Compartment Syndrome and Volkmann Ischemic Contracture

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Acknowledgment: The authors gratefully acknowledge the contributions of Dr. Ayan Golgonu and Dr. Kagan Özer, who wrote the chapter on compartment syndrome that appears in the sixth edition of Green’s Operative Hand Surgery.

These videos may be found at ExpertConsult.com:
- 51.1 Surgical technique of upper extremity fasciotomy, cadaver demonstration.
- 51.2 Ten-year follow-up on flexor origin slide for “moderate”-type Volkmann contracture.
- 51.3 Eighteen-month follow-up on functional free muscle transfer for finger flexion for “severe”-type Volkmann contracture.

COMPARTMENT SYNDROME

Acute compartment syndrome (ACS) of the extremity is a diagnosis requiring emergent surgical intervention. It is characterized by sustained elevation of tissue pressure within an osseofascial or fascial compartment that exceeds tissue perfusion pressure. This results in local circulatory impairment, ischemia, cellular anoxia, and ultimately tissue death. ACS represents the acute phase of the injury during which time surgical intervention can reduce the extent of irreversible muscle injury. Timely diagnosis and treatment are critical in reducing the extent of permanent changes within muscle and nerve tissue. Even with emergent treatment, there may be permanent disability in the affected extremity and a subsequent need for additional surgery, including amputation.21 Volkmann ischemic contracture represents the late sequelae of compartment syndrome. This disorder has a spectrum of fixed muscle contractures and muscular and neurologic impairments. Numerous authors have contributed to our understanding and treatment of this condition. Table 51.1 summarizes some of the historical events in the recognition of this entity, its etiology, its pathologic findings, and its sequelae.

Types of Compartment Syndromes

Acute Compartment Syndrome

Acute compartment syndrome (ACS) occurs when tissue pressures rise high enough within an osseofascial compartment to cause tissue ischemia. The resultant tissue injury can range from reversible muscle swelling to permanent tissue necrosis depending on the magnitude and duration of tissue pressure elevation.

When pathologic tissue pressure elevation has been present for less than 4 hours, ACS is in the early stage; when pathologic tissue pressure elevation has been present for more than 4 hours, ACS is in the late stage.

Impending Compartment Syndrome

Impending compartment syndrome represents a clinical setting in which a compartment syndrome is at risk of developing; however, tissue pressure is not yet sufficiently elevated to cause muscle ischemia. Clinical scenarios in which this may occur include limb reperfusion after prolonged ischemia, posttraumatic swelling of a limb, or high-energy injuries.

Exercise-Induced or Exertional Compartment Syndrome

Exercise-induced compartment syndrome is a reversible tissue ischemia due to a noncompliant fascial compartment that is unable to accommodate muscle expansion occurring during exercise. It has been described in both the upper and lower extremities and is different from ACS in that the symptoms are reversible after cessation of exercise. Emergent surgery is not usually indicated.10,29 Both traditional fasciotomy and endoscopically assisted fasciotomy have been used to treat exercise-induced compartment syndrome.

Crush Injury or Crush Syndrome

Crush injury is the external compression of an extremity, as might occur in a building collapse or construction injury or in an obtunded patient who lays on an extremity for a prolonged period. The compression of the extremity leads to muscle ischemia and reperfusion injury as the compression is relieved. This process of events can lead to compartment syndrome. Crush syndrome is a localized crush injury with systemic manifestations. Reperfusion of the affected extremity can rapidly release muscle breakdown products into the system, which can lead to renal failure or death.13

Neonatal Compartment Syndrome and Neonatal Volkmann Contracture

Both neonatal compartment syndrome and neonatal Volkmann contracture have been reported. Awareness of this diagnosis is important because early recognition and treatment can improve the functional outcome and growth in neonatal compartment syndrome. In addition to swelling of the forearm, there is often a characteristic skin lesion on the proximal lateral arm, known as the sentinel lesion of neonatal compartment syndrome.46
Established neonatal Volkmann contracture cannot be improved by early intervention; however, awareness of this diagnosis can aid in counseling of the family and treatment of the patient (Figure 51.1, A and B).

**Volkmann Ischemic Contracture**

Volkmann ischemic contracture is the end result of prolonged ischemia, is associated with irreversible tissue necrosis, and has a spectrum of presentations.

**Etiologic Findings and Incidence**

Compartment syndrome in the upper extremity is most commonly associated with trauma. A variety of conditions and injuries can lead to ACS. These include fractures, penetrating trauma, closed soft tissue injuries, infection, animal and insect bites, extravasation injuries, ischemia-reperfusion injury, external compression by tight dressings or casts, burns, or crush injuries. Compartment syndrome in the absence of fracture should raise concern about an underlying bleeding disorder (Figure 51.2).

The incidence of upper extremity compartment syndrome is difficult to determine. The estimated incidence in pediatric upper extremity fractures is approximately 1%. In a large series of trauma patients, the incidence of fasciotomy associated with all upper extremity traumas was 0.41%. Branco and colleagues noted a decreasing incidence of the need for surgical fasciotomy over a 10-year period, despite stable injury severity scores, with the overall incidence for upper and lower extremity trauma decreasing from 3.2 to 0.7%.

Historically, ACS of the upper extremity in the pediatric population was most commonly reported in association with supracondylar humerus fractures. Likely, this was related to the historical treatment methods of casting with the elbow in a position of hyperflexion. Currently, pediatric fracture patterns most associated with forearm compartment syndrome are both-bone forearm fractures and supracondylar humerus fractures associated with distal radius fractures (i.e., floating elbow fractures).
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FIGURE 51.2 This 4-year-old presented with hand and forearm swelling after minor trauma. Other areas of bruising were noted. After workup, the child was found to have hemophilia A. He underwent fasciotomy of his hand and forearm. A, Swelling of the hand and forearm. B, After dorsal fasciotomy of the hand and volar extended carpal tunnel release and forearm fasciotomy. C, Bruising of the periorbital region.

Pathophysiologic Findings

Compartment syndrome is more prevalent in males younger than 35 years of age, penetrating trauma, open fractures, elbow dislocations, and vascular injuries. The need for surgical fasciotomy increases dramatically when vascular injury is present. Compartment syndrome in the hand is most commonly associated with intravenous injections.

Pathophysiology of ACS is complex. Several theories and models have been developed. The common prerequisite is a soft tissue structure (usually fascia) that prevents muscle expansion when the muscle is exposed to increased fluid volume. In all cases, the final common pathway is cellular anoxia.

Matsen presented a “unified concept” of compartment syndrome that incorporates several mechanisms of vascular compromise all leading to cellular injury. Increased compartmental pressure occurring from either internal injury (edema, reperfusion, or bleeding) or external injury (tight cast or dressing, pressure garment) causes a decreased perfusion gradient between arteriole and venous pressures and a resultant decrease in local tissue perfusion. Decreased tissue perfusion results in further tissue insult, increased capillary leakage, and further increase in intracompartmental pressure. This causes a vicious cycle of increasing cellular ischemia leading to further capillary leakage and swelling and increasing compartment pressures (Figure 51.3).

Ongoing ischemia eventually leads to cell death and lysis of the myocyte. Degradative enzymes are activated and released into the interstitial tissues, causing further tissue necrosis. The extent of muscle injury depends on the duration of ischemia and the metabolic rate of the tissue. Prolonged ischemia can ultimately lead to liquefactive necrosis of the muscle compartment.

Diagnosis

Diagnosis of Acute Compartment Syndrome

The diagnosis of ACS is principally based on clinical examination. Maintenance of a high index of suspicion, particularly in the setting of at-risk injuries and conditions, aids in the prompt recognition and treatment of this condition.

Although compartment syndrome is frequently associated with fractures, many other causes can also lead to ACS. Causes are commonly separated into intrinsic causes (typically, bleeding or swelling into a compartment) and extrinsic causes (applied external pressure preventing a compartment from expanding) (Box 51.1). The physician must keep this in mind because ACS not associated with fracture frequently has a delayed diagnosis and worse clinical outcomes.
BOX 51.1 Injuries That May Lead to Compartment Syndrome

- Fracture in child
- Supracondylar fracture of humerus, especially one associated with distal radius fracture
- Both-bone forearm fractures (intramedullary nailing of pediatric both-bone and radial head fractures is associated with difficult reduction and prolonged tourniquet times)
- Distal radius fracture
- Upper extremity fractures associated with vascular injury
- Soft tissue trauma without fracture
- Blunt trauma without fracture or skin violation
- Fracture in adult
- Fracture of humerus, especially one associated with distal radius fracture
- Edema associated with burn injury
- Electrical burns associated with deep muscle damage and edema
- Systemic inflammation and fluid resuscitation
- Burn eschar causes an extrinsic compression
- Animal and insect bites (e.g., Crotalus [pit viper] snakebite)
- Treatment of impending compartment syndrome from snakebite should be with antivenin

The hallmarks of diagnosis of ACS have classically been described as the six Ps of compartment syndrome (Table 51.2): pain, pressure, paresthesias, pallor, paresis, and pulselessness. Some authors have added or substituted poikilothermia of the extremity as one of the Ps. This use of the term poikilothermia is not completely accurate but indicates that the affected extremity is cool relative to body temperature. In most series, pain has been the earliest and most reliable finding. However, pain peaks at 2 to 6 hours of ischemia and then gradually subsides as muscle necrosis progresses and nerve function becomes impaired. Therefore, patients with a late presentation of or late diagnosis of compartment syndrome have less pain than those with an earlier presentation.

It has been suggested that in pediatric patients, “it’s the As, not the Ps” that signal ACS.6 These As are an increasing requirement for analgesia, the presence of anxiety (or restlessness), and the presence of agitation (or crying).149 Children are unable to articulate the feeling of paresthesias, and sensibility testing is unreliable.

Certain conditions can make the clinical diagnosis of ACS challenging. Altered levels of consciousness as can occur in head trauma, a medically induced coma, or obtundation from other causes can obscure the normal pain response that is one of the early signs of compartment syndrome. Similarly, distracting pain from polytrauma, neurologic injury in the affected limb, and/or regional anesthetic blockade can mask the signs and symptoms of compartment syndrome. Diagnosis is also more.

<table>
<thead>
<tr>
<th>Sign or Symptom</th>
<th>Description</th>
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<tbody>
<tr>
<td>Pain</td>
<td>Described as deep, constant, often poorly localized pain that is disproportionate to the physical findings; often poorly responsive to analgesics. Pain is accentuated with passive stretching of the involved compartment. Although this is the most consistent and reliable finding, cases of “silent” compartment syndrome of the lower extremity have been reported (Badhe) and likely also occur in the upper extremity. Pain usually peaks at around 2 to 6 hours of ischemia and then subsides. Patients who present late with acute compartment syndrome may not report as much pain.</td>
</tr>
<tr>
<td>Pressure</td>
<td>Affected compartment(s) are firm and noncompressible; often described as “rock hard”</td>
</tr>
<tr>
<td>Paresthesias</td>
<td>Numbness or “pins-and-needles” sensation in the cutaneous distribution of the nerves that traverse the affected compartment.</td>
</tr>
<tr>
<td>Pallor</td>
<td>Usually pale, but the extremity may also appear blanchy and often cool (see poikilothermia)</td>
</tr>
<tr>
<td>Paralysis</td>
<td>Late and unreliable finding. Muscle paralysis may be pain related. When true paralysis is present, this is a poor prognosticator for recovery.</td>
</tr>
<tr>
<td>Pulselessness</td>
<td>Also usually a late finding and poor prognosticator for recovery</td>
</tr>
</tbody>
</table>

Table 51.2 Diagnostic Findings in Compartment Syndrome
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TABLE 51.3  Measurement of Compartment Pressures

<table>
<thead>
<tr>
<th>Aspect of Measurement</th>
<th>Considerations</th>
</tr>
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<tbody>
<tr>
<td>Patient position</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Supine</td>
</tr>
<tr>
<td></td>
<td>Measured extremity should be at heart level</td>
</tr>
<tr>
<td></td>
<td>Measurements in uncooperative patients and children should be made with the patient sedated or in the operating room with the patient sedated</td>
</tr>
<tr>
<td>Needle</td>
<td>18-g straight, side-port, slit catheter, or wick</td>
</tr>
<tr>
<td>Transducer</td>
<td>Handhold digital (Stryker pressure transducer)</td>
</tr>
<tr>
<td></td>
<td>Arterial line monitor</td>
</tr>
<tr>
<td>Needle placement</td>
<td>Perpendicular to muscle belly</td>
</tr>
<tr>
<td></td>
<td>Several readings at multiple depths</td>
</tr>
<tr>
<td></td>
<td>Near fracture site (see Figure 51.4, localized compartment)</td>
</tr>
<tr>
<td></td>
<td>Near area of maximum tension</td>
</tr>
<tr>
<td>Threshold pressure</td>
<td>30 to 40 mm Hg</td>
</tr>
<tr>
<td></td>
<td>Within 30 mm Hg of diastolic blood pressure or mean arterial pressure</td>
</tr>
<tr>
<td>Contraindications to measuring compartmental pressure</td>
<td>Underlying neurovascular structures at risk</td>
</tr>
<tr>
<td></td>
<td>Coagulopathy</td>
</tr>
<tr>
<td></td>
<td>Diffuse cellulitis</td>
</tr>
</tbody>
</table>

FIGURE 51.4  Localized compartment syndrome. This 16-year-old boy sustained a coronoid process fracture and a mildly angulated distal radius fracture through an old fracture malunion. He had pain and localized swelling at the distal forearm and subjective numbness in the median and ulnar distributions in the hand. His fingers were clenched and his thumb was flexed. He had pain with passive extension of the fingers. He underwent extended carpal tunnel release and fasciotomy. Compartment swelling was localized to the distal forearm. The proximal incision was closed at the time of primary surgery. Delayed primary wound closure was done after 4 days. A, Localized forearm swelling and clenched posture of the fingers and thumb. B, On postoperative day 2, persistent swelling of distal forearm musculature is present.

difficult in children and infants who may have difficulty in cooperating with examination, are nonverbal, or are apprehensive and crying. Also, the thicker layer of subcutaneous fat in children may contribute to a false sense of a soft compartment on palpation, further complicating the diagnosis. Diagnosis or exclusion of compartment syndrome on clinical grounds alone is often impossible.

If the clinician is uncertain of the diagnosis based on equivocal physical findings, compartment pressure can be measured (Figure 51.4 and Table 51.3). Several different methods can be used. Continuous pressure measurements can be obtained with a wick catheter or connection to a continuous pressure monitor. Typically, the newer digital devices are used to assess or monitor the compartment. These devices are sensitive to patient motion and should not supplant repeat clinical examinations. Although controversial, the thresholds/indications for fasciotomy are an absolute pressure greater than 30 to 40 mm Hg or pressures within 30 mm Hg of either the diastolic blood pressure or the mean arterial pressure.

Although pulse oximetry of the affected extremity has not been found to be useful, near-infrared spectroscopy (NIRS) may prove to be useful in the early diagnosis and monitoring of an impending compartment syndrome. This technique was proposed as a method of monitoring for compartment syndrome as early as 2001, but it has not been widely adopted because of its cost and problems with availability of sensors and equipment. NIRS is noninvasive and capable of measuring the oxygenation state of at-risk tissues and may gain wider use in the future. NIRS is limited by the depth of tissue penetration (2 to 3 cm) and the presence of hematoma within the compartment.

In these questionable clinical situations, the safer intervention is to perform a fasciotomy. Bulging of the muscle compartment and clinical softening of the extremity at the time of fasciotomy confirms the diagnosis.

Diagnosis of Exertional Compartment Syndrome

Chronic exertional compartment syndrome of the upper extremity has been described in the flexor compartment of the forearm and in the anconeus muscle; in the hand, it has been described specifically in the adductor of the thumb. Patients with chronic exertional compartment syndrome typically complain of pain that starts as a dull ache within the first 30 minutes after starting an activity. Burning, cramping, or aching pain progresses as the activity is continued. The pain escalates to a level of discomfort where the patient can no longer continue or where it adversely affects the patient’s performance. Activities associated with exertional compartment syndrome of the upper extremity include sports such as rowing, bicycle riding, or motorcycle riding or repeated episodes of manual labor requiring a prolonged grip or pinch. The pain and tissue firmness resolve spontaneously with cessation of the activities. Diagnostic studies have principally involved measurement of compartment pressures before, during, and after exercise. More recently, magnetic resonance imaging has been used before and after exercise as a diagnostic tool. A signal change in

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T2 imaging in an isolated fascial compartment associated with activity supports the diagnosis of exercise-induced compartment syndrome.25

Pertinent Anatomy

The compartments of the upper extremity are listed in Table 51.4. Any of these anatomic spaces can be affected by tissue pressure elevation. Although elevated tissue pressures are most commonly seen in osseofascial spaces, other tissue structures provide a rigid barrier that does not expand sufficiently to accommodate swelling. In the upper extremity, the fascial components and the skin can act as barriers that require decompression at the time of surgery.

Treatment

The goal of treatment is to prevent tissue necrosis, avert neurovascular compromise, and avoid permanent functional deficits. These devastating complications can be minimized or avoided with early recognition and prompt intervention.

The first step in treatment is to remove all possible extrinsic causes of pressure, including circumferential dressings, cast padding, and casts. Casts have been shown to restrict compartment expansion by 40%. Release of a cast and subsequent pressure elevation by 40 to 60% depending on the presence of dry or wet blood on the cast padding.26 The limb should be elevated only to heart level. Although limb elevation may decrease swelling, it can also reduce perfusion to the affected limb, causing exacerbation of tissue ischemia.

A history of bleeding disorders or use of anticoagulation therapy should be obtained from the patient or family member.

Laboratory analysis should include a complete blood count, prothrombin time, and partial thromboplastin time. If there is a suspicion of a bleeding disorder, a hematologist should be involved in the evaluation and treatment of the patient. Urinalysis for myoglobin, serum electrolytes, creatinine, and myoglobin should be obtained in the setting of prolonged ischemia, crush injury, or ischemia-reperfusion injury. Medical management of shock, hypoxia, metabolic acidosis, and electrolyte imbalance should be addressed immediately. Supportive care with vigorous hydration, correction of metabolic function, and treatment of hyperkalemia is necessary to prevent sequelae such as renal failure, shock, hypothermia, cardiac arrhythmias, or cardiac failure. Supportive care should be initiated in the emergency room but should not delay surgical treatment.

Hypertonic mannitol has been used to lower intracranial pressure. The use of mannitol in decreasing extremity swelling has been described in animal models and limited case studies. It has not gained widespread use for treatment of impending or acute compartment syndrome. Nonetheless, it has been hypothesized that the use of mannitol may have played a role in the decreased incidence of compartment syndrome in trauma patients at a single medical center over a 10-year time period.9 Hypertonic mannitol has been found to decrease endothelial swelling and may help reduce muscle necrosis in its function as an oxygen-free radical scavenger.62 In one clinical report, patients were given a 100-mL bolus of 20% mannitol, followed by an infusion of 10 g/hour for 6 to 24 hours.11

Emergent surgical decompression (fasciectomy, or release of the fascia overlying the affected compartments) performed as quickly and safely as possible is needed for ACS. Fasciectomy

<table>
<thead>
<tr>
<th>Compartments</th>
<th>Contents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arm</td>
<td>Biceps and brachialis muscles, brachial artery, and median nerve</td>
</tr>
<tr>
<td></td>
<td>Triceps muscle and ulnar and radial nerves</td>
</tr>
<tr>
<td>Forearm</td>
<td>Flexor carpi radialis, palmaris longus, pronator teres, flexor carpi ulnaris, and flexor digitorum superficialis muscles</td>
</tr>
<tr>
<td></td>
<td>Flexor digitorum profundus, flexor pollicis longus, pronator quadratus muscles, and anterior interosseous nerve and artery</td>
</tr>
<tr>
<td></td>
<td>Brachioradialis, extensor carpi radialis longus, and extensor carpi radialis brevis muscles</td>
</tr>
<tr>
<td></td>
<td>Extensor digitorum communis, extensor carpi ulnaris, extensor pollicis longus, abductor pollicis longus, extensor pollicis brevis, and supinator muscles, and posterior interosseous nerve</td>
</tr>
<tr>
<td>Hand</td>
<td>Anconeous</td>
</tr>
<tr>
<td></td>
<td>Abductor pollicis brevis, opponens pollicis, and flexor pollicis brevis muscles</td>
</tr>
<tr>
<td></td>
<td>Abductor digiti minimi, flexor digiti minimi, and opponens digiti minimi muscles</td>
</tr>
<tr>
<td></td>
<td>Adductor pollicis muscle (two heads)</td>
</tr>
<tr>
<td></td>
<td>Each is a separate compartment</td>
</tr>
</tbody>
</table>

*Reported isolated involvement in exertional compartment syndrome.

The supinator muscle is not typically a component of the extensor compartment, but decompression can be done through the brachioradialis/extensor carpi radialis longus interval.

Compression of the neurovascular structures by rigid Cleland and Grayson ligaments can lead to skin necrosis and/or loss of the finger.

TABLE 51.4 Compartments of the Upper Extremity

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within the first 8 hours after diagnosis is associated with a lower risk of permanent functional impairment. Release of the epimysium surrounding the muscle may also be necessary. Necrotic tissue should be excised because it may become a nidus for infection or lead to subsequent fibrosis and contracture. Questionable tissue should be left in place for a second look at a later date. Late fibrosis of necrotic muscle can lead to compression of the adjacent nerves and further impair extremity function. Other concomitant procedures may be indicated based on the cause of the compartment syndrome, including fracture reduction and stabilization, vascular repair, and nerve exploration if indicated. Nerve repair or grafting should be performed at the time of definitive wound closure.

Late or delayed diagnosis increases the risk for severe complications, including infection, neurologic injury, need for amputation, and death. In the past, concerns about an increased risk of infection have led some authors to recommend not performing a fasciotomy after 24 hours has elapsed since the onset of symptoms. We do not feel this risk outweighs potential benefits and no longer consider this a contraindication for surgery. Currently, there are more options for wound management and antibiotic therapy. Removal of necrotic muscle can decrease the severity of subsequent muscle fibrosis and joint contracture. Debridement of nonviable muscle improves the environment for the neurovascular structures and may allow early functional reconstruction of the lost muscle(s) by means of tendon transfers or free functional muscle transfers (FFMTs).

In addition, the length of time that elevated pressure sufficient to cause tissue necrosis has been present is often unclear, and some muscle preservation may be facilitated with late fasciotomy. In some cases, good results in children have been reported following fasciotomy as late as 72 hours after the injury (within the acute swelling phase). Dramatic, essentially full, recovery has been reported following compartment syndrome of the lower leg in children after delayed presentation.

**Surgeon’s Preferred Method of Treatment**

The surgical incision for the upper extremity is extensile from the brachium to the carpal tunnel. The extent of the release performed is tailored to the clinical and intraoperative findings. Release of the dorsal forearm and compartments of the hand requires separate incisions when indicated. A separate incision for a dermatotomy of each of the fingers may also be added to prevent skin necrosis and loss of the fingers.}

**Release of the Compartments of the Arm.** The anterior and posterior compartments of the arm can be decompressed through a single median incision. This allows access to the neurovascular structures of the arm, the medial fascia of the biceps and brachialis in the anterior compartment, and the fascia of the triceps. Excision of the medial intermuscular septum will provide additional decompression of both compartments (Figure 51.5). The incision can be easily extended to the elbow crease and incorporated with the incision for decompression of the forearm. This also allows release of the lacertus fibrosus and access to the brachial artery. When there is no anticipated need to evaluate the brachial artery or to decompress the forearm compartments, fasciotomies can be performed through separate anterior and posterior midline incisions to decompress the flexor and extensor compartments, respectively.

**Release of the Compartments of the Forearm.** Several skin incisions have been described for the forearm. Because the surgical incisions are long and extensile, almost any incision can be used to decompress the forearm compartments (Figure 51.6). Because the incisions are left open, we prefer an incision that minimizes exposure of neurovascular structures and can be extended in a proximal direction into the medial arm and in a distal direction into the carpal tunnel (see Figure 51.6, A and B). Once the skin incision has been made, the antebrachial fascia is incised longitudinally from the lacertus fibrosus to the wrist flexion crease. This decompresses the superficial flexor compartment. The deep flexor compartment is most easily and safely exposed through the ulnar side of the forearm. We begin at the mid to distal forearm and identify the interval between the flexor carpi ulnaris and flexor digitorum superficialis. The flexor digitorum profundus and flexor pollicis longus fascias are exposed and released through this interval (Figure 51.7). This is the most important component of this procedure because the
deep flexor compartment is usually the one first and most affected by increased compartmental pressure. Through the same interval, the fascia overlying the pronator quadratus is released.

During the dissection, if the muscles appear pale after release of the fascia, additional release of the epimysium of the pale muscle should be performed. For these muscles, if the epimysium is not released, reperfusion injury will lead to additional swelling within the muscle and further muscle damage.

Clinical evaluation of the remaining tension in the dorsal forearm compartment and/or hand should be done to determine whether additional release of the extensor and hand compartments should be added.

The extensor compartments are released through a midline longitudinal dorsal incision extending from the lateral epicondyle to the distal radioulnar joint. This will allow release of the mobile wad and the extensor compartment (Figure 51.8 and see Figure 51.6, C and D).

**Release of the Compartments of the Hand.** The hand has 10 separate compartments. It is rarely necessary to release all 10 compartments, and intraoperative assessment and/or measurement of compartment pressures should be used to determine the extent of release needed (Figures 51.9 and 51.10).

**Volar Release.** Decompression should start with an extended carpal tunnel release. Carpal tunnel release will usually adequately release the Guyon canal without division of the volar carpal ligament (roof of the Guyon canal) and directly decompress the ulnar neurovascular structures. The carpal tunnel incision can be extended to the second volar web space. In the distal portion of the incision, the volar fascia of the adductor pollicis muscle can be released. Also, the fascia extending from the long finger metacarpal to the palmar fascia (separating the deep radial and ulnar midpalmar spaces) can be released. This will help decompress the volar interosseous muscles. The thenar and hypothenar muscles are decompressed through separate incisions as needed.

**Dorsal Release.** The dorsal interosseous muscles (and volar interosseous muscles) are decompressed through dorsal incisions between the second and third metacarpals and fourth and fifth metacarpals. The first dorsal interosseous muscle is decompressed through an incision placed in the first dorsal web space. The dorsal fascia of the adductor pollicis can also be released through this incision (see Figure 51.9).

**Release of the Fingers.** Tense swollen fingers can result in skin and subcutaneous tissue necrosis. The tight fibers of Cleland and Grayson ligaments can compress and obstruct the digital arteries. Dermatomy of all involved fingers reduces the risk of necrosis of the skin and possible loss of a digit. Dermotomies should be done in the midaxial plane to prevent subsequent contracture. When possible, the dermotomy should be performed on the side that will cause the least amount of scar irritation. The preferred locations for finger and thumb dermatomies are shown in Figure 51.9, A and B.
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FIGURE 51.8  This 5-year-old patient sustained a type 3 100% displaced supracondylar humerus fracture that was treated with closed reduction and pinning. Approximately 6 hours after surgery, the patient had increased pain and analgesia requirements. On examination, the compartments were tense and swollen. He was taken emergently for fasciotomy of the arm and forearm. A, Appearance of the arm after closed reduction and pinning after patient returned to the operating room. B, Fasciotomy of the arm and forearm. C, Final finger and wrist extension after 6 months. D, Final finger flexion after 6 months.

See Case Study 51.1 and Video 51.1 for further information on compartment release of the arm, forearm, and hand.

Postoperative Management

All surgical incisions are left open. We prefer not to use retention sutures. Even if there is minimal swelling of the muscle(s) during the primary release, muscle swelling will usually increase after perfusion has improved. If nerves and arteries are not exposed, a negative-pressure wound dressing (e.g., VAC, Kinetic Concepts, Inc., San Antonio, TX) can be used. We use lower pressures for the negative pressure dressing than in other wounds, usually just enough to maintain good seal on the dressing. If nerves or arteries are exposed, we prefer to use a moist gauze dressing. Dressing changes should be done in the operating room at 24 to 48 hours. Partial delayed primary wound closure can be performed at that time if swelling has decreased and/or to provide coverage over open neurovascular structures. Definitive delayed primary wound closure should be performed only after swelling has decreased. Some cases will require repeat debridement of necrotic tissue. Split-thickness skin grafting for closure is necessary in many patients. Younger patients with high-energy or crush injuries are more likely to require split-thickness skin grafting at 48 hours. In our practice, we prefer to manage the wound until swelling decreases sufficiently to allow delayed primary wound closure if possible, which may require 7 to 10 days. In the hand, only the incision for the carpal tunnel release should be considered for delayed primary wound closure. The other palmar and dorsal incisions as well as the dermotomy incisions will heal quickly by secondary intention. If the skin cannot be closed without tension, split-thickness skin grafting with or without dermal substitutes such as Integra (Integra, Plainsboro, NJ) should be used.

Therapy should be started immediately following surgery to promote maximum active and passive range of motion of the fingers. Splinting should be done for soft tissue stabilization and/or for treatment of other associated injuries. Cessation of therapy during healing of skin grafts may be necessary, but therapy should be resumed as soon as tissue healing allows. Once the soft tissues are adequately healed, nighttime splinting is continued to prevent contractions of the wrist and fingers. Splinting is continued until scars and soft tissues are mature and supple.

Outcomes and Expectations

Outcomes following fasciotomy depend on the duration and severity of the compartment pressure elevation and the resultant extent of muscle necrosis. Early prompt fasciotomy within the first 4 hours usually results in minimal sequelae. Delayed management results in muscle fibrosis and contracture that varies with the extent of muscle necrosis and nerve

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CASE STUDY 51.1  Compartment Syndrome of the Upper Extremity

Surgical Approach to Treatment
All or portions of this approach can be used, depending on the clinical setting. The approach is extensile from the arm to the distal forearm, and separate incisions are required to release the extensor compartment (when indicated). Multiple incisions are used to release the compartments of the hand.

Fasciotomy of the Upper Extremity
Incisions are designed to provide the most coverage of neurovascular structures (at the time of later closure). Linear incisions across flexion creases are avoided. After the skin incision, the antebrachial fascia is released, allowing assessment of the superficial flexor compartment. The interval between the flexor digitorum superficialis and flexor carpi ulnaris is opened to provide access to the deep flexor compartment of the forearm.

After release of the flexor compartments, muscles are inspected and epimysiotomies are performed for tense or dysvascular muscles. Once the flexor compartments are released, the extensor compartment is assessed. If tense, this is released through a dorsal midline incision.

Release at the elbow requires division of the lacertus fibrosus with exploration of the median nerve and brachial artery. Fasciotomies in the hand are performed through a carpal tunnel release, release of the thenar and hypothenar eminences, and dorsal incisions for the release of the interosseous muscles.

Decompression of the fingers may be necessary to prevent digital loss. This is performed through midlateral incisions, avoiding pinching surfaces, including the radial border of the thumb, ulnar borders of the index and long fingers, and radial borders of the ring and small fingers.

The Cleland ligaments are released when necessary to decompress the digital arteries (see Video 51.1).
Other Disorders of the Upper Extremity

CRITICAL POINTS Compartment Syndrome

**Diagnosis**
- Diagnosis is usually a clinical one
- Pain out of proportion to clinical findings
- Pain with passive motion
- Firm, “rock hard” compartment
- Objective findings include elevated compartment pressures and 30 to 40 mm Hg or within 30 mm of diastolic blood pressure or mean arterial pressure

**Management**
- Correct any underlying coagulopathy
- Perform emergent fasciotomy
- Perform vascular repair or reconstruction when indicated
- Treat associated injuries
- Repeat surgical débridement
- Close the wound with delayed primary closure, split-thickness skin graft, or flap
- Perform early reconstruction when indicated with tendon transfer or functional muscle transfer

**Arm Management**
- Anterior and posterior compartments
- Medial incision to decompress both compartments
- Deltoid may require epimysiotomy

**Forearm Management**
- Deep volar, superficial volar, and extensor compartments
- Deep compartment must be released
- Best decompressed through the interval between the flexor carpi ulnaris and the ulnar side of the flexor digitorum superficialis
- Extensor compartment may soften with release of volar compartments but requires separate assessment and release if indicated

**Hand Management**
- Ten separate compartments
- Start with extended carpal tunnel release
- Release additional compartments as necessary

**Finger Management**
- Tense, swollen fingers can result in digital necrosis
- Midlateral dermotomies are performed dorsal to the neurovascular bundles

**Pitfalls**
- Failure to diagnose in a timely manner
- Failure to adequately release compartments
- Inadequate excision of necrotic tissue leading to scarring and fibrosis around nerves

**Results**
- Best results with early decompression
- Chronic cases have less satisfactory results

**Pitfalls**
- Corticosteroids should not be used
- Pain relief is an unreliable indicator

**VOLKMANN CONTRACTURE**

Volkmann ischemic contracture is the end result of prolonged ischemia and associated with irreversible tissue necrosis. Established Volkmann contracture has a much different presentation than ACS. It has a broad clinical spectrum, based on the extent of muscle necrosis and degree of nerve injury. Unlike ACS, patients with an ischemic contracture do not have pain but rather have deformity and dysfunction resulting from the pathologic dystrophy, gangrene, muscle weakness, fracture non-union, and soft tissue tethering associated with skin grafting.

**Outcomes**
- Secondary surgery is usually necessary to improve the outcome of these delayed cases.
- Complications of compartment syndrome and its treatment are common. In a metaanalysis, Kalyani and colleagues reported a complication rate of 42% (18 of 43 patients). Duckworth and associates reported a complication rate of 32% (29 of 99 patients). The most common complication was a neurologic deficit. Other complications included contracture, reflex sympathetic dystrophy, gangrene, muscle weakness, fracture non-union, and soft tissue tethering associated with skin grafting.
ischemic event and subsequent muscle scarring and fibrosis. Nerve dysfunction can occur either from the initial trauma or subsequent ischemic insult or secondary to the fibrosis around the nerves. The muscle fibrosis and neurologic deficits lead to deformity of the joints distal to the site of ischemia. The deformity is progressive over the ensuing weeks to months. In children, an untreated deformity will progress until skeletal maturity because the ischemic muscles are unable to elongate during limb growth. Even when the contractures are treated, the affected extremity is shortened due to the tethering across the physis.

**Classification**

Several classification systems have been described for Volkmann contracture of the forearm. Most are based on the extent of muscle involvement and the severity of the clinical disability. The classification systems can be useful in guiding treatment plans for functional reconstruction. Most authors recognize the

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**FIGURE 51.10** A 2-year-old sustained a crush injury to his left hand, with fractures of the second to fourth metacarpal bases and an open wound at the volar metacarpal heads. He presented with a tense swollen hand. He was taken emergently for fasciotomies of the hand and underwent percutaneous pinning of the displaced metacarpal base fractures. A, Appearance of the hand at presentation. B, Metacarpal base fractures, 100% displaced in the sagittal plane. C, Fasciotomies of the volar hand. D, Fasciotomies of the dorsal hand. E, Final extension at 9 months following injury. F, Final flexion at 9 months following injury.
tremendous variability of the clinical presentations and the subsequent limitations of the classification systems.\textsuperscript{73,83,84,94}

The most commonly used, and our preferred, classification system is that proposed by Tsuge.\textsuperscript{85} Established Volkmann contracture was divided into mild, moderate, and severe types, according to the extent of muscle involvement (Figures 51.11 through 51.13 and Table 51.5). Tsuge's category of severe contractures included cases of moderate tissue necrosis that were exacerbated by fixed joint contractures, a scarred soft tissue envelope, or failed surgeries.

Within each classification type, there is a broad range of clinical presentations. The heterogeneity of presentation makes it difficult to apply a specific treatment based solely on the classification system. The variability also confounds meaningful outcome and comparison studies.

**Treatment**

There is a limited role for the nonoperative treatment of established contracture. There may be some benefit from therapy to stretch and splint mild contractures. In children, splinting is

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**TABLE 51.5 Tsuge Classification for Volkmann Contracture of the Forearm**

<table>
<thead>
<tr>
<th>Type</th>
<th>Findings</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Localized Volkmann contracture, usually involves principally the deep flexor compartment (flexor digitorum profundus of long and ring fingers) most affected Little or no nerve involvement</td>
<td>Nonsurgical treatment Stretching Splinting Must be maintained through skeletal maturity Surgical treatment Selective fractional lengthening Selective flexor origin slide</td>
</tr>
<tr>
<td>Moderate</td>
<td>Most or all of flexor digitorum profundus and flexor pollicis longus Partial flexor digitorum superficialis Neurologic impairment present Sensory in median nerve more than ulnar nerve</td>
<td>Surgical treatment Flexor origin slide Fractional lengthening Shortening of forearm Proximal row carpectomy</td>
</tr>
<tr>
<td>Severe</td>
<td>All of flexor compartment Varying involvement of extensor compartment Severe neurologic defects, including sensory deficits and intrinsic dysfunction</td>
<td>Surgical treatment Tendon transfer Functional free muscle transfer Both may need to be performed Both may require adjunctive flexor slide, infarct excision, and/or contracture release Nerve reconstruction frequently necessary</td>
</tr>
</tbody>
</table>

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CHAPTER 51  Compartment Syndrome and Volkman Ischemic Contracture

FIGURE 51.12  A, Cross-sectional representation of moderate Volkman contracture (according to Tsuge classification) at the midforearm level. B, Patient with Tsuge moderate Volkman contracture. With the wrist in a position of volar flexion, the fingers cannot be brought into full passive extension. C, Finger flexion contracture is worsened by the wrist being brought into a neutral position. D, Patient demonstrates full active finger flexion.

FIGURE 51.13  A, Cross-sectional representation of severe Volkman contracture (according to Tsuge classification) at the midforearm level. B, Patient with severe contracture with extensive intrinsic wasting secondary to neurologic involvement at the forearm.

Operative Treatment

A variety of surgical techniques have been proposed, including bony and soft tissue management (Table 51.6).

Bone Procedures. Skeletal shortening or fusions are frequently performed in conjunction with some of the soft tissue procedures listed in Table 51.6. Shortening procedures include shortening osteotomy of the radius and ulna and proximal row carpectomy.

These procedures have been used to match the skeletal length to the shortened fibrotic muscle. One concern with bone procedures is that the principal contracture is within the flexor compartment. Shortening the forearm indiscriminately lengthens the muscle resting length of both the flexor and extensor muscles, neglecting the predominant involvement of the contracture within the flexor compartment muscles. Shortening procedures raise additional concerns in children because the forearm is already shortened by the initial ischemic insult to the bone and growth plates.

Bony reconstructive procedures are useful for residual problems related to nerve dysfunction or for long-standing contractures not amenable to additional soft tissue release. Options include wrist fusion, trapeziometacarpal joint fusion, or thumb metacarpophalangeal joint fusion. Severe progressive finger deformities can also be managed with arthrodesis in a more...
TABLE 51.6 Surgical Options for Management of Volkmann Ischemic Contracture of the Forearm

<table>
<thead>
<tr>
<th>Operation</th>
<th>Authors</th>
<th>Late Management of Compartment Syndrome; Surgical Options for the Forearm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Proximal row carpentomy | Zancolli, 1979<sup>16</sup>  
Goldner, 1975<sup>16</sup> | Addresses wrist flexion contracture without causing additional scarring in the forearm  
(1) Nonselective shortening of both extensors and flexors  
(2) Limb is already shortened from ischemic insult |
| Diaphyseal shortening | Rolandis, 1985<sup>16</sup>  
Domanasiewicz, 2008<sup>16</sup>  
Pavanini, 1975<sup>16</sup> | Satisfactory correction of contracture  
Does not preclude other procedures  
(1) Nonselective shortening of both extensors and flexors  
(2) Limb is already shortened from ischemic insult  
(3) High nonunion and refracture rate reported |
| Arthrodesis | Botte, 1998<sup>16</sup>  
Goldner, 1975<sup>16</sup> | Wrist: Maintains wrist in physiologic position; if additional tendon transfers are needed, if wrist is fused, one less donor muscle is needed and wrist extensor muscles are available as donors for finger function  
PIP: Fingers maintained in a more functional position, may be only option to correct intrinsic imbalance  
Wrist:  
(1) Loss of tenodesis effect of wrist position  
(2) Decreased grip strength  
(3) Loss of motion  
PPIP:  
(1) Loss of finger flexion  
(2) Resultant loss of grip strength |
| Soft Tissue |         |                                                               |
| Fractional or tendon “Z”-lengthening | Goldner, 1975<sup>16</sup> | Straightforward surgical release of flexion contracture  
(1) Loss of active flexion  
(2) More disruption of muscle resting length and loss of flexion strength |
| Infarct excision with tendon reconstruction/transfer | Seddon, 1964<sup>17</sup>, 1965<sup>17</sup>  
Tsuge, 1975<sup>16</sup> | Tsuge advocated infarct excision for mild contractures with involvement of only one or two fingers  
(1) Loss of finger flexion contracture  
(2) Decrease of scar excision replaced with more scar tissue later |
| Flexor-pronator slide | Page, 1923<sup>16</sup>  
Scaglietti, 1957<sup>16</sup>  
Gosset, 1956<sup>16</sup>  
Sharma, 2012<sup>16</sup> | Allows greatest correction of flexion contracture with least impact on muscle resting length  
(1) Will not fully correct finger flexion contracture that is not passively correctable in maximal wrist flexion  
(2) Wide surgical dissection with potential injury to CIA/PIA |
| Functional free muscle transfer | Zuker, 1989<sup>16</sup>  
Zuker, 2007<sup>16</sup>  
Manktelow, 1979<sup>16</sup>  
Manktelow, 1989<sup>16</sup>  
Dai, 1989<sup>16</sup> | Only procedure that can restore function for severe contracture according to Tsuge classification  
(1) Difficult microsurgical procedure requiring an experienced team  
(2) Failure can only be remedied with a second functional free muscle transfer |

Functional position. These procedures are ideally done after skeletal maturity but may need to be performed earlier if there is progressive deformity. In these cases, we attempt to fuse the joint and preserve the growth plate (i.e., chondrodesis).

**Soft Tissue Procedures.** Soft tissue procedures include excision of the infarcted muscle, fractional or “Z”-lengthening of the affected muscles, muscle sliding operations (flexor origin muscle slide), neurolysis, tendon transfers, and functional free-tissue transfers, as well as combinations of these procedures.<sup>16,17,22,34,43,50,52,61,64,72-75,85</sup> Excision of scarred fibrotic nerves without distal function followed by nerve grafting has been described to try and establish some protective sensation in the hand.<sup>44</sup> Fixed contractures of the joints can be addressed with soft tissue release, including capsulotomy and collateral ligament resection or excision, depending on the joints involved.

**AUTHORS’ PREFERRED METHODS OF TREATMENT**

Our preferred methods of treatment depend on the general classification of severity of contracture, individualized to the patient presentation.

**Mild (Localised) Type (Deep Flexor Compartment Without Neurologic Deficit).** For mild contractures that have failed to respond to nonsurgical management, our preferred treatment is a muscle sliding operation initially described by Page and subsequently endorsed by several others.<sup>16,22,34,43,50,52,61,64,72-75,85</sup> We have found this procedure effective as long as good active flexor function is present. We do not combine this procedure with infarct excision, nor have we found it necessary to release the distal insertion of the pronator teres to correct pronation contracture.<sup>16,22</sup>

A limited flexor slide may be done for mild deformity, affecting only a portion of the flexor digitorum profundus. Because the flexor digitorum profundus originates solely from the ulna, the flexor pronator mass does not have to be released from the medial epicondyle and the ulnar nerve does not have to be transposed. This limited approach reduces potential scarring and vascular compromise to the remaining muscles and nerves in the flexor compartment.

**Moderate Type (Deep and Superficial Flexor Compartment With Neurologic Deficit).** For moderate deformity, we prefer a flexor muscle origin slide to correct the tightness of the flexors, provided that there is still adequate remaining strength in the
flexors. Because neurologic impairment is characteristic of the moderate injury, we combine the flexor slide with neurolysis of both the median and ulnar nerves. A separate incision to release the carpal tunnel may also be needed. Depending on the functional deficits, tendon transfer can be combined with flexor origin slide, either as a staged or simultaneous procedure.

Reconstruction of Thumb Function. Our preferred transfer for thumb flexion is to transfer the brachioradialis or extensor carpi radialis longus to the flexor pollicis longus. The extensor indicis proprius can be used for thumb opposition at a later date.

Reconstruction of Finger Flexion. When the finger flexors are very weak or absent, an FFMT may produce a better functional result than a tendon transfer. However, if an FFMT is not an option, tendon transfers may be considered depending on the availability of donors. The best option is transfer of the extensor carpi radialis longus to the flexor digitorum profundus because this transfer is synergistic and easy to relearn. Other lesser options include the biceps brachii elongated with graft, the brachioradialis, the extensor carpi ulnaris, and the extensor indicis proprius. Many of these secondary options do not have sufficient excursion to match the flexor muscles, but in the absence of other options, they can provide some improvement in grasp. Lastly, if there is minimal involvement of the flexor digitorum superficialis muscle, this muscle can be used as a donor to the flexor digitorum profundus.

Nerve Reconstruction. When sensory impairment is severe and there has been no recovery, the nerve should be carefully evaluated at surgery. A densely scarred atrophic nerve or avascular nerve requires resection back to fascicles that appear healthy followed by sural nerve grafting to restore protective sensation to the hand (Figure 51.14).

Severe Type (Superficial and Deep Flexor Compartments, Extensor Compartments, and Severe Neurologic Deficits). Severe contractures are best treated with FFMTs. The donor vessels are usually either the radial or anterior interosseous artery for an end-to-end anastomosis or the brachial artery for an end-to-side procedure. The donor motor nerve is the anterior interosseous nerve, which should be resected back to fascicles that appear healthy. Our preference for the donor muscle is the gracilis. For severe contractures with extensive involvement of both flexor and extensor compartments, a double free muscle transfer should be considered. Tendon transfer, nerve graft reconstruction, and late osseous reconstructive procedures may improve final functional outcomes.

Surgical Techniques

Flexor Pronator Slide. Both volar and ulnar incisions have been described for the surgical approach for a flexor pronator slide. We favor the incision and technique initially described by Page with minor modifications (Figure 51.15). The surgical incision begins on the medial distal arm and continues along the ulnar border of the forearm all the way to the wrist. The ulnar nerve is identified and mobilized for several centimeters proximal and distal to the medial epicondy, including proximal release at the arcade of Struthers. Approximately 4 to 6 cm of intermuscular septum between the brachialis and triceps is excised to prevent kinking of the ulnar nerve after transposition. The flexor pronator mass is elevated off of the medial epicondy, taking care to preserve the medial collateral ligament and elbow joint capsule. Inadvertent disruption of the joint capsule is repaired. The origins of the flexor carpi ulnaris, flexor digitorum profundus, and flexor digitorum superficialis are mobilized off of the ulna and interosseous membrane. The dissection is carried out above the periosteum toward the radius. The common interosseous artery arises as a branch of the ulnar artery and must be protected. This artery crosses the flexor digitorum profundus, where it bifurcates into the anterior and posterior interosseous arteries (see Figure 51.15, C). The posterior interosseous artery enters the posterior compartment at the proximal edge of the interosseous membrane. Because the posterior interosseous artery is the dominant blood supply to the extensor compartment, protection of this branch is mandatory.

Continuing the dissection toward the radius, the origin of the flexor pollicis longus is released from proximal to distal. Special attention is needed in this area to protect the anterior interosseous nerve. Throughout the procedure, the wrist and fingers are manipulated to check whether the contracture is improving and to help determine whether there are still areas of tightness within the muscle origin. The dissection must
often be carried down to the level of the wrist to release adhesions between the flexor tendons and pronator quadratus before full correction is achieved. If necessary, the carpal tunnel should be opened and tendon adhesions released in this area. It has been our experience that the lacertus fibrosus and superficial fascia around the antecubital fossa are often tight and are contributing to the residual elbow flexion contracture. The fascia can also tether the superficial flexors of the wrist and fingers, contributing to incomplete correction of the wrist and fingers with the muscle sliding only. Release of this fascia helps correct the deformity and also prevents median nerve compression between the heads of the pronator teres. Slight undercorrection, which can be addressed by postoperative splinting and rehabilitation, may decrease the reduction in muscle power resulting from the muscle slide. When a pronation contracture is present and has not been corrected by the release of the flexor-pronator origin, we release the pronator quadratus from the distal ulna. Even with a complete release of both the pronators and the volar distal radioulnar joint capsule, complete correction of the pronation deformity may not be possible due to fibrosis and contracture of the interosseous membrane. At the completion of the muscle slide, the ulnar nerve is transposed to an anterior subcutaneous position. The hand is cast in a position of forearm supination with the wrist and fingers in full extension. Immobilization is continued for a period of 6 weeks to allow the flexor pronator to heal adequately to its new origin (Figure 51.16) and Case Study 51.2.

**Restoration of Extrinsic Flexor Function.** For moderate or severe contractures, a muscle slide may leave adequate flexor muscle function. Commonly, flexor strength and excursion are weakened and can benefit from flexor reconstruction. The options for tendon transfer depend on the extent of muscle necrosis in the forearm. Donor muscles from the extensor compartment can be used to reconstruct finger flexion and thumb flexion and opposition. Our preferred transfers are transfer of the extensor carpi radialis longus through the interosseous membrane to the flexor digitorum profundus, transfer of the brachioradialis to the flexor pollicis longus, and transfer of the extensor indicis proprius to restore opposition of the thumb.

Secondary options may be available in the flexor compartment when there is involvement of the deep flexor compartment only. A transfer of the flexor digitorum superficialis to the

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**FIGURE 51.16** A, Diagram of surgical incision for flexor origin slide. B, Schematic diagram of deeper layer of dissection for flexor origin slide. C, Ulnar incision with ulnar nerve identified and mobilized. Flexor pronator mass elevated from the medial epicondyle with preservation of the medial collateral ligament and elbow joint capsule. D, Dissection proceeds in a radial direction with protection of the interosseous arteries. E, Complete release heralded by full finger wrist extension with the wrist held in extension. **FDS**, flexor carpi radialis; **FDS**, flexor digitorum superficialis; **PT**, palmaris longus; **PT**, pronator teres.
CHAPTER 51  Compartment Syndrome and Volkmann Ischemic Contracture

CASE STUDY 51.2  Moderate Volkmann Contracture

A 15-year-old right hand dominant football player sustained a minimally displaced but angulated left both-bone forearm fracture. The fracture was treated in a long-arm cast with extreme molding at the midforearm. When the cast was removed at 6 weeks, the patient was noted to have a flexion contracture of the thumb and fingers. The fingers could be extended only with maximum wrist flexion. He could actively flex his fingers into a full fist. The patient was seen in the hand surgery department at 9 months after injury (eFigure 51.1).

At 9 months following injury, the patient underwent a flexor origin slide. A cast was applied with the wrist, fingers, and thumb in maximum extension for 6 weeks after surgery. At the time of cast removal, the patient was seen in therapy. A nighttime extension splint was made and recommended for use for 1 year after surgery. (The patient reported that he used this splint only for a few weeks.)

At 10 years of follow-up, the patient is working in light construction and builds motorcycles. He feels happy with the outcome and grip strength, although he reports occasional fatigue in the forearm with prolonged use (eFigure 51.2 and Video 51.2).

eFIGURE 51.1 Moderate Volkmann contracture. A and B, Presentation at 9 months following injury. C, Intraoperative passive extension.

eFIGURE 51.2 Ten-year follow-up after flexor origin slide. A, Full finger extension with wrist in full extension; slight thumb interphalangeal flexion. B, Full thumb and finger flexion with grip strength 85% of contralateral side. C, Mild shortening of the affected forearm.
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**CRITICAL POINTS Flexor Pronator Slide for Volkmann Contracture**

**Preoperative Planning**
- Indications include mild and moderate contractures of the forearm muscles, and preoperative magnetic resonance angiogram or arteriogram when indicated for previous vascular injury or by an abnormal vascular examination.
- Bipolar curettage is useful for this procedure.

**Surgical Technique**
- Incision from ulnar side from middle third to distal third junction of the arm, following the course of the ulnar nerve and continuing along the border of the forearm to the wrist flexion crease (extensile ulnar approach).
- Mobilize and transpose the ulnar nerve.
- Release the flexor-pronator origin off of the medial epicondyle, preserving the medial collateral ligament and joint capsule.
- Release the origins of the flexor carpi ulnaris, flexor digitorum profundus, flexor digitorum superficialis, and flexor pollicis longus from the ulna and intermuscular septum, working from proximal to distal and ulnar to radial.
- Identify and protect the common interosseous artery and its division into the anterior and posterior interosseous arteries.
- Frequently passively extend the wrist and fingers to identify structures that remain tight; the release is complete when full passive wrist and finger extension is achieved.
- A separate release of the carpal tunnel and release of tendon adhesions in this area may be necessary if complete slide fails to achieve full extension.
- Residual pronation contracture is addressed by release of the pronator quadratus from its ulnar origin and release of the volar dorsal radioulnar joint capsule; complete correction may not be possible due to interosseous membrane tightness.
- A large drain is placed below the flexor pronator mass (it is removed on the first postoperative day).
- No fascial closure; skin and subcutaneous tissue only are closed.

**Postoperative Care**
- Immobilization in long-arm cast with the elbow at 90 degrees and the forearm in supination; the wrist and fingers are in full extension, and the thumb is in abduction and extension; the initial cast should be bivalved if swelling is a concern.
- Duration of immobilization is 2 to 3 weeks in a long-arm cast; convert to short-arm cast with wrist and finger extension and thumb abduction-extension for an additional 3 to 4 weeks (full 6 weeks postoperatively).
- Rehabilitation: Begin elbow motion and nerve gliding at time of conversion from long-arm cast to short-arm cast; begin wrist and finger range-of-motion exercises with therapy guidance at time of cast removal; strengthening may begin as tolerated starting at 6 weeks; custom thermoplast orthosis at night until skeletal maturity.
- Activities: The patient may return to sports when adequate strength and motion allow participation in the sport of choice; we encourage swimming activities to develop strength and motion.

**CRITICAL POINTS Tendon Transfer for Finger and Thumb Flexion**

**Preoperative Planning**
- Careful preoperative clinical assessment of available donor muscle strength.

**Surgical Technique**
- Extensor carpi radialis longus to flexor digitorum profundus, brachioradialis to flexor pollicis longus, or extensor indicis proprius to radial side of the thumb metacarpal.
- The extensor carpi radialis longus is harvested from the radial base of the index metacarpal through a longitudinal incision.
- The harvest incision of the extensor carpi radialis longus is extended or a separate proximal incision is made at the junction of the middle third and distal third of the forearm to create a tunnel through the interosseous membrane.
- The brachioradialis is exposed through the same incision used to expose the extensor carpi radialis longus proximally. The brachioradialis must be freed from the forearm fascial attachments to improve excursion.
- The extensor indicis proprius is exposed through a separate incision at the metacarpophalangeal and mobilized proximally.
- A volar incision is made at the distal third of the volar forearm to identify and mobilize the flexor digitorum profundus tendons. The flexor pollicis longus is identified through the same incision. The flexor digitorum superficialis tendons are excised. The flexor digitorum profundus tendons are sutured together as a single tendon unit.
- The extensor carpi radialis longus is transferred through the interosseous membrane; the opening created in the interosseous membrane must be sufficient in size to prevent constriction of the transferred muscle-tendon unit.
- The extensor carpi radialis longus is woven into the flexor digitorum profundus tendons using a weave technique.
- The tenorrhaphy is placed under sufficient tension to restore the resting cascade of the fingers but still allow full passive extension of the fingers with the wrist in neutral position.
- The brachioradialis to flexor pollicis longus transfer is completed in a similar manner.
- The extensor indicis proprius is mobilized proximally, then passed through a subcutaneous tunnel around the ulnar side of the wrist. An ulnar-sided incision around the level of the pisiform is made to retrieve the extensor indicis proprius and to create a subcutaneous tunnel from the ulnar wrist to the radial side of the thumb metacarpal neck.
- The extensor indicis proprius is secured to the radial side of the thumb metacarpal neck with the thumb in a position of abduction and pronation, as described by Burkhalter.12

**Postoperative Care**
- Immobilization is in a splint or cast: Dorsal block with wrist at 30 degrees of flexion, metacarpophalangeal at 70 degrees of flexion, thumb in abduction and pronation with interphalangeal joint in neutral.
- The duration of immobilization is 4 weeks, during which time the patient is instructed in passive range-of-motion exercises.
- At 4 weeks, a thermoplast splint may be worn full time and place-and-hold exercises may begin.
- At 6 weeks, the transition is made to nighttime use of the splint only.
- Rehabilitation with place-and-hold exercises beginning at 4 weeks; passive extension after 6 weeks.
- Activities: The patient may return to sports when there is adequate strength and motion to participate in the sport of choice; minimum of 6 months after tendon transfer.

flexor digitorum profundus will provide the patient with good flexor strength and sufficient excursion to achieve functional grasp. Also, the flexor carpi radialis and flexor carpi ulnaris may be spared and available as donor muscles for transfers. Neither of these muscles has excursion matching that of the flexor digitorum muscles.

When the extensor compartment has been compromised by the ischemic event, the muscles are not available for transfer. For these patients, the only option for functional reconstruction remains an FFMT.
Restoration of Extrinsic Flexor Function: Free Functional Muscle Transfer. Functional reconstruction following Volkmann ischemic contracture was the first clinical application of FFMFT. Several different donor muscles have been used to restore finger flexion, including the pectoralis major (lateral portion), medial gastrocnemius, latissimus dorsi, and gracilis. When using the latissimus dorsi muscle, we place the caudal portion of the muscle as the origin on the medial epicondyle and use the tendinous portion to attach to the profundus tendons. The gracilis (and also latissimus) has independent neuromuscular territories that can be separated and used to restore independent thumb and finger flexion. If the latissimus dorsi is being used for functional reconstruction and independent neuromuscular territories are needed, the latissimus cannot be reversed. The tendinous insertion of the latissimus dorsi should become the neotendon of the forearm flexors, and the muscle should be separated distally to reconstruct finger and thumb flexion independently.

Our preferred muscle for reconstruction is the gracilis, due to its size, match, excursion, and power. Specific points related to FFMFT in the setting of Volkmann ischemic contracture are highlighted here.

Prior to FFMFT, procedures may be necessary to prepare the recipient site. Excision of the infarcted muscle is not necessary.
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because the tendons are released distal to the area of muscle infarct. However, fixed contractures and tendon adhesions distal to the planned tenorrhaphy site need to be addressed.

The median and ulnar nerves (if clinically impaired) are explored. Areas of necrotic or constricted nerve are excised and grafted using a sural nerve graft. If both the median and ulnar nerves are affected over a long distance and there is not enough sural nerve graft, the median nerve should have priority of reconstruction using autogenous graft. Ulnar nerve reconstruction can be performed or augmented with nerve allograft if necessary. We prioritize median nerve function because of the importance of radial-sided sensation for pinch as well as the higher likelihood of recovery in both sensory and motor function. In our experience, the median nerve is more frequently and more severely affected, likely due to its anatomic location.

At the time of FFMT, surgical incisions are planned to provide the best soft tissue coverage over the transferred muscle and tenorrhaphy site. The recipient site should be prepared before the donor muscle is harvested. A two-team approach is frequently used, but division of the donor tendon insertion should not be completed until the recipient vessels and motor nerve are identified. This is particularly important when using an FFMT for reconstruction in patients with Volkmann contracture. In these cases, it is often necessary to obtain intraoperative frozen sections to identify healthy fascicles of the recipient motor nerve. In addition, the vascular anastomosis should be performed proximal to the ischemic tissue bed. Even though an adequate arterial inflow may be found within the reconstruction site, the venous outflow is unreliable. The vascular pedicle and recipient nerve should be mobilized as much as possible to obtain sufficient length for proper positioning of the transplanted muscle.

The vascular anastomosis is completed first, and the muscle is allowed to perfuse for 10 to 15 minutes until the venous outflow does not appear overly dark. The anastomosis is completed as quickly as possible to minimize permanent ischemic changes to the muscle. Irreversible muscle loss increases with time in a nonlinear relationship. It is our experience that if the muscle ischemia time exceeds 2 hours, the muscle will likely survive but the function will never return. If the cause of the prolonged ischemia time can be identified (usually, compromised venous outflow leading to reperfusion problems) and rectified (e.g., vein grafting to different recipient vessels), the surgeon can consider harvesting a different donor muscle at the same procedure.

The muscle is secured to its new origin site on the medial epicondyle using nonabsorbable suture. The origin may be secured to the periosteum and surrounding fascia. Bone tunnels or suture anchors can also be used. The muscle neoorigin should be spread out as much as possible. The tendons of the flexor digitorum superficialis are excised from the musculotendinous junction to the wrist flexion crease. The flexor pollicis longus tendon is transected distal to its musculotendinous junction. Traction is applied along the tendon to ensure proper tendon gliding. Flexor pollicis longus function is reconstructed separately using a tendon transfer if available (either the brachioradialis or extensor carpi radialis longus). If these muscles are not functional, independent flexor pollicis longus function can be restored by using the separate neuromuscular territories of the gracilis muscle to allow independent finger and thumb flexion. The transferred muscle is stretched to restore its resting length, and the site for the distal tenorrhaphy is marked with the wrist and fingers in full extension. The neurotomy is completed last; its site is placed as close as possible to the transplanted muscle. Wound closure should be performed with attention to avoiding compression of the vascular pedicles. Tight skin closures should be avoided at all costs because a compartment-like syndrome or venous outflow obstruction can occur when the reperfused muscle swells following the surgery.

Transfer of Gracilis Muscle. The gracilis muscle is usually harvested with the patient supine in a frogleg position. Either ipsilateral or contralateral gracilis can be used for finger flexor reconstruction because the recipient vascular pedicle and obturator nerve have sufficient length to accommodate the distance to the recipient site. Technique highlights are listed in the critical points box.

Outcomes

Outcomes following Volkmann contracture in the upper extremity are difficult to assess. Studies are limited by the small numbers of patients, the great variability in the initial presentations, the varied surgical techniques used, and the difficulty in tracking patients over the long term as they grow to skeletal maturity. Ullert and Hovius attempted to provide some information regarding outcomes. They found that all patients who had developed the contracture during childhood had a relatively shortened extremity. Substantial improvements in hand function were noted in patients who underwent FFMT. Tendon lengthening alone often resulted in recurrence of contracture. Finally, in patients who had sufficient remaining muscle, procedures that combined infarct excision, tenolysis, neurolysis, and tendon transfer produced good hand function.

FIGURE 51.17 Schematic drawing of gracilis muscle used for free functional muscle transfer for reconstruction of finger flexion.
CRITICAL POINTS Free Functional Muscle Transfer for Finger Flexion

Preoperative Planning
- Indications are no active finger flexion and no available tendon transfers
- Microsurgical instrumentation and magnification are required

Surgical Technique
Preparation of the Donor Site
- Extensive surgical incision along the forearm
- Excision of necrotic and/or fibrotic muscle may not be necessary after division of the flexor tendons

- Neurolysis of the median and ulnar nerves
- Nerve reconstruction when indicated using sural nerve autograft
- Identification and preparation of the recipient nerve: recipient nerve options are the anterior interosseous nerve and the median nerve; recipient artery and vein options are the anterior interosseous artery (end-to-end), radial artery (end-to-end), ulnar artery (end-to-end), and brachial artery (end-to-side)

Harvest of the Gracilis
- Frog position of the leg; a sandbag can be used to help positioning
- Mark the axis of dissection of the gracilis from the pubic tubercle to the tendon insertion of the pes anserinus
- A distal incision is made and the tendon of the gracilis is identified between the sartorius and the semitendinosus
- Tension applied to the distal tendon will help identify the axis of the muscle
- A skin paddle is incorporated into the surgical incision
- Dissection is carried down to the fascia; the gracilis is harvested within its fascial sheath (Figure 51.18); a wide harvest of the gracilis is taken anterior to the gracilis
- The resting length of the gracilis is marked with the hip in full abduction and the knee in full extension; marking sutures are placed at 5-cm intervals
- Dissection is best completed from distal to proximal and from posterior to anterior
- The tendon is released distally
- The muscle origin is divided after the neurovascular pedicle is mobilized and isolated

- The dominant vascular pedicle enters the muscle proximally on the anterior deep surface; the vascular pedicle arises from the profunda femoral artery; the obturator nerve enters the muscle proximal to the dominant pedicle; the nerve is stimulated to ensure that it produces a muscle contraction
- The nerve is divided and the vascular pedicle is ligated only after the recipient site is ready

Muscle Transfer
- Arterial and venous anastomosis first; allow muscle to perfuse for 10 minutes
- Establish and secure new muscle origin at the medial epicondyle; reset the muscle resting length; the resting length is reset with the wrist and fingers in full extension
- Use a weave to secure the gracilis tendon to the flexor digitorum profundus and flexor pollicis longus; although the resting length is set with the wrist and fingers in extension, once that has been established, the repair is performed with the wrist and fingers in flexion to minimize tension at the repair site; flexor pollicis longus function is reconstructed with a tendon transfer (extensor carpi radialis longus or brachioradialis); alternatively, the separate neuromuscular territory of the gracilis can be used to create separate functional reconstruction for thumb flexion: this requires two motor donor nerves
- Neuroraphy is completed last; the neuroraphy is completed close to the transplanted muscle

Postoperative Care
- Immobilization is with a long-arm splint followed by a long-arm cast, with the elbow at 90 degrees of flexion, wrist at 30 degrees of flexion, and metacarpophalangeals at 90 degrees of flexion; the thumb is in palmar abduction and interphalangeal flexion
- Duration of immobilization is 4 to 6 weeks
- Rehabilitation: After cast removal, gentle passive range of motion; more firm passive motion may start at 3 months; muscle stimulation may also start at 3 months; light and progressive strengthening after onset of muscle reinnervation
- Activities: The patient should not participate in sports until at least 1 year after reinnervation, and then only when he or she has adequate strength and motion to participate in sport of choice

Outcomes of Flexor Origin Slide. Since the original description of this procedure by Page in 1923, there have been few reports about it.27,22,73,89 The most detailed report of outcomes for this procedure was published by Sharma and colleagues, who reported on patients with moderate Vollmann contracture with no fixed joint contractures, noting improvements in dexterity, sensibility, ability to flex and extend the fingers, and grip strength. In 15 of 19 patients, dexterity scores were in the good range. In 14 of 19, grip strength was in the good range and reached 20 to 87% of that of the contralateral hand (average, 75%). The authors attribute their good outcomes to careful patient selection and emphasis on postoperative therapy.73

In our experience, substantial functional improvement of mild and moderate congenital contractures can result following flexor muscle slide and nerve reconstruction when indicated. Normal function is not anticipated, but a hand with protective sensation and functional grasp can often be achieved.

A flexor origin slide for severe contractures can improve passive finger and wrist motion and is useful as a first stage of treatment before functional reconstruction with tendon transfer or FFMT (Figure 51.19 and Case Study 51.3). All of these procedures have better outcomes in patients with preserved hand intrinsic function.

Outcomes of Free Functional Muscle Transfer. Since the first case report of an FFMT for reconstruction of finger flexion, several other case reports and small series have been reported. Zuker and colleagues reported on a series of FFMTs performed in 1991 for seven patients using a gracilis donor muscle. All patients achieved less than 2 cm of pulp to palm grip. Grip strength was approximately 25% of the contralateral side. All patients achieved a functional nondominant hand.77 In 2011, they reported on a modification in their technique to provide independent thumb flexion by separating the fascicles of the obturator nerve and using two donor nerves, providing separate innervation to the neuromuscular territories of the gracilis muscle.80 Krimmer and associates used the gracilis muscle in 15 patients with the primary indication being Vollmann contracture. Reinnervation occurred in 13 of 15 muscles. Ten patients achieved full active motion.80 Liu and coworkers used the medial gastrocnemius muscle in 20 patients, reporting electromyographic activity at 6 to 20 months. Functional outcomes were reported as satisfactory, but the clinical pictures suggest
CASE STUDY 51.3 Severe Volkmann Contracture

A 5-year-old boy developed a severe Volkmann contracture after a supracondylar humerus fracture. One year later the patient underwent a flexor origin slide to reduce the contracture at the wrist and fingers. At 2 years following injury, he underwent a free functional muscle transfer to restore finger and thumb flexion. Nerve grafting of the median nerve was performed at the same time (see Figure 51.19 and Video 51.3).
incomplete finger flexion, likely related to the short excursion of the muscle.33

**Restoration of Intrinsic Function After Volkmann Contracture of the Forearm**

**Nerve Dysfunction Associated With Forearm Compartment Syndrome.** Intrinsic dysfunction in the setting of a Volkmann ischemic contracture of the forearm results from nerve ischemia in the forearm. Intrinsic dysfunction remains an extremely difficult problem to address both operatively and nonoperatively. Nerve reconstruction is performed when there is absent sensory and motor function or a clearly necrotic nerve is identified at the time of exploration. Some recovery of sensation can be expected. Motor recovery is less predictable. There are few available tendon transfers for functional restoration because donor tendons are often necessary for extrinsic flexor reconstruction. If protective sensation is not improved with neurolysis or nerve reconstruction, nerve transfer from the dorsal sensory branch of the radial nerve to the ulnar digital nerve of the thumb and radial digital nerve of the index finger should be considered for restoration of sensate pinch. Also, if ulnar nerve function is intact, transfer of the common digital nerve to the fourth web space to the radial digital nerve of the index finger and ulnar digital nerve of the thumb is another option for sensory restoration for pinch.

**Volkmann Contracture of the Hand**

Volkmann contracture isolated to the hand is much less common than contracture in the forearm. Little is written about this problem, and treatment options are limited. Volkmann contracture of the hand commonly involves contracture of the first web space with the inability to open the thumb for grasp. The fingers assume variable degrees of the intrinsic-plus posture, with metacarpophalangeal joint flexion and proximal interphalangeal joint extension secondary to ischemia within the interosseous muscles. In combination with the thumb deformity, the hand cannot be opened sufficiently to grasp even medium-sized objects (Figure 51.20).

The first web space contracture is addressed with excision of the first dorsal interosseous muscle and the fibrotic portion of the adductor pollicis. Options to maintain the web space include placement of a bone block from the iliac crest between the thumb and index metacarpal shafts, or fusion of the
FIGURE 51.19 This 5-year-old boy developed a Volkmann contracture after sustaining a supracondylar humerus fracture. One year after his initial injury, he underwent a flexor origin slide to reduce wrist and finger flexion contractures. One year later he underwent a free functional muscle transfer of the gracilis for restoration of finger flexion. The extensor carpi radialis longus was transferred to restore flexor pollicis longus function. An opponensplasty was performed 9 months later using the extensor indicis proprius. A, Preoperative extension 1 year after flexor origin slide. B, Preoperative flexion. C, Necrotic flexor muscles have been partially resected. The flexor digitorum profundus tendons are sutured together distally. The flexor pollicis longus tendon has been independently separated. The median nerve has been dissected. The anterior interosseous nerve (AIN) branch has been isolated for neurorrhaphy to the nerve to the gracilis. D, Insetting of functional free gracilis muscle. The median nerve has been reconstructed with a sural nerve graft over a 7-cm segment. E, Finger and wrist extension at 1 year after free gracilis transfer and median nerve reconstruction. F, Finger and thumb flexion at 1 year after his last surgery. G, Opposition to small finger at 1 year. See Video 51.3. AIN, Anterior interosseous nerve; FDP, flexor digitorum profundus; FPL, flexor pollicis longus.

The trapeziometacarpal joint. If skin is deficient, these procedures may require augmentation with a four-flap “Z”-plasty, pedicled rotational flap, or even free tissue transfer. Our preferred technique would be dependent upon the extent of contracture. The choice of free tissue transfer would depend on the body habitus of the patient, but a posterior interosseous forearm rotational flap or a free lateral arm flap would be our first choice. Sacrifice of the index finger with a ray resection may be a good alternative for some patients, particularly those with substantial dysfunction of the index finger. This provides an adequate web space for grasp, removes the need for tissue transfer, and allows the resected index metacarpal to be used as a bone block between the first and third metacarpals to maintain the web space.

Intrinsic tightness of the fingers clinically presents as a metacarpophalangeal joint flexion and interphalangeal joint extension. Intrinsic tightness can sometimes be distinguished from joint contracture by performing an intrinsic tightness test.

Milder cases of intrinsic contracture can be addressed with excision of portions of the oblique and lateral bands at the level of the distal third of the proximal phalanx. Fixed joint contractures are addressed with concomitant capsulectomy and collateral ligament releases as needed. Intrinsic muscle sliding procedures have been described but have limited indications...
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Complications and Volkmann Ischemic Contracture

A 38-year-old diabetic presented to the orthopedic department more than 24 hours after lying on his hand for an unknown period of time during a diabetic coma. A, Appearance of the hand at time of presentation. B, Fasciotomy performed immediately after presentation, volar incisions. C, Fasciotomy performed immediately after presentation, dorsal incisions. D, Volkmann contracture of the hand developed despite release. E, First web space release with excision of fibrotic muscles and release of the intrinsics to the index and long fingers at the level of the metacarpophalangeal joint.

CRITICAL POINTS Late Management of Volkmann Contracture

Pitfalls and Their Prevention

- Distinguishing Volkmann contracture from pseudo–Volkmann contracture is important:
  - Historical features may favor one diagnosis over the other; clinical findings of nerve involvement are less likely in pseudo–Volkmann contracture; magnetic resonance imaging will not show ischemic muscle changes in pseudo–Volkmann contracture.
- Failure to address severe muscle necrosis with wide excision of necrotic or fibrotic muscle may result in nerve ischemia and contracture: When necrotic and/or fibrotic muscle is identified, early excision of it and reconstruction of the compartment with healthy soft tissue will limit scarring and ischemia of the nerves.
- Progressive or recurrent deformity during growth: regardless of the treatment provided this will occur. Night splinting should be continued through skeletal maturity.
- Flexor-pronator slide: (1) Incomplete correction at the time of surgery will not improve postoperatively with therapy; obtain maximum correction during surgery; (2) injury to the common interosseous artery (which supplies the dorsal forearm compartment) can be prevented by careful dissection, especially in the proximal third of the forearm.
- Free functional muscle transfer: (1) With prolonged surgical ischemia (>2 hours), even if the muscle survives, muscle function will likely be poor; consider a second muscle transfer; (2) therapy that is too aggressive during the early stage of healing can lead to rupture of muscle fibers; use gentle rehabilitation for the first 6 months following FFMT.

Common Complications

- If the deformity recurs after a soft tissue procedure, arthrodesis of the wrist may be necessary and may need to be combined with proximal row carpectomy.
- Inadequate active range of motion or strength after flexor origin slide may need to be managed with FFMT.

Complications

Complications are managed with FFMT:

- Complications of Functional Free Muscle Transfer: FFMT is a complex procedure associated with multiple potential complications, which can be divided into acute and long-term complications and also into those at donor sites and those at recipient sites.
- Acute complications at the recipient site include flap loss, partial flap loss (either skin paddle or partial muscle loss), and ischemia of the nerves.

With severe contractures. These muscle sliding procedures have better outcomes with some underlying intrinsic muscle function preservation. Tenolysis of the flexor and extensor tendons and release of the intrinsic muscles at their insertion may be necessary to treat severe contractures. Despite these extensive releases, overcorrection of the contracture is not typically seen.

There is a limited role for tendon transfers in treatment of these deformities. Tendon transfers such as the flexor digitorum superficialis (ring finger) or extensor indicis proprius for reconstruction of opposition may improve function after addressing the contracture.
hematoma, and infection. **Long-term complications at the recipient site** include scarring and tendon adhesions requiring revision surgery, attenuation at the tendon repair site, and inadequate recovery of muscle power for the desired level of function. In pediatric patients, the bone growth may be more rapid than the transferred muscle growth, which can lead to joint contracture.

**Acute complications at the donor site** include hematoma or seroma formation. The latissimus dorsi donor site is frequently complicated by seroma formation. Quilting and use of fibrin sealant at the donor site may reduce this complication.4 Drains are a prerequisite to lessen this complication and should be retained until the patient is ambulating and drain output is less than 25 mL in 24 hours. Several authors have reported transient peripheral or sciatic nerve palsy related to intraoperative positioning.18,19 **Long-term donor site complications** include painful unshrinkable, fibrous tendonsite, donor site pain or dysesthesia, and functional losses. Russell and colleagues investigated changes in shoulder girdle function following latissimus dorsi harvest, noting a 5% to 10% decrease in shoulder girdle strength.20 Deutinger and colleagues measured a decrease in adductor strength of 11% following harvest of the gracilis muscle.21 Despite the measured decrease in muscle strength, the relative weakness is rarely noted by the patient, and therefore this possibility is not a contraindication for donor selection.

**REFERENCES**


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