Chronic exertional compartment syndrome (CECS) refers to exercise-induced, reversible increases in pressure within well-defined inelastic fascial compartments leading to compromised tissue perfusion followed by functional loss, ischemic pain, and neurologic symptoms. Symptoms typically resolve when the activity ceases and there are usually no permanent sequelae. In the upper extremity, this condition most commonly affects athletes during sports requiring repetitive and vigorous gripping, such as rowers. In addition to clinical history and examination, a number of methods aid diagnosis, including compartment pressure measurements, magnetic resonance imaging, and near infrared spectroscopy. When symptoms persist despite conservative treatment, multiple operative techniques have been described to treat CECS including open, mini-open, and endoscopic release of involved compartments. We review the pathophysiology, diagnostic modalities, treatment strategies, and outcomes data for CECS of the upper extremity while highlighting areas of residual controversy. (J Hand Surg Am. 2017;42(11):917–923. Copyright © 2017 by the American Society for Surgery of the Hand. All rights reserved.)

Key words  Athlete, chronic exertional compartment syndrome, fasciotomy, peripheral nerve, upper extremity.

Chronic exertional compartment syndrome (CECS) refers to exercise-induced, reversible increases in pressure within well-defined inelastic fascial compartments after muscle hyperemia and expansion leading to compromised tissue perfusion, followed by ischemic pain, functional loss, and occasionally neurologic symptoms upon sustained exertion. Symptoms typically resolve when the activity ceases and there are usually no permanent sequelae.

ANATOMY

The muscles and tissues of the forearm are divided into 3 main compartments by deep, relatively inelastic fascia: the volar, dorsal, and mobile wad compartments. The volar flexor compartment is subdivided into superficial, intermediate, and deep components.
The muscles of the superficial volar compartment include the flexor carpi ulnaris, pronator teres, flexor carpi radialis, and palmaris longus when present. The flexor digitorum superficialis (FDS) comprises the intermediate compartment. The deep volar compartment includes the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus. The median and ulnar nerves traverse well-defined intermuscular intervals from the level of the antecubital fossa through the forearm and may be secondarily compressed during elevated intracompartmental pressures and dynamic repetitive muscle contraction. The median nerve lies between the superficial and deep heads of the pronator teres in the proximal forearm and then below the FDS arch before it enters the interval between the superficialis and profundus muscle bellies in the midforearm. Distal to the retro-epicondylar groove, the ulnar nerve lies between the 2 heads of the flexor carpi ulnaris (Fig. 1) and enters the midforearm between the flexor carpi ulnaris and FDS.

The mobile wad includes the brachioradialis and extensor carpi radialis brevis and longus. The dorsal extensor compartment contains 9 muscles: extensor digitorum communis, extensor digiti minimi, extensor carpi ulnaris, anconeus, supinator, abductor longus, extensor pollicis brevis, extensor pollicis longus, and extensor indicis. The CECS of the forearm more frequently involves the volar compartments than the extensor compartments.

PATHOPHYSIOLOGY
Normal muscle will hypertrophy with exertion but return to baseline within a few minutes; intracompartmental pressures follow a similar pattern. In documented cases of CECS, muscles expand up to 20% in volume against inelastic fascia and intracompartmental pressures rise in accordance with Laplace’s law. Resultant microvascular compromise and reduced venous return lead to ischemic pain, ultimately manifesting as workload intolerance and loss of function. Symptoms resolve completely between periods of activity and recur once the activity is resumed. Patients with lower-extremity CECS have higher intracompartmental pressures at rest as well as with exercise compared with normal individuals, although this has not been specifically reported for CECS of the forearm.

CLINICAL PRESENTATION
A thorough history and physical examination are vital in the diagnosis of CECS. Patients present with atraumatic exercise-induced pain, cramping, and tightness in the involved compartment. They may also experience loss of grip strength and distal paresthesias. Onset and severity of the exertional pain often become predictable, recurring at similar durations and intensities of exercise. Patients are usually involved in activities requiring prolonged, repetitive gripping motions with short periods of rest, such as rowing, and symptoms can present within 2 to 5 minutes of full exercise. Symptoms may persist for some time after cessation of the inciting exercise as the pressures in the compartment equilibrate to restore sufficient microcirculation and venous outflow in the involved compartment. Symptoms typically recur.
upon return to sport even after extended periods of activity modification and cessation of training.

Static physical examination is usually normal and unrevealing. Provocative signs for peripheral compressive neuropathies are often negative and vascular examination is normal. Dynamic examination of the athlete and the involved compartments during training is essential for the diagnosis. The compartment(s) are noted to be firm, taut, and tender to palpation. Distal paresthesias may be present. In

TABLE 1. Outcomes of Wide-Open, Mini-Open, and Endoscopic-Assisted Fasciotomies

<table>
<thead>
<tr>
<th>Technique</th>
<th>Studies</th>
<th>Fasciotomies, n</th>
<th>Proportion of Patients With Full Resolution (%)</th>
<th>Complications</th>
<th>Average Time of Return to Training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open</td>
<td>Croutzet et al (2009); Harrison et al (2013); Barrera-Ochoa et al (2016)</td>
<td>54</td>
<td>53 of 54 (98.1%) with 1 recurrence of symptoms</td>
<td>8 of 54 (14.8%) with 1 mild sensory ulnar paresthesia, 4 with hematomas, 1 skin problem, and 2 superficial infections</td>
<td>3.88 wk (range, 1–6 wk)</td>
</tr>
<tr>
<td>Mini-open</td>
<td>Zandi and Bell (2005); Brown et al (2011); Barrera-Ochoa (2016)</td>
<td>42</td>
<td>36 of 42 (85.7%) with 3 partial resolutions of symptoms and 3 with no improvement</td>
<td>9 of 42 (21.4%) with 5 instances of scar widening, 2 hematomas, 1 cutaneous problem, and 1 superficial infection</td>
<td>4.77 wk (range, 0–16.2 wk)</td>
</tr>
<tr>
<td>Endoscopic</td>
<td>Hijjawi and Nagle (2010); Jans et al (2015)</td>
<td>155</td>
<td>154 of 155 (99.4%) with 1 recurrence of symptoms after 8 mo</td>
<td>5 of 155 (3.2%) with 5 instances of subcutaneous hematomas</td>
<td>6.11 wk (range, 6–24 wk)*</td>
</tr>
</tbody>
</table>

*One patient from the case report by Hijjawi and Nagle reported complete resolution of symptoms at 24 weeks, but it is unclear if this referred to a return to training or resolution at a follow-up.

FIGURE 3: 3-Tesla noncontrast MRI of the right elbow with axial short tau inversion recovery imaging of the elbow and forearm A before and B after exercise. On the pre-exercise images, muscle bulk and signal are normal. On the post-exercise images, T2 hyperintense signal is noted within the brachioradialis and extensor carpi radialis longus and brevis muscles. These dynamic MRI findings are consistent with exercise-induced muscle edema in the extensor compartment of the proximal forearm in this elite rower.
FIGURE 4: Volar compartment release with peripheral nerve decompression. A The interval and investing fascia between the brachioradialis and pronator teres is identified after mobilization of the cephalic vein and lateral antebrachial cutaneous nerve (LABCN) (scissors). A volar fasciotomy is then performed and the radial artery and venae comitantes (forceps) are mobilized. B The interval between the radial vessels and the radial sensory nerve (RSN) along the underside of the brachioradialis is developed to identify the superficial pronator teres myotendinous junction (which lies radial to the radial vessels and ulnar to the RSN). C Z-Lengthening of the pronator teres myotendinous insertion is performed in the RSN–radial vessel interval. D The radial vessels are mobilized radialward and the median nerve is identified ulnar to the radial vessels. The deep head of the pronator teres (forceps) is then released along the radial border of the median nerve. E After release of the deep head of the pronator teres, the fibrous arch of the FDS is seen tethering and compressing the median nerve. This finding accounts for dynamic median nerve symptoms during intensive rowing. F Complete neurolysis of the median nerve in the proximal forearm. The arborization of the median nerve includes the visualized branches to the pronator, flexor carpi radialis, and FDS, and the anterior interosseous nerve takeoff is seen on the radial border of the median nerve (pointer). G After median nerve decompression and when radial tunnel decompression is indicated, it may be performed via the same volar compartment fasciotomy incision. The RSN is fully neurolysed along the deep surface of the brachioradialis. Crossing radial recurrent vessels are ligated. The RSN is further neurolysed to the radial nerve proper bifurcation into the posterior interosseous nerve (PIN) and RSN. H The radial nerve bifurcation is clearly visualized. The brachioradialis is elevated to reveal the underlying fibrous arch of the extensor carpi radialis brevis (ECRB) (forceps). The ECRB fibrous arch was taut along the PIN takeoff, which accounted for symptoms consistent with radial tunnel syndrome (reproducible pain in the proximal-middorsal-radial forearm with rowing and activities of daily living). The fibrous ECRB arch may dynamically compress the PIN with forceful repetitive wrist extension. I Tenotomy of the ECRB fibrous arch is performed and the underlying arcade of Frohse is seen (the proximal leading edge of the supinator [pointer]). J The supinator is completely released through its distal margin. Distal intramuscular tendinous formations are released.
several reported cases there were associated neurological symptoms including digital numbness. Compressive fascial bands across the median nerve beneath the FDS arch have been noted during decompression (Fig. 2).1,3

DIAGNOSTIC WORKUP
The patient’s clinical history is the most important factor for diagnosis. Thorough static and dynamic physical examination aids in eliminating other etiologies. Intracompartmental pressure measurements before, during, and after exercising for the diagnosis of CECS of the forearm are predicated on protocols reported for the diagnosis of CECS of the lower extremity. The diagnostic criteria for CECS in the lower extremity published by Pedowitz et al1 include resting pressure greater than 15 mm Hg, pressure 1-minute after exercise > 30 mm Hg, and/or pressure 5 minutes after exercise greater 20 mm Hg. These criteria have been used for over 2 decades.

Controversy remains about the role, diagnostic validity, and sensitivity of these criteria. Variable baseline values for resting and post-exertion forearm intracompartmental measurements, coupled with a spectrum of delays in intracompartmental pressures returning to baseline, make reproducible diagnostic thresholds elusive. Studies suggest that further research is necessary under standardized conditions to investigate diagnostically relevant parameters because previous authors were unable to find uniform recommendations.5

Hutchinson6 suggested the use of a change of 10 mm Hg in intracompartmental pressure as diagnostic, independent of baseline measurements. Roscoe et al7 proposed continuous dynamic intramuscular compartment pressure monitoring in diagnosing CECS to improve the diagnostic validity, compared with the criteria of Pedowitz.1 A diagnostic threshold of 105 mm Hg at 5 minutes into exercise during dynamic intracompartmental measurements has also been proposed.

Achieving accurate and reproducible intracompartmental pressure measurements during exercise is technically demanding, whether it is done at specified intervals or in a continuous fashion. The level of catheter placement, position of the catheter within the compartment, and variation in the position of the extremity all affect pressure measurements. Some clinicians omitted invasive monitoring and offered surgical intervention based on clinical history alone.4

Recently, dynamic magnetic resonance imaging (MRI) (Fig. 3) has been highlighted as a potential diagnostic aid in the diagnosis of CECS.8–10
Verleisdonk et al\textsuperscript{10} used dynamic MRI as a diagnostic measure for chronic exertional compartment syndrome of the lower leg because there was a significant increase in T2 signal intensity of the involved compartment after exercise compared with controls. The T2 postexercise hyperintensity resolved after decompression fasciotomy. In a study of CECS in the forearm of motocross racers, Gielen et al\textsuperscript{9} found that postexertional MRI showed an increase in signal intensity and signal-to-noise ratio in the FDS and flexor digitorum profundus in all motocross racers with CECS and a minor signal intensity and signal-to-noise ratio increase in the extensor carpi radialis longus compared with asymptomatic motocross racers. By measuring the change in signal intensity and signal-to-noise ratio in forearm muscles after exertion, those authors were able to discern between symptomatic and asymptomatic motocross racers.

Van den Brand et al\textsuperscript{8} demonstrated that the diagnostic sensitivity of noninvasive near-infrared spectroscopy is equivalent to that of intracompartmental pressure measurements. Near-infrared spectroscopy diagnoses CECS by measuring hemoglobin saturation of tissues, which mostly reflects the oxygen saturation in tissues and thus the level of local ischemia.\textsuperscript{8,11} This technique is not currently widely used in clinical practice.

When there are associated neurological symptoms, nerve conduction studies (NCS) are recommended. Static nerve conduction studies and electromyography results are usually normal; nevertheless, the role of dynamic NCS is available at select centers. However, dynamic NCS may demonstrate the same inaccuracies encountered with other dynamic nerve compression problems such as pronator syndrome. Myelin damage is required to produce an alteration in nerve conduit velocity.

**TREATMENT**

When symptoms persist despite nonsurgical management, surgical fasciotomy is considered for symptom relief and return to athletic capacity.\textsuperscript{3,4,12}
Athletes are counseled that not all patients experience full symptom resolution after fasciotomy. 13,14 Although most cases documented in the literature report complete resolution of symptoms and a return to athletic training, some report only partial symptom resolution and several incidences of scar widening. 3,10,13 Table 1 lists outcomes after open, mini-open and endoscopic fasciotomy. 3,4,12,13,15,16

Some authors suggest that endoscopic-assisted and mini-open fasciotomies reduce time to return to competition. 4,15,16,17 These techniques evolved owing to the belief that only the proximal two-thirds of the compartment needs to be released, because that is where the expansile muscle bellies lie. 16 Harrison et al 16 and Croutzet et al 15 reported complete resolution of preoperative symptoms in 6 and 8 athletes, respectively, after mini-open fasciotomy; all athletes returned to sports in 4 and 6 weeks, respectively. Barrera-Ochoa et al 16 compared results of mini-open and open fasciotomies and found no significant difference in visual analog scale and Quick-Disabilities of the Arm, Shoulder, and Hand questionnaire scores, patient satisfaction, or return to activity time in 34 motocross racers. More minor complications occurred after mini-open fasciotomies, including hematomas, unspecified skin issues, and superficial infections. 16

Open fasciotomy allows full visualization of neurovascular structures and the muscles within the compartment, as well as full fascial release, and may minimize complications. 3,16 When there are associated preoperative nerve symptoms or electrophysiological evidence of concomitant compressive peripheral neuropathy, we and others advocate consideration of formal peripheral nerve decompression at the time of open fasciotomy through the same incision. 3 The median and radial nerves in the proximal forearm can be fully decompressed through a volar radial proximal forearm incision at the time of volar compartment releases (Fig. 4). Alternatively, the radial tunnel may be decompressed via a dorsal interval (ie, brachioradialis–extensor carpi radialis longus) if the extensor compartment requires release and there are dynamic findings of radial nerve compression (Fig. 5).

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REFERENCES