

The Importance of the Lumbricals

When was the last time you thought about the function of the plantar lumbricals? How about their role in the gait cycle?

When was the last time you actually evaluated them? Are they really that important? Read on and decide.

The lumbricals of the foot attach proximally to the sides of adjacent tendons of the flexor digitorum longus (with the exception of the first, which only attaches to the medial side), attach distally to the medial aspect of the head of the proximal phalynx and continue on to the extensor hoods in the second through the fifth toes.^{1,2} Their typical function is described as flexion of the proximal phalynx and extension of the proximal and distal interphalangeal joints. They have the unique ability to compress the metatarsal-phalangeal and interphalangeal joints.³ There is also a small adductory moment to counteract abductory shear, due to the tendon passing medial to the metatarsal-phalangeal joints.⁴ These are "open-chain" functions. Unless you are in the habit of waving to people with your toes, they often are used quite differently in the gait cycle with the foot affixed to the ground.

The lumbricals are most active from midstance to pre-swing.^{5,6} That means they act predominantly in the closed chain. The lumbricals, along with the other intrinsic muscles of the foot, play a role in maintaining the medial longitudinal arch of the foot.⁷ Along with the interossei, they play a role in stabilization of the forefoot during stance phase⁸ and rearfoot during pre-swing.⁹ Bahram Jam¹⁰ proposes that overpronation is due to a lack of neuromuscular control of the intrinsic foot muscles to stabilize the tarsal and metatarsal bones and modulate pronation speed.

Thinking from a distal to proximal orientation (a "closed-chain" mode of thinking), they actually plantarflex the metatarsal on the fixed phalynx, assist in dorsiflexion of the ankle and help to keep the toes from clawing from over-recruitment of the flexor digitorum longus. When was the last time you watched someone's toes while they were walking? How about casual observation during your exams?

Clawing toes during gait, which are considered abnormal, are defined as an extension of the metatarsophalangeal articulation and flexion of the proximal and distal interphalangeal joints.¹¹ They result from a foot attempting to stabilize itself during the terminal stance and pre-swing phases of gait. This is an attempt to help propel the body forward, often accompanied by overactivity of the flexor digitorum longus, tibialis posterior, flexor pollicis longus and gastrocsoleus groups.^{12,13} Overactivity of these groups causes reciprocal inhibition of the long toe extensors and ankle dorsiflexors (i.e., tibialis anterior), causing the toes to buckle further and a loss of ankle dorsiflexion; in short, diminished ankle rocker.¹⁴ (Please see "The Basics of Gait" at www.chiroweb.com/archives/25/23/14.html.)

Now think about the changes in the gait cycle in the above scenario. There will be a resultant

shortened step length, diminished ankle rocker, increased forefoot rocker and premature heel rise. This will necessitate an increased extension at the metatarsophalangeal joints, shifting the tendon of the lumbricals upward and behind the transverse metatarsal joint axis. This causes even more extension at this joint. Over time, this causes displacement of the fat pads anteriorly from under the metatarsal heads¹⁵ and is one of the main reasons for metatarsal head pain (metatarsalgia). In the past, have you made the apparent simple diagnosis of metatarsalgia, shin splints, stress fractures or Morton's neuroma without knowing a more plausible cause? Do you now feel you have better answers to these clinical phenomena?

Now think about changes up the kinetic chain and the potential musculoskeletal implications of muscle inhibition, overfacilitation and joint dysfunction, often with neurological sequelae. With lumbrical dysfunction (weakness) and lack of ankle dorsiflexion, you have less hip extension. So, you borrow some from the lumbar spine with increased compressive forces and an increase in lordosis, which causes an increase in thoracic kyphosis and cervical lordosis.

We still need to get this leg up and forward to continue our progression ahead, so we fire our hip flexors instead of the abdominal oblique muscles. Because there needs to be cooperation of the abdominals and hamstrings to maintain pelvis neutrality, this further fuels inhibition of the gluteals, thus compounding the loss of hip extension. Now how about a little increased shoulder flexion on the contralateral side to assist getting that leg forward. Don't forget that we have altered the thoracic kyphosis and thus changed scapulohumeral mechanics. Now we have neck/shoulder pain - all from bad feet? Maybe. These muscles developed and exist for a good reason. Do your best not to dismiss them and their function the next time you see a tortured foot.

When patients have continued dysfunction, consider the base and where it all begins. Consider function in the context of where it occurs. Proper evaluation of the feet and gait can provide valuable clues as to the etiology or manifestation of continued problems. Is this important? You decide.

References

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