. Ryssman says treatment is dictated by the location and type of deformity, disease severity, and the presence of infection and other complications. Complex structural deformities of the foot and ankle, especially those caused by neurological disorders or trauma, present some of the most time-intensive and technically challenging cases for orthopedic surgeons and involve some of the most difficult recoveries for patients. Many of these deformities result from a combination of muscular, anatomical and neurological dysfunction.

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comorbidities as well as by patient goals and degree of social support.

“Some patients simply aren’t good candidates for surgery because of comorbidities, or the deformity may just be too great to fix. I can usually straighten the foot, but if the patient still can’t use it because of skin loss or a recurring infection, surgery isn’t in his or her best interest. In such cases, the only real options are to leave the foot alone or perform an amputation,” he explains.

“But other considerations are critical, too. What is the patient’s chief complaint, and what is the social situation at home? Is there support? You have to really listen to the patient before deciding what to do.”

In straightforward cases, the deformity can be corrected in a single stage. Patients remain in a cast for 10 weeks and then transition to a boot. A month or so later, they begin wearing shoes, with full recovery expected within six months to a year.

Other deformities are not suitable for acute correction and internal fixation, have a significantly increased risk of complications or poor soft tissue coverage, or are multiplanar or rigid. These may require a staged surgery involving percutaneous osteotomy, significant releases and external fixation with a device such as the Taylor spatial frame.

The frame, which consists of two or more rings connected to six telescopic struts that can be lengthened or shortened, allows gradual postoperative correction of the deformity by changing strut lengths according to a computer-generated treatment plan.

“By making 1-millimeter adjustments to the struts every day, the rings are slowly repositioned and the deformity is gradually corrected at a pace that allows the skin and soft tissue to heal,” Dr. Ryssman says. “This can take up to six months, and although it is challenging for patients, the technique has helped correct very severe deformities.”

**A case study**

One such case involved a Somali man in his early 20s, who first saw Dr. Ryssman in April 2012. A gunshot to the young man’s spine had caused neurological damage leading to severe bilateral equinocavovarus foot deformity (Figures 1-3).

“This young patient was ambulating on the tops of his feet and would have to swing his legs through his crutches,” Dr. Ryssman says. “The deformity was fixed and rigid, and I initially thought there was no way to treat him, but then decided we might be able to work with the frame. I was concerned, however, because he didn’t have family here, although an aunt was willing to help. Eventually, after three or four meetings, I decided to perform the surgery on one foot, which turned out to be extremely complex. The normal boney architecture was so twisted it was difficult to apply the fixator.”

Adjusting to the fixator was a challenge for the patient, too, who fainted the first time he saw it, Dr. Ryssman says. He gradually grew used to it, however, and by the time he left the hospital was proficient at making the strut adjustments.

A year later, Dr. Ryssman surgically corrected the other foot, again using the external fixator. Today, both feet are plantigrade, and the young man is able to walk in braces without pain (Figures 4-5 and Figures 6-8).

“His feet are not normal, but there is a significant difference, and we achieved our goal,” Dr. Ryssman says. “Proceeding with the correction was a very difficult decision. The patient was essentially living alone and his English was imperfect. I wondered whether I was doing the right thing for him given his social situation and whether this might be too much for him. But it ended up making a tremendous difference in his life. These cases are all multifactorial; they require a great deal of planning and a long recovery for patients, but at the one-year mark, when they are recovered and walking straight, it’s very satisfying and sometimes quite emotional to see patients and their families so happy.”

**Figures 4-5. First foot corrected.**

**Figures 6-8. In images of the final correction, both of the patient’s feet are plantigrade.**
Talipes equinovarus (clubfoot) is a congenital deformity in which the foot is inclined inward, axially rotated outward and downward pointing. It is one of the most common problems seen in pediatric orthopedics, with incidence rates ranging from 1 in 1,000 live births in the U.S. to about 7 in 1,000 live births among Polynesian Islanders and the Maoris in New Zealand. Males are affected twice as often as females, and the incidence of bilateral cases is 50 percent.

A small percentage of cases are associated with a generalized syndrome such as amniotic band syndrome or with neuromuscular disorders, including spina bifida and arthrogryposis, but most are idiopathic. Some studies have suggested that variations in the protein-encoding gene PITX1 or its downstream transcriptional targets may increase susceptibility to clubfoot, but the precise etiology has not yet been identified.

Management then and now

Clubfoot treatment is among the earliest orthopedic therapies; Hippocrates described a method of gentle manipulation and bandaging that is remarkably similar to the approach Ignacio Ponseti, M.D., developed in the 1940s and published in 1963. Ponseti’s method, which involves serial casting and percutaneous Achilles tenotomy of the affected foot followed by bracing to maintain the correction, is now the de facto gold standard for clubfoot treatment worldwide.

It was ignored for nearly 40 years, however, when the standard of care was aggressive surgical correction involving tendon lengthening and release of capsular and ligamentous structures. Release procedures, although successful in the short term, frequently demonstrated overcorrection, pain and stiff scar healing on follow-up, and children often required further surgery to address these and other complications.

Due to these disappointing and often life-altering results and increasing pressure from parents, Ponseti’s noninvasive method gained momentum at the turn of the millennium. Since then, hundreds of comparative studies have shown its superiority to surgery with regard to primary correction rate, functional outcome and recurrence rate in both idiopathic and nonidiopathic clubfoot. If correctly done, the Ponseti method is successful in greater than 85 percent of cases.

The Ponseti method

Clubfoot treatment should begin in the first weeks of life to have the best chance for a successful outcome. Typical clubfoot cases usually require weekly manipulation and casting of the foot for six weeks; complex or syndromic cases may require more treatment. About 80 percent of infants require an Achilles tenotomy toward the end of casting. To maintain the correction, children generally wear a foot abduction brace 23 hours a day for three months, followed by part-time bracing at night and naptime until age 3.

“There is no doubt that the method is long, arduous and very involved,” says Todd A. Milbrandt, M.D., a pediatric orthopedic surgeon at Mayo Clinic’s campus in Rochester, Minnesota, who studied with Dr. Ponseti. “Nevertheless, most people now feel it is worth it to get better results.”

Recurrences also are an issue. “With this treatment you do get recurrences,” Dr. Milbrandt says. “The biggest reason is that parents become tired of the routine, so children stop wearing the brace as often as needed and the clubfoot comes back. If that occurs, the next step is to recast to correct the deformity, perform the Achilles tenotomy again, and then put the child back into shoes.”

About 30 to 40 percent of children successfully treated with the Ponseti method will need a tibialis anterior tendon transfer to correct residual muscle imbalance from a strong tibialis anterior muscle and weak antagonists. It’s unclear why the tibialis anterior muscle maintains relatively normal function and strength in clubfoot deformities, but the imbalance produces dynamic supination when a child walks.

One method of correction is the anterior tibialis tendon transfer, which involves transferring the entire tendon to the third cuneiform. The child then wears a non-weight-bearing cast for approximately six weeks. Improved muscle balance is usually apparent as soon as the cast is removed, and recurrences are very rare, Dr. Milbrandt says.

**Figure.** Pedobarograph showing rocking on toes.
Scapholunate Injuries: A Paradigm Shift?

The scapholunate is an interosseous carpal ligament that provides stability to the proximal carpal row. It consists of dorsal, proximal and palmar segments that bridge the scaphoid and lunate. When the ligament complex tears, the scaphoid tends to flex while the lunate extends, creating translational and rotational instability between the two (Figure 1). Instability is generally defined as the inability to bear physiological loads with an associated loss of normal wrist alignment.

Injuries to the scapholunate, along with the resulting instability, are among the most common and significant ligament injuries. They often occur during sports when an athlete falls on an outstretched hand, for example, and when not properly diagnosed and treated, can lead to functional disabilities and progressive degenerative osteoarthritis. Although arthritis can occur even after surgical repair or reconstruction, early diagnosis and appropriate treatment may restore normal function and prevent further damage to the joint.

Mayo Clinic orthopedic surgeons James H. Dobyns, M.D., and Ronald L. Linscheid, M.D., were among the first to conceptualize and describe traumatic carpal instability in 1972. They introduced the defining terms dorsal intercalated carpal instability and volar intercalated instability and described other problems related to abnormal wrist motion.

Since then, carpal instability has continued to vex hand surgeons. Diagnosis can be difficult in early injuries, and the diagnostic gold standard remains wrist arthroscopy.

In the last few years, however, advances in the understanding of wrist biomechanics and kinematics have led to improvements in the early, noninvasive detection of abnormal joint motion. Through the collaborative efforts of Mayo Clinic radiologists, biomedical engineers, basic science specialists and hand surgeons, the role of 4-D CT imaging has shown promise for the noninvasive
diagnosis of dynamic carpal instability, which can only be detected during joint motion (Figure 2).

“This state-of-the-art CT technology provides information about carpal motion in real time at velocities similar to those of activities of daily living,” explains Sanjeev (Sanj) Kakar, M.D., an orthopedic surgeon at Mayo Clinic’s campus in Rochester, Minnesota. “Three-dimensional movies of the patient’s wrist can be played back, allowing physicians to see and measure discrepancies in joint motion and altered carpal loading from these subtle ligament injuries.”

Not only does 4-D CT technology allow earlier and better assessment of dynamic joint instabilities, it also helps locate the exact site of ligament injury (Figure 3).

“The team at Mayo is currently studying patients with scapholunate instability using this technology with the goal of precisely identifying which part of the ligament is injured and directing treatment to that area as opposed to a shotgun approach where one treatment suits all,” Dr. Kakar says.

**Building a better ligament**

Management of scapholunate instability is both complex and controversial. Therapeutic options range from splinting and casting or arthroscopic treatment to ligament reconstruction using autograft tendons. Nearly 40 years after the seminal work of Drs. Dobyns and Linscheid, the results of surgery remain unpredictable.

Reconstruction presents special challenges for hand surgeons, and a number of techniques have been proposed to overcome them. But the problem of poor tissue regeneration continues to be a vexing one. So Mayo Clinic researchers, including Dr. Kakar, Andre J. van Wijnen, Ph.D., and Michael J. Yaszemski, M.D., Ph.D., are experimenting with the use of scaffolds seeded with progenitor cells and growth factors to regenerate ligamentous tissue.

In a study published in the November 2015 issue of Tissue Engineering Part A, they describe
Arthrofibrosis is a recognized complication of total knee arthroplasty (TKA) and one of the leading reasons for re-operations and revision surgery. Often defined as flexion less than 90 degrees postoperatively (Figure 1), it is characterized by abnormal scarring leading to restriction of the joint. The scar tissue may form intra-articularly and extra-articularly, and may persist in a subset of patients despite manipulation under anesthesia (Figure 2).

Clinically, patients present with limited knee range of motion. Some may have pain with activities of daily living, such as walking, standing or climbing stairs; others may have unusual postoperative pain and swelling unrelated to deep periprosthetic infection or mechanical failure. In severe cases, the arthrofibrosis may become progressive, with fibrous tissue thickening and tightening the entire capsule, making joint movement nearly impossible.

Mayo Clinic research led by Matthew P. Abdel, M.D., an orthopedic surgeon at Mayo Clinic’s campus in Rochester, Minnesota, has shown that despite advances in surgical technique, implant design and pain management, the incidence of arthrofibrosis following primary TKA has remained virtually unchanged for the past two decades. A review of nearly 10,000 patients who underwent cemented posterior-stabilized TKAs at Mayo Clinic’s campus in Minnesota between 2000 and 2012 revealed that 5.8 percent developed limited range of motion, with 2.8 percent requiring manipulation under anesthesia (MUA).

These numbers were relatively unchanged from the previous decade, when the rate of limited motion was 5.4 percent, with 2.9 percent of patients requiring MUA. With contemporary MUA, mean flexion improved from 61 to 109 degrees, but a subset of 14 patients failed to maintain at least 90 degrees of flexion.

Dr. Abdel says there is limited information available to determine which patients will develop arthrofibrosis postoperatively. “Two patients who are kin can have the same surgery with the same surgeon, and one will develop arthrofibrosis, and the other won’t,” he notes.

Theoretically, the disease process could be prevented by interventions that affect the profibrotic cascade, but because the pathology of arthrofibrosis is not well-understood, finding pharmaceutical and biologic targets has been challenging. Myofibroblasts (α-smooth muscle actin-expressing fibroblasts) are one potential target, as shown by Dr. Abdel and his translational laboratory in a landmark 2012 article published in the Journal of Orthopaedic Research.

While other studies have shown increased myofibroblasts in human elbow contractures and in animal models of arthrofibrosis, the timeline of myofibroblast development wasn’t known until recently. That led a team of Mayo Clinic investigators to undertake a study examining the number of myofibroblasts in an animal model of knee contracture over a 24-week period.

The study involved 18 rabbits that under-
went a validated surgical procedure on their right limbs that causes a knee joint contracture. Both the animal model and modified procedure were created at Mayo.

The rabbits were then divided into three groups of six. Group I underwent two weeks of postoperative immobilization. Group II underwent eight weeks of immobilization, and Group III underwent eight weeks of immobilization plus 16 weeks of remobilization. The left collateral limbs of the rabbits served as controls, along with five additional rabbits that did not undergo surgery. The percent and number of total myofibroblasts and total cell counts were measured and compared with controls at two, eight and 24 weeks.

Results showed that the relative number of myofibroblasts was significantly elevated in the operated limbs compared with nonoperated limbs at two weeks after surgery. After peaking at two weeks, these numbers declined, with the relative number of myofibroblasts dropping from 19.9 plus or minus 3.1 percent at two weeks to 3.0 plus or minus 2.7 percent at eight weeks.

Indeed, at eight weeks of immobilization and 16 weeks of remobilization — a total of 24 weeks — there were no significant differences in the relative number of myofibroblasts between the operated and control limbs.

Dr. Abdel notes that although it was suspected that myofibroblasts were an early phenomenon, this was the first study to characterize them during the formation of a joint contracture. This further validates the idea that pathology starts early in arthrofibrosis and that interventions to inhibit fibrosis should likely start early in the disease process, too.

A search for such interventions is ongoing at Mayo Clinic. A 2014 study published in Bone & Joint Research found that intra-articular injections of the potential anti-fibrotic agent decorin caused significant alterations in several fibrotic genes, but did not alter the clinical contractures. Dr. Abdel, who was lead study author, says the timing of administration of decorin was likely responsible for the lack of improvement.

Another study looked at the results of surgical capsular release in an animal model of joint contracture. In that study, published in the Journal of Orthopaedic Research in 2013, capsular release decreased extension loss immediately after surgery as well as after 16 weeks of remobilization, echoing clinical experience in the surgical treatment of arthrofibrosis.

“We have published multiple papers on animal models of arthrofibrosis, trying to replicate the clinical scenario identically, in an effort to execute true translational research,” Dr. Abdel says. “We have taken this work from the bedside to the bench and then back to the bedside — that is, identifying a clinical problem such as fibrosis, taking it to the lab to test it in novel animal models and then taking it back to study in clinical trials.”

**For more information**


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