Hand and Wrist in Athletes

Neurovascular injuries of the hand in athletes

David E. Ruchelsman^a and Steve K. Lee^b

ABSTRACT

Neurovascular syndromes at the wrist and hand of athletes remain relatively rare clinical entities. These represent unique pathologies that are most often secondary to the repetitive motion and stresses in these regions accrued during racquet sports or sports with recurrent impact to the hand and wrist. The anatomy, pathophysiology, diagnosis and treatment algorithms are reviewed for the more common syndromes that afflict this distinct, yet diverse, patient population. A wide spectrum of nerve and vascular disorders may affect the performance of an athlete. Because these are relatively rare conditions, a high index of suspicion is paramount to accurate diagnosis and timely referral and treatment.

Keywords

athlete, hand and wrist trauma, nerve syndromes, upper extremity, vascular injury

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INTRODUCTION

epetitive and forceful motion, loading and blunt impact to the wrist, palm and digits of athletes occur in a variety of sports directly adjacent to the regional neurovascular structures, which are relatively superficial and fixed within fibro-osseous conduits and fascial compartments. These factors account for the spectrum of nerve and vascular pathologies affecting the wrist and hand in this unique, yet diverse, patient population. Local compression of the median, ulnar and radial nerves, as well as, vascular insufficiency secondary to aneurysm formation, arterial thromboses, or vasospastic phenomena at the wrist or palm and digits may significantly affect an athlete's performance. Prompt diagnosis, referral and initiation of treatment are paramount to achieving a safe and expeditious return to sports. Nonoperative management, including activity and posture modification, as well as, protective padding and splints, may alleviate neuritic or ischemic symptoms. In severe or recalcitrant cases, nerve decompression and microvascular operative interventions are indicated.

NERVE SYNDROMES IN THE ATHLETIC HAND

Carpal Tunnel Syndrome

Median nerve compression at the wrist is the most common focal compression neuropathy in the upper extremity in the general population, as well as in athletes. Carpal tunnel syndrome (CTS) has been reported in a variety of athletic populations (body-builders, ³ cyclists, ⁴• hockey players, race-car drivers, ⁵ rock-climbers, ⁶ rowers, swimmers, ³ and wheelchair athletes⁷). The pathomechanics implicated include forceful, repetitive wrist dorsi-palmarflexion coupled with the unique prolonged wrist postures required in select activities.⁸ In the appropriate clinical setting, the evaluating hand surgeon should consider the abuse of growth-hormone and testosterone^{9,10} as potential etiologies in the presentation of CTS in athletes. Symptoms of median nerve compression at the wrist may present in athletes either insidiously or more acutely with each trial of effort. Additionally, high-energy wrist trauma sustained during athletic events (distal radial fracture, radiocarpal dislocation, greater or lesser arc perilunate injuries) carries the risk of associated acute CTS or median nerve contusion.

Median nerve anatomy at the distal forearm and within the carpal tunnel is well-described. ^{11,12} The median nerve enters the hand deep to the transverse carpal ligament between the flexor carpi radialis and flexor digitorum superficialis. The motor fibers of the median nerve innervate the superficial

Correspondence to Steve K. Lee, MD, NYU Hospital for Joint Diseases, 301 East 17th Street, Suite 413, New York, NY 10003

Tel: +212 598 6697; fax: +212 598 6560;

e-mail: steve.kichul.lee@gmail.com

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^aDivision of Hand Surgery, Department of Orthopaedic Surgery, New York University Hospital for Joint Diseases, New York, NY

^bAssistant Professor of Orthopaedic Surgery, New York University School of Medicine, and Associate Chief, Division of Hand Surgery, Department of Orthopaedic Surgery, New York University Hospital for Joint Diseases, New York, NY

head of the flexor pollicis brevis, abductor pollicis brevis, opponens pollicis, and the two radial lumbricals; its sensory fibers supply the radial three and one-half digits volarly.

Symptoms may include paresthesias, motor weakness, or a pure pain syndrome involving the distal forearm, wrist and hand. Classically, symptoms are intensified at night. Results of provocative maneuvers (Tinel's sign in all three zones of the carpal tunnel, the median nerve compression test, and Phalen's test) should be documented. The sensitivities of the pressure provocative and Phalen's test have been shown to be superior to the sensitivity of Tinel's sign. ¹³ The presence of thenar atrophy should be noted, and the strength of thumb palmar abduction should be graded. Two-point discrimination also is assessed. The presence of median nerve symptoms from compression within the carpal canal is distinguished from more proximal compression (pronator syndrome or anterior interosseous nerve compression syndrome) by the clinical history and thorough physical examination of proximally-innervated musculature (pronator teres and flexor pollicis longus) and the sensory territory of the palmar cutaneous nerve. While the diagnosis of CTS is established clinically, 14• recent clinical guidelines established by the American Academy of Orthopaedic Surgeons on the diagnosis of CTS¹⁵ include a Grade B recommendation based on level II and III evidence that electrodiagnostic testing should be performed if clinical or provocative tests are positive and operative management is being

Nonoperative management includes resting and nighttime neutral wrist splints, activity modification, and nonsteroidal anti-inflammatory medications. We do not use carpal tunnel corticosteroid injections in our treatment algorithm unless the athlete presents with acute symptoms immediately referable to repetitive wrist use during athletic activity. Carpal tunnel release is indicated in athletes with persistent symptoms despite an adequate trial of nonoperative treatment. We believe that it is essential to ensure release of the distal volar forearm fascia up to 4 cm proximal to the distal volar wrist crease. 16• Anomalous muscles, tendinous interconnections, or lumbricals with the carpal tunnel should be noted because they may be the source of compression.

The decision to use formal postoperative rehabilitation remains based on the surgeon's experience and individual patient factors. Factors influencing time to return to sports following decompression include the operative approach and athlete motivation. Cyclists and racquet sport athletes are encouraged to use padded gloves postoperatively to minimize pillar pain and recurrence.

Ulnar Nerve Compression at the Wrist

This neuropathy affects athletes involved in sports similar to those with CTS. The underlying pathomechanics include repetitive mechanical compression of the ulnovolar portion of the palm and the underlying ulnar nerve within Guyon's canal.

In the distal forearm, the ulnar nerve lies adjacent to the ulnar artery. At the wrist, the ulnar neurovascular bundle enters Guyon's canal, a well-defined fibro-osseous conduit bordered volarly by the volar carpal ligament and dorsally by

the transverse retinacular ligament (pisohamate ligament). The ulnar and radial borders are the pisiform and hamate, respectively. The nerve bifurcates at the distal extent of the canal into a superficial sensory branch, supplying the ulnar one and a half digits and palm, and a deep motor branch, supplying the intrinsic muscles of the hypothenar eminence, the palmar and dorsal interossei, the third and fourth lumbricals, the deep head of the flexor pollicis brevis, and the adductor pollicis. 17

The superficial location of the ulnar neurovascular bundle in the hand, fixed canal volume, and proximity of the hamate and pisiform, make the nerve and artery susceptible to cyclical mechanical compression, as well as blunt trauma. 18,19 Edema, inflammation, or space-occupying lesions (ganglion cyst, tumors, anomalous muscles, or thrombosed ulnar artery) within the canal manifest as ulnar nerve symptoms.

An understanding of the regional anatomy explains the variable presentations of ulnar nerve compression at the wrist in athletes. Shea and McClain's²⁰ classification of ulnar neuropathy at the wrist in relationship to three distinct anatomic zones of the ulnar nerve within Guyon's canal is well-accepted. Type I pathology represents proximal compression and thus manifests as both motor and sensory dysfunction; type II compression represents isolated ulnar nerve motor neuropathy secondary to compression of the deep motor branch along its' course deep to the hypothenar musculature and radial around the hook of the hamate; type III is a pure ulnar nerve sensory neuropathy as a result of compression superficial to the hypothenar muscles and distal to the take-off of the motor branch.

Symptoms may, therefore, range from ring and small finger paresthesias to grip weakness. A Tinel's sign can be elicited in the digital sensory distribution of the ulnar nerve. Motor testing and grading according to the Medical Research Council scale of ulnar-innervated intrinsic muscles in the hand (first dorsal interosseous-index digit abduction, adductor pollicis- key pinch), pinch and grip strength and two-point sensory discrimination are performed in all athletes with suspected ulnar nerve compression within Guyon's canal. Vascular disorders (hypothenar hammer syndrome) are distinguished from nerve compression with use of the Allen test, noninvasive vascular studies and angiography. Thorough examination with provocative maneuvers (elbow flexion with pressure provocation²¹) and specific evaluation of the flexor digitorum profundus to the small finger and dorsal ulnar sensory nerve territory differentiate proximal from distal compressive ulnar neuropathy.

Radiographs are indicated in the setting of trauma (hamate or pisotriquetral fracture) or suspected carpal instability or space-occupying lesion. Electrodiagnostic studies supplement the physical examination and may help elucidate the level of ulnar nerve compression when the examination is equivocal.

Decompression of the ulnar nerve at the wrist is indicated in the presence of motor weakness or when symptoms persist or worsen despite nonoperative modalities (activity modification, nonsteroidal anti-inflammatory medications and protective padding or splinting). Protective padding and splints, as well as posture modification may be helpful at all stages of treatment to alleviate symptoms and are emphasized postoperatively.

Hook of Hamate Fractures

While hamate hook fractures constitute only 2% of all carpal fractures, 22 they most often occur in select sports, such as golf, tennis, baseball and hockey and in the hand that grips the base of the club, racquet, bat, or stick. Delayed diagnosis and persistent symptoms may negatively effect athletic performance.²³ In both the acute or sub-acute or chronic settings, the athlete will complain of vague discomfort at the volar-ulnar aspect of the palm, which is exacerbated with power grip. Tenderness is elicited with palpation over the hook of the hamate. While wrist motion often is painless and unlimited, resisted ring and small finger digital flexion may reproduce pain and weakness. In chronic cases, dysesthesias may be present in the ulnar nerve distribution and rupture of the ring and small finger flexor digitorum profundus tendons may occur secondary to attrition.

The complex three-dimensional carpal anatomy and resultant osseous overlap limits the utility of standard radiographic views. Although a carpal tunnel view may reveal the pathology, its use is limited in the acute setting because the positioning of the wrist required to see the base of the hook is not well-tolerated. We advocate early use of CT with coronal and sagittal plane reconstructions to confirm the

Closed treatment with cast immobilization²⁴ and open reduction with internal fixation²⁵ have yielded variable outcomes as a result of the intrinsic muscular forces acting on the hook of the hamate and the mass effect created by hardware. Ulnar neuropathy secondary to a hook of the hamate fracture is treated with complete excision of the fracture and neurolysis of the ulnar nerve in the acute and chronic settings. After isolation and protection of the motor branch of the ulnar nerve, the entire hook and all comminuted fragments are excised to the level of the fracture to minimize the risk of persistent or progressive symptoms.

Return to sports is symptom-dependent. Scar desensitization is initiated early to facilitate return. After excision, early rehabilitation with progressive motion and strengthening is started. The athlete may benefit from use of a glove with a volar-ulnar pad over the area of the incision.

Piso-triquetral Compartment

Repetitive racquet sports may create pisiform hypermobility or frank subluxation, racquet player's pisiform.²⁶ Resultant pisotriquetral synovitis, chondromalacia, or arthrosis can create ulnar nerve compression. Nonoperative modalities such as activity modification, protective padding or splinting, nonsteroidal anti-inflammatory medications, and pisotriquetal steroid injections can be instituted to alleviate symptoms. For refractory cases or patients with motor weakness, neurolysis and subperiosteal excision of the pisiform²⁶⁻ ²⁸ to preserve the flexor carpi ulnaris are performed.

Cyclist Handlebar Palsy

Long-distance cyclists are at risk for distal or low ulnar neuropathy, handlebar palsy. 29,30 The proximity of the ulnar nerve to the hamate and pisiform and the diminutive hypothenar palmar fascia make the nerve susceptible to compression when the cyclist assumes the power pedaling posture with the ischial tuberosities unloaded and his body weight transferred to the hyperextended wrists and hypothenar eminences. In a prospective study of long-distance cyclists, Patterson *et al.*^{31•} reported that 92% of cyclists experience either motor or sensory symptoms, or both. Interestingly, no significant differences in the incidence of motor or sensory symptoms were found among cyclists of various experience levels. The electrophysiologic sequelae of longdistance cycling on the ulnar nerve have been documented. 4. To decrease the incidence of cyclist's palsy, activity modification, handlebar padding, padded cycling gloves and frequent changing of hand positions are recommended.

Radial Nerve Compression Syndromes at the Wrist

Cheiralgia Paresthetica or Wartenberg Syndrome

Wartenberg³² first described entrapment neuropathy involving the sensory branch of the radial nerve (SRN) cheiralgia paresthetica. 33,34 The anatomy of the SRN has been previously described. 35–37• Most commonly, the SRN is compressed at the site where it pierces the fascia between the brachioradialis and extensor carpi radialis longus to become subcutaneous. The SRN emerges dorsal to the brachioradalis 8–9 cm proximal to the radial styloid.³⁷•

Dellon and MacKinnon³³ hypothesized that restriction of free gliding of the SRN at its' fascial hiatus was responsible for the associated neuritic symptoms. Symptomatic SRN compression is most often seen in athletes involved in sports that require repetitive, forceful pronation-supination of the forearm, which may induce SRN traction. Direct trauma also is an etiology, and athletes may present with symptoms referable to the SRN after a strike to the dorsoradial aspect of the midto-distal forearm above their protective gloves during a hockey or lacrosse match or against another player's helmet in football.

Classic symptoms include neuritic pain radiating to the thumb and paresthesias over the dorsum of the thumb and dorsoradial hand. A Tinel's sign can be elicited over the site of compression. The symptoms are exacerbated with wrist ulnar deviation, and therefore a false-positive Finklestein's test may be present.³³

Most athletes are responsive to nonoperative treatment with ice, rest, nonsteroidal anti-inflammatory medications, corticosteroid injections, and splinting in supination.³³ Neurolysis is only recommended for recalcitrant cases and involves decompression of the SRN from the radial styloid through the fascia joining the brachioradialis and extensor carpi radialis longus. Given the variable cutaneous innervation patterns along the dorsum of the hand, it is essential to confirm that these symptoms do not represent compression of the lateral antebrachial cutaneous nerve. An injection with a local anesthetic will serve as both a diagnostic, as well as a therapeutic modality before considering operative decompression.

Distal Posterior Interosseous Nerve Impingement *Syndrome*

The posterior interosseous nerve (PIN) is the terminal branch of the radial nerve. At the wrist, it lies on the floor of the fourth extensor compartment ulnar to Lister's tubercle before crossing the scapholunate ligament and innervating the dorsal wrist capsule. 38 Dorsal wrist pain in athletes participating in sports that demand forced, repetitive wrist hyperextension(gymnasts, weight-lifters, and football linemen) is not uncommon. These symptoms most commonly are caused by dorsal wrist ganglia, scapholunate ligament pathology, or dorsal wrist impingement.

Additionally, repetitive dorsal wrist trauma may lead to distal PIN impingement syndrome as described by Carr and Davis.³⁹ Athletes present with dorsal wrist pain exacerbated with maximal wrist dorsiflexion and tenderness over the fourth extensor compartment. Pain relief with selective anesthestic block of the PIN 3 cm proximal to the radiocarpal joint aids in the diagnosis. While over 50% of athletes may improve with immobilization and anti-inflammatory medications,³⁹ those who remain symptomatic and unable to return to their desired level of play are indicated for PIN neurectomy. At the time of surgery, significant PIN perineural fibrosis and hypertrophy usually are observed. Furthermore, dorsal wrist ganglia arising from the scapholunate ligament may cause symptomatic local PIN impingement.³⁸ Therefore, at the time of ganglia excision, concomitant PIN neurectomy also should be considered.

Digital Nerve Neuropathies

Localized nerve compression syndromes at the digital level remain rare. Perineural fibrosis of the thumb ulnar digital nerve (bowler's thumb) is the most commonly reported digital nerve syndrome. 40-42 The thumb ulnar digital nerve is subjected to recurrent direct trauma at the level of the metacarpophalangeal joint with cyclical gripping and release of a bowling ball as a result of its' superficial location. The athlete presents with pain and paresthesias along the ulnar side of the thumb, and a localized Tinel's sign may be elicited. 41 Twopoint discrimination often is normal but may be altered in advanced cases. With continued activity, there is progressive perineural scarring and cicatrix formation such that the subcutaneous mobility and excursion of the digital nerve become exceedingly limited. In severe cases, nerve thickening may be palpated as a subcutaneous cord. Perineural fibrosis also may occur in the long and ring fingers by an identical mechanism. 43 The fibrosis also may involve the adjacent flexor sheath.41

Nonoperative measures are the mainstay of treatment. Modification of the bowler's grip and delivery, as well as ergonomic aids such as a protective thumb shell orthosis often ameliorate symptoms and allow athletes to return to sport.⁴¹ Neurolysis with or without dorsal-ulnar transposition ^{40,41,44} are indicated only in the presence of a neuroma or cord unresponsive to nonoperative modalities. These patients should be counseled that a prolonged postoperative recovery should be anticipated.⁴¹

VASCULAR PATHOLOGIES IN THE ATHLETIC HAND

Vascular pathology associated with repetitive blunt impact hand trauma during high-level sports activities is well established. A spectrum of vascular disorders has been reported in many racquet-club sports, such as tennis, 45 golf, 46 badminton, 47 and hockey, 48 and other sports in which the palms and digits are subjected to direct cyclical blunt impact, such as baseball, ^{49•} handball ⁵⁰ and karate. ⁵¹ Vascular insufficiency in the hands of these athletes may be secondary to aneurysm formation, arterial thromboses, and vasospastic phenomena at the wrist or palm and digital levels. Additionally, traumatic ring avulsion injuries⁵² with resultant vascular compromise have been reported in subcohorts of athletes using braided or nylon rope during competition (rodeo athletes, waterskiers).⁵³ The treatment of sports-related digital avulsion injuries is beyond the scope of this review.

Arterial Aneurysms and Thromboses at the Wrist

The vascular supply to the hand is derived from the superficial and deep palmar arches that represent the terminal branches of the ulnar and radial arteries, respectively. Reciprocal contributions to the superficial and deep arches by the radial and ulnar arteries, respectively, create complete arches in approximately 80% of the population. The ulnar arteryderived superficial palmar arch is the primary contributor to the common digital arteries, which then bifurcate into the proper volar digital arteries.

Repetitive wrist, palm, or digital trauma may disrupt the internal elastic laminae of the regional arteries. Resultant aneurysm formation may be followed by mural thrombi formation, thrombotic occlusion, distal embolic phenonomena, or vasospasm. 48,54 Arterial aneurysms or thromboses have been reported in several athletic populations in the ulnar and radial arteries at the wrist, the superficial and deep palmar arches, and the common digital vessels. These vascular pathologies are more likely to be clinically symptomatic in hands with an incomplete superficial palmar arch, the predominant contributor to the common digital arteries.

Hypothenar hammer syndrome refers to the symptom complex of pain, cold sensitivity, paresthesias in the ulnar digits, cyanosis and weakness related to ulnar artery and superficial palmar arch insufficiency^{48,54} from aneurysm, pseudoaneurysm or thrombus formation. Distal vasospasm may occur secondary to the increased sympathetic tone stimulated by the vascular injury. Repetitive ulnar artery trauma against the hook of the hamate occurs as the artery passes superficial to the pisohamate ligament and distal to the pisiform. 54,55 The vulnerability of the ulnar artery is derived from its relatively superficial location within Guyon's canal, the absence of a substantial protective layer, and its proximity to the hook of the hamate.

Professional baseball players may be exposed to more repetitive hand trauma than any other athletic cohort. Indeed, Ginn et al. 49• reported on the prevalence of microvascular abnormalities already present in the hands of clinically asymptomatic professional baseball players prior to the onset of clinically significant ischemia. Sugawara et al. 56 have shown that the development of digital ischemia is correlated with accumulated playing time. Catchers, in particular, are susceptible to microvascular injury.⁵⁷ This finding is explained by the significant volume of pitches they must catch and current catcher mitt design. As opposed to other positional players who receive the ball in the webbing of the glove (away from the hand), catchers endure more global hand trauma with each catch as a result of single-break, one-handed mitt designs. This accounts for the significant abnormalities on the ulnar side of their hands including reduced digital-brachial indices and Doppler anomalies at Guyon's canal.

Physical examination may demonstrate the presence of a hypothenar mass, which may be pulseless (thrombosis) or pulsatile (aneurysm). A timed Allen test⁵⁸ may demonstrate an incompetent ulnar artery-superficial arch system. Sensory changes also may be present. Long standing arterial pathology may manifest as chronic nail bed changes, digital ulceration, or frank digital gangrene. A complete neurologic examination is performed to assess for concomitant ulnar nerve pathology. Doppler ultrasound, digital-brachial pressure indices, radionuclide flow studies and arteriograms comprise the diagnostic armamentarium and are useful for planning microvascular operative intervention.

When symptoms are mild, nonoperative treatment includes activity modification and supplemental hand padding to reduce the magnitude of energy absorbed by the hypothenar eminence and palm. Microvascular operative intervention is indicated for severe recalcitrant cases, ulnar artery (pseudo-)aneurysm in an ulnar artery-dominant hand, or in patients with incomplete arch formation. Options include excision and patch grafting, resection with end-toend repair, and resection and interpositional vein graft reconstruction. Treatment of thrombosed arterial segments is dependent on the location and extent of the thrombosis. In the appropriate clinical setting, thrombolysis, resection and embolectomy, and arterial reconstruction may be considered. Arterial reconstructive strategies include end-to-end repair, reverse interposition vein grafting and bypass grafting.

DIGITAL MICROVASCULAR DISORDERS

Digital hypoperfusion from repetitive digital microvascular trauma is seen in baseball, 49 , 56 , 57 handball, 50 and volleyball players. Multiple digital arterial aneurysms and thromboses have been reported in karate participants. 51,59 Both digital vascular and nerve trauma at the level of the metacarpal head during sports may cause symptoms of digital weakness, paresthesia, or pain. Symptoms related to acute microvascular ischemia and neurological trauma must be differentiated. Diagnostic modalities include digital Allen testing, digitalbrachial indices, digital ultrasound, digital thermography, and electrodiagnostics.

Baseball catchers, in particular, are prone to digital ischemia. Their mitts tend to concentrate force at the level of the index metacarpal head. The adjacent digital neurovascular bundle therefore sustains repetitive forceful trauma. This accounts for the prevalence of symptoms, soft-tissue hypertrophy, and vascular changes in the index finger of the gloved hand of catchers. 49•,56,57 Angiograms have demonstrated digital artery occlusion, segmental thrombosis, and regional stenosis.⁵⁷ The vibrational impact from catching balls thrown at high velocities has been implicated in patients in whom angiograms have demonstrated microvascular occlusion distal to the level of the proximal interphalangeal joint. 56 Vasospastic phenomena within the pitching hand of baseball players is thought to be caused by sympathetic hyperstimulation from the increased local digital pressures during pitching. Vascular compromise secondary to hypertrophic lumbricals or Cleland's ligaments also has been reported in the throwing hands of baseball pitchers.⁶⁰

For mild symptoms, recommendations include rest and supplemental padding for mitts and gloves. Pharmacologic intervention is patient and pathology specific and may include thrombolytics, vasolytics (calcium-channel blockers, topical nitroglycerin), or sympatholytic agents.⁵⁵ Digital sympathectomy may be considered for severe ischemic changes. If lumbrical hypertrophy is seen on imaging, decompression of the lumbrical canal may be considered.

CONCLUSION

While these neurovascular syndromes at the wrist and hand of athletes remain relatively rare clinical entities, the associated symptoms have deleterious affects on athletic performances. A thorough understanding of the regional nerve and vascular anatomy and appreciation for the repetitive motion and stresses accrued in the wrist and hand of both high-level and recreational athletes are essential for accurate diagnosis and early referral to upper extremity surgeons for further evaluation and treatment. Along with nonoperative and operative management strategies, athletes must be counseled appropriately with regard to anticipated outcomes and preventative strategies to avoid recurrence.

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A cadaveric study was performed to detail the cutaneous innervation patterns of the dorsal hand. Specifically, the anatomic details of the lateral antebrachial cutaneous nerve, the superficial branch of the radial nerve, and the dorsal branch of the ulnar nerve were delineated. Innervation branching patterns were classified in order to delineate safe dorsal skin incisions.

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healthy professional baseball players in all positions, with a significantly higher prevalence in catchers, prior to the development of clinically important ischemia. These included diminished digital brachial indices and abnormal flow in the ulnar artery at Guyon's canal. Repetitive trauma resulting from the impact of the baseball also leads to digital hypertrophy in the index finger of the gloved hand of catchers at the level of the proximal phalanx and the proximal interphalangeal joint. Catchers also reported a higher prevalence of subjective hand weakness in the gloved hand.

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