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Refinements in the Treatment of Volkmann Ischemic Contracture of the Forearm: A Thematic Review

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Summary: Volkmann contracture of the forearm is a devastating complication of muscle ischemia. It is most commonly associated with trauma and a sequela of compartment syndrome. In the last few decades, much has improved in our ability to treat these patients. Our preferred treatments are presented along with representative case studies that illustrate the functional gains that can be achieved with a focus on moderate and severe contractures. Refinements in care that have evolved over the past 40 years of experience in treating these patients are presented. *(Plast Reconstr Surg Glob Open 2024; 12:e5532; doi: 10.1097/GOX.0000000000005532; Published online 23 February 2024.)*

INTRODUCTION

More than 150 years ago, Richard von Volkmann reported on a contracture of the forearm that would later become eponymously associated with his name.[1] Volkmann contracture can cause devastating effects on wrist and hand function. Historically, treatment was predominately concerned with the correction of contractures more than the restoration of function. The advent of microsurgery and the ability to transfer muscles as a functional unit has radically changed the landscape for treatment options. This review will present a brief history and description of Volkmann contracture, and current methods of treatment for moderate and severe established Volkmann contracture of the forearm.

HISTORICAL PERSPECTIVE

In his 1870 publication, while writing on infantile paralysis (polio), Volkmann described a contracture of the forearm:

> " … The fingers become drawn into the palm. The wrist is at the same time slightly flexed. Extension of the fingers is opposed by more or less resistance but if the wrist be flexed strongly, the fingers may be passively extended … only one of the patients suffered from 'infantile paralysis'. In the other, the palsy was caused by too tight bandaging of a fractured forearm."[1]

In 1881, von Volkmann ascribed irreversible contractures of the flexor muscles of the hand to ischemic processes in the forearm, believing that the problem was caused by massive venous stasis and simultaneous arterial insufficiency secondary to overly tight bandages.[2–5] (**See figure, Supplemental Digital Content 1**, which shows a portrait of Richard von Volkmann 1830–1889, **http://links.lww.com/PRSGO/D14**.) In 1906, Hildebrand was the first to refer to this condition as Volkmann ischemic contracture.

Over the next 100 years, much would be done to identify and understand the cause of Volkmann contracture. Specifically, compartment syndrome was identified as the causative etiology, and many investigators undertook the study of compartment syndrome. This culminated with the development of the unified concept of compartment syndrome, which is still accepted today.[6,7]

The early identification and treatment of compartment syndrome with emergent fasciotomy decreased the incidence of Volkmann contracture. However, cases still occur and cause devastating effects on limb function. Although the incidence has markedly decreased, the treatment of established Volkmann contracture changed little over the ensuing 120 years. Treatment typically included excision of the infarcted muscle to try to correct contracture, but it did little to restore function. Tendon transfers could be used to replace some function, but the donor musculotendinous units were often compromised by the initial event or would have to work through a scarred soft-tissue bed. Nerve dysfunction associated

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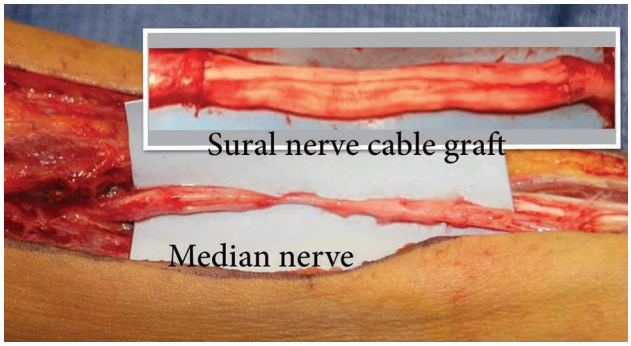

Fig. 1. Focally constricted area of the median nerve that was present in a patient with severe Volkmann contracture. The fibrotic section of the nerve was resected and the defect was reconstructed using a sural nerve autograft.

with compartment syndrome was recognized, but little was done to treat this component of the deficit. Two very small series of patients were treated with a flexor origin muscle slide. Despite functional improvement, this did not gain popularity as a treatment option.[8,9] The advancement of microsurgical techniques, and specifically of functional free muscle transfer, has dramatically changed the outlook for these patients.

CLINICAL DESCRIPTION

Volkmann ischemic contracture is the consequence of prolonged ischemia and irreversible tissue necrosis resulting from compartment syndrome. During the ischemic event, the most vulnerable muscle group is the deep flexor compartment of the forearm, which includes the flexor digitorum profundus (FDP) muscle and the flexor pollicis longus (FPL). In the mildest form of ischemic contracture, these muscles are affected, and often only a portion of these muscles.[10–12] The flexor digitorum superficialis, pronator teres, and wrist flexors are involved with more severe or prolonged ischemia. In the most severe cases, ischemia and subsequent necrosis of the extensor compartment can occur. Nerve involvement can be mild to severe, depending on the extent of ischemia and fibrosis. Nerve involvement in Volkmann ischemic contracture is likely a combination of the initial ischemic event and the late mechanical effects of fibrosis (Fig. 1).[13]

Established Volkmann contracture has a different presentation than acute compartment syndrome. Patients with Volkmann contracture do not have pain but rather have deformity and dysfunction resulting from the prior ischemic event and subsequent muscle fibrosis. There is a broad clinical spectrum of presentation, as each case represents a different extent of muscle necrosis and nerve injury. The muscle fibrosis and neurological deficits lead to deformity of the joints distal to the site of ischemia. Deformity progresses over weeks to months following the ischemic event and then stabilizes. In children, the deformity will progress until skeletal maturity; fibrotic muscles are unable to lengthen at the rate of bone growth. Even when contractures are treated, the affected extremity is shorter due to the tethering of growth across the physis and the ischemic effect on the physis. In the pediatric population, recurrent contracture is common due to the differential growth of bone and fibrotic muscle.

Takeaways

Question: How have the options changed in the treatment of moderate and severe Volkmann contracture?

Findings: The flexor origin slide retains muscle resting length and can produce excellent outcomes in patients with moderate contracture and good active finger flexion. Functional free muscle transfer can restore grasp in moderate and severe contractures.

Meaning: The functional landscape for patients with moderate and severe Volkmann contracture has markedly improved over the last 30 years.

CLASSIFICATION

Several classification systems have been described for Volkmann contracture of the forearm. All classifications are limited by the variability in the extent of both muscle and nerve involvement.[11,12,14,15] Although patterns of necrosis associated with muscle compression have some consistency, fracture location or the etiology of the compartment syndrome also plays a role in the pattern of necrosis.

Our preferred classification system is that of Tsuge.[11] Established Volkmann contracture is divided into mild, moderate, and severe types, according to the extent of muscle involvement. The Tsuge category of severe contractures includes cases of moderate tissue necrosis that are exacerbated by fixed joint contractures, a scarred soft-tissue envelope, or failed surgery. Within each classification type, there is a wide range of clinical presentations. The heterogeneity of presentation makes it difficult to apply a specific treatment based solely on the classification system and confounds meaningful outcome and comparison studies. (**See figure, Supplemental Digital Content 2**, which shows mild-type contracture, **http://links.lww.com/PRSGO/D15**.) (**See figure, Supplemental Digital Content 3**, which shows moderate-type contracture, **http://links.lww.com/PRSGO/D16**.) (**See figure, Supplemental Digital Content 4**, which shows severe-type contracture, **http://links.lww.com/PRSGO/D17**.)

Variations on Volkmann Contracture

Two distinct entities should be reviewed. One is neonatal Volkmann contracture, which is an in utero compression or ischemic event resulting in partial or complete limb necrosis. Unlike neonatal compartment syndrome, which requires emergent fasciotomy, there is no emergent treatment for neonatal Volkmann contracture. (**See figure, Supplemental Digital Content 5**, which shows neonatal compartment syndrome and neonatal Volkmann, **http://links.lww.com/PRSGO/D18**.) The etiology of neonatal Volkmann is unknown but thought to result from both extrinsic and intrinsic factors. Extrinsic factors include oligohydramnios, multiple gestation, fetal macrosomia, abnormal in utero posture, difficult extractions, maternal diabetes, and excessive maternal weight gain. Intrinsic factors include a hypercoagulable state, leading to intravenous or intra-arterial thrombosis.[16] The presentation varies

depending on the time of ischemia relative to the time of delivery, ranging from wrist and finger flexion contracture without skin changes to evidence of skin and muscle necrosis of varying severity. Determining if there is still a component of compartment syndrome is critical. If present, the neonate requires emergent surgery with fasciotomy and excision of necrotic tissue. Necrotic tissue without signs of acute compartment syndrome can be debrided electively within the first 2–4 weeks, provided that the neonate is stable for surgery and general anesthesia. Contractures without tissue necrosis can be addressed throughout development.[17–20]

The other entity is a mimic of Volkmann contracture, described as pseudo-Volkmann contracture. Distinguishing Volkmann contracture from pseudo-Volkmann contracture can be difficult. The patient history of fracture as well as timing and severity of pain helps in distinguishing these diagnoses. Pseudo-Volkmann is most commonly caused by tethering by scar or entrapment of the FDP or the FPL to the fractures of the radius or ulna. Other causes include adhesions around internal fixation plates, or muscle scarring secondary to infection. Most commonly, a pseudo-Volkmann will mimic a mild contracture.[21–23] At surgical exploration, the muscles will seem normal but adherent to the bone. A myotenolysis or tenolysis alone will usually allow for correction of the contracture.

TREATMENT

Treatment is based on patient age, contracture severity, and associated injuries and/or neurological involvement. Diagnostic imaging including radiographs of the uninvolved limb provide information on fracture healing and alignment and growth discrepancy compared with the uninvolved limb. Magnetic resonance imaging (MRI) can evaluate the extent of muscle damage within each compartment and may augment clinical findings. MRI may assist surgical planning and provide prognostic information to the patient and their family. MRI can also help distinguish between Volkmann contracture and pseudo-Volkmann contracture. Magnetic resonance angiography or traditional angiography is useful for patients who have severe involvement who are anticipated to need a functional free muscle transfer, helping to plan the recipient vessel anastomosis site and assess perfusion to the hand.

Treatment is tailored to the severity of muscle and nerve involvement. Intervention may require a combination of muscle and joint contracture releases, tendon transfers, joint fusions, and free functional muscle transfers. Surgery is customized to the functional losses and the remaining donor muscles that are available for transfer.

Mild Type (Localized Muscle Contracture)

Therapy is beneficial to minimize contracture formation following acute compartment syndrome. In children, splinting must continue until skeletal maturity. Therapy should include maintenance of passive joint motion, preservation and strengthening of remaining muscle function, and splinting. Established mild Volkmann contracture may require surgical release. Mild contractures involve all

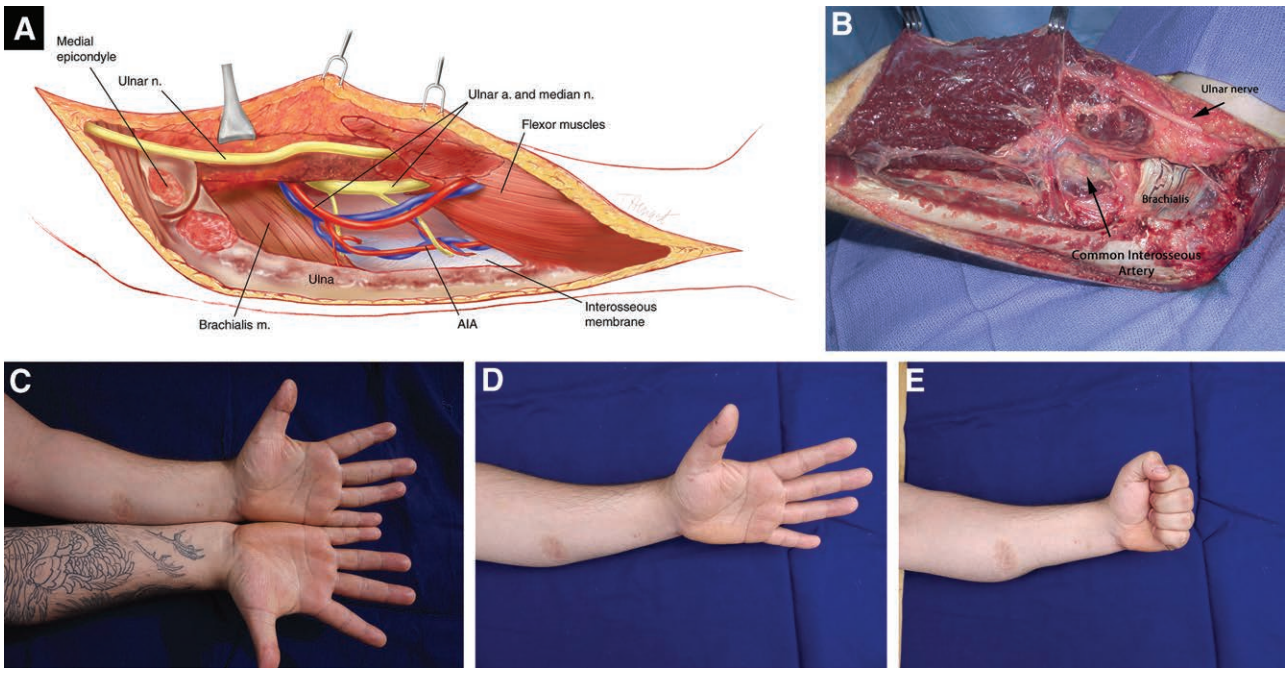

Fig. 2. Flexor origin muscle slide. A, Diagrammatic representation of a flexor muscle slide, done through an extended ulnar approach. 2B, Intraoperative photograph showing the muscle release off of the ulna, interosseous membrane, and radius. C, Ten-year follow-up and functional outcome of patient seen in Figure 4. This patient was age 16 at the time that he developed a compartment syndrome. A flexor muscle slide operation was performed approximately 6 months after the initial injury. The left (affected) forearm is noted to be slightly shorter than the right. D, Full wrist and finger extension are achieved. E, Excellent flexion of the fingers, with strength sufficient to be working as a mechanic. (**See Video 1 [online]**).

or part of the deep flexor compartment, specifically the ring or small FDP muscle, or FPL. An isolated release of these muscles as a limited flexor origin slide or a direct release of the tendon distally with tenodesis to the adjacent functional profundus tendons will both correct the contracture and restore function. For the FPL, a one- or two-level fractional or step-cut lengthening can correct the contracture. This may weaken the muscle power, and a tendon transfer may be necessary for improved pinch strength. Alternatively, a primary tenotomy with tendon transfer may be used.[8,9,11,12,14,24,25]

Moderate Type (Deep and Superficial Flexor Compartment with Neurological Deficit)

Moderate contractures require surgical intervention. When some flexor muscle function is preserved,

Table 1. Technique Outline of the Flexor-pronator Muscle Slide

Technique Points	Pearls and Caveats
• The surgical incision is an extended ulnar approach. It begins approximately 4–5 cm proximal to the medial epicondyle following the course of the ulnar nerve. It continues along the ulnar border of the forearm all the way to the wrist	• As described by Tsuge, this incision can be made along the volar forearm. We believe this causes increased scarring. It is also more difficult to access the flexor muscle origins. • The ulnar incision can be tailored to a shorter incision to address milder contractures
• The ulnar nerve is completely mobilized and transposed	• 4–6 cm of intermuscular septum between the biceps and triceps is excised to avoid nerve compression over this fascia
• The muscle release begins at the medial epicondyle, elevating the flexor-pronator muscle mass	• Preserve the medial ulnar collateral ligament and joint capsule/repair capsule if the joint is violated
• The muscle release continues down the forearm working from proximal to distal and ulnar to radial, elevating the muscle origin from the ulna, across the interosseous membrane to the radial side of the radius • The wrist and fingers are passively manipulated into extension throughout the procedure to check where there is still soft-tissue tension on the origin • The release is complete when full wrist and finger extension are achieved	• The common interosseous artery is found in the proximal forearm and should be preserved. It bifurcates at approximately 6 cm from the elbow flexion crease, dividing into the anterior and posterior interosseous branches, with the posterior interosseous artery supplying the majority of the dorsal compartment
• Carpal tunnel release may be necessary to release tendon adhesions within the carpal canal	• Tendon adhesions in the carpal canal can also be released by elevation through the extended ulnar approach, using an elevated to free any adhesions from the floor of the carpal canal
• The incisions is closed over a large drain • The extremity is immobilized in elbow extension and maximum wrist and finger extension, and thumb extension and abduction	• Long-arm cast for 2–3 wk • Short-arm cast for an additional 3 wk • Skeletally immature patients should continue with night time extension splinting through skeletal maturity

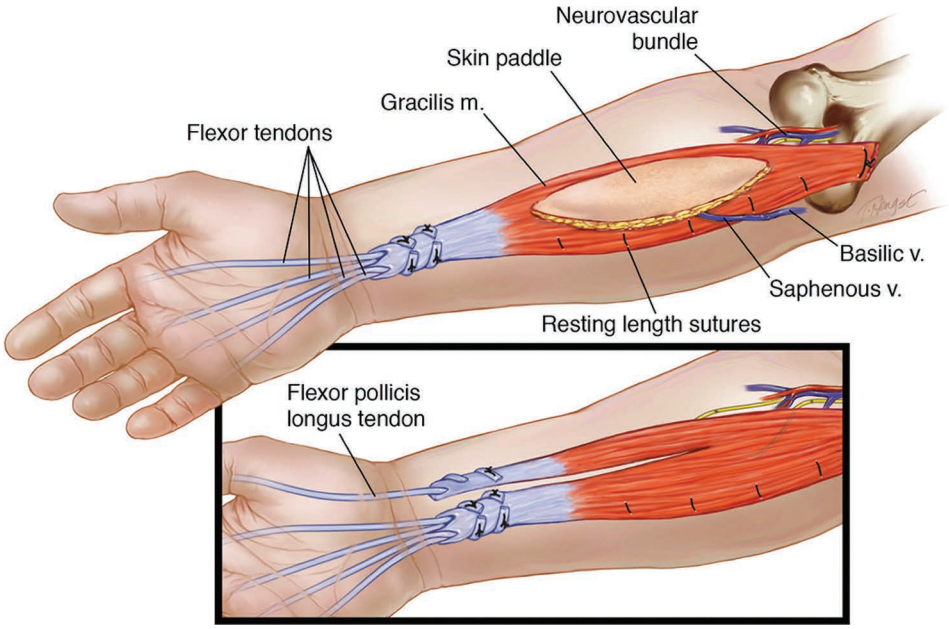

Fig. 3. Schematic demonstrating the use of a free functional muscle transfer for reconstruction of finger flexion in established Volkmann contracture.

a procedure addressing muscle tightness can correct the contracture and still maintain flexor function. Traditionally, this has been performed as a lengthening of the muscle tendon unit. In Volkmann contracture, the muscle resting length has been shortened through scarring of the muscle unit, such that further compromise of the muscle resting length through muscle lengthening results in a greater decrease in the remaining muscle strength. Alternatively, a flexor-pronator muscle slide allows the entire muscle group to move distally, allowing for correction of the joint contracture associated with the shortened muscle while preserving the existing muscle power (Fig. 2). (**See Video 1 [online],** which shows the 10-year follow-up after flexor muscle origin slide demonstrating finger flexion and extension.)

Moderate contractures are characterized by neurological deficits. The severity of neurological deficits contributes to the patient's hand disability and plays a role in determining surgical treatment. The median nerve is the most susceptible to injury, lying between the superficial and deep flexor muscles. Mild median nerve involvement may be treated with neurolysis combined with a flexor origin slide, with a separate release at the carpal tunnel. More severe involvement and/or involvement of the ulnar nerve is more likely to require nerve reconstruction of the scarred and infarcted nerve. Table 1 provides an outline of the surgical technique of the flexor-pronator muscle origin slide.

A flexor origin slide or muscle lengthening procedure is unlikely to provide much functional improvement in moderate contractures where there is little remaining

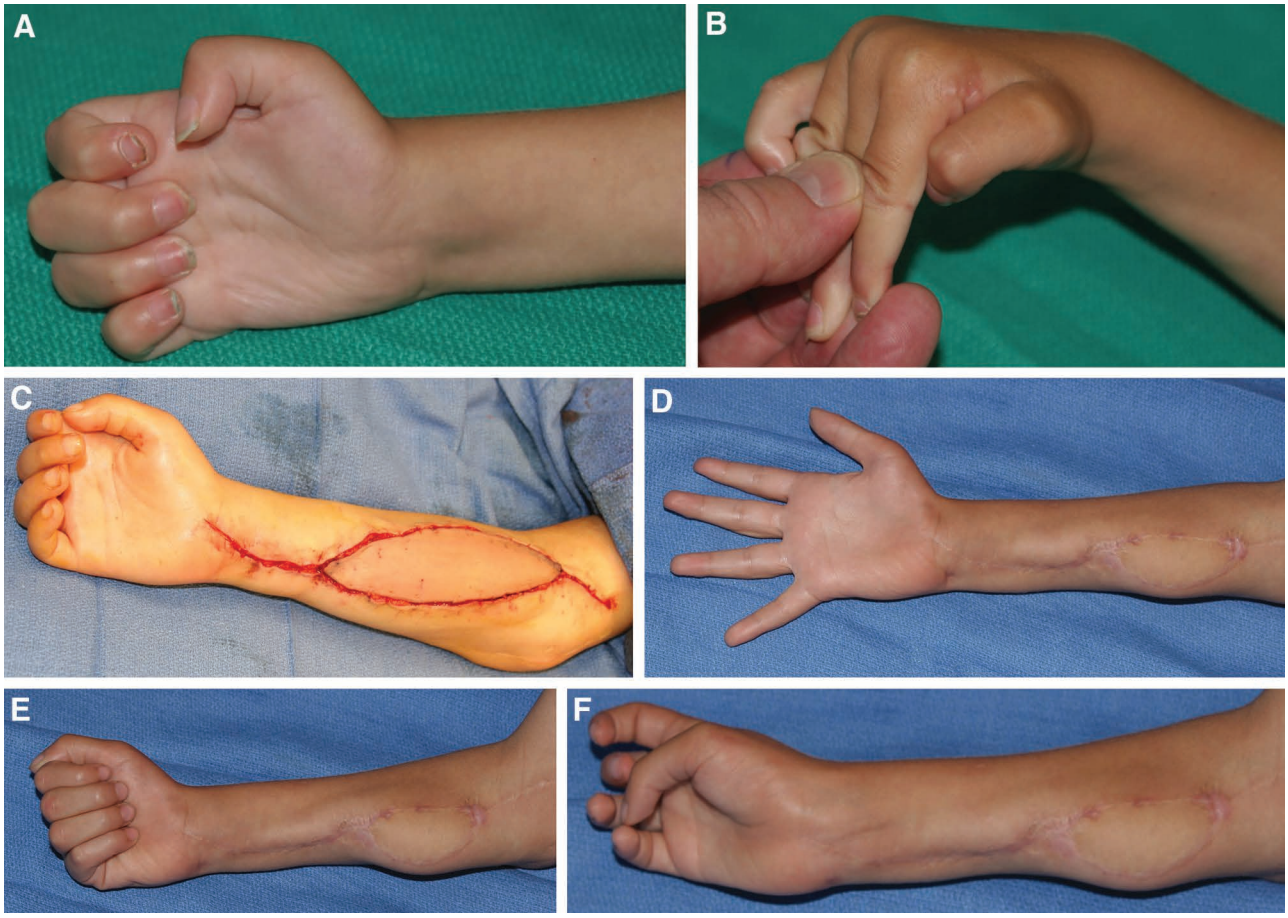

Fig. 4. At age 6, this patient sustained a markedly displaced supracondylar humerus fracture. He had a missed compartment syndrome. Two years after his injury, he was referred to the hand service for evaluation. At that time, he was noted to have sequelae of his prior compartment syndrome, with the presentation of Volkmann ischemic contracture of the forearm. He had minimal active finger flexion and contractures of the metacarpophalangeal joints and proximal interphalangeal joints. These contractures were partially actively and passively correctable with positioning of the wrist in flexion. A, The clinical appearance of his severe-type Volkmann contracture. With the wrist in neutral, there is obligate flexion of the fingers and thumb. B, In the same patient, with the wrist brought into flexion, the fingers can be passively extended at the proximal interphalangeal joints, but the fingers remain tightly flexed at the metacarpophalangeal joints. C, This intraoperative photograph shows the appearance of the hand and forearm after reconstruction of the flexor tendons to the fingers using a free gracilis muscle. The vascular pedicle was anastomosed to the radial artery. The nerve fascicles on the gracilis were independently repaired to the pronator branch of the median nerve and the anterior interosseous nerve to provide independent function of the finger flexors and the thumb flexor. (**See Video 2 [online]**). D, One-year postoperative finger extension. E, One-year postoperative finger flexion. F, One-year postoperative thumb opposition.

muscle power or where a significant neurological impairment exists. A free functional muscle transfer and nerve reconstruction may provide the best chance of meaningful functional return. Adjunctive procedures such as tendon transfers or joint fusions may be necessary either as a simultaneous or staged procedure to improve functional results.

Severe Type (Superficial and Deep Flexor of the Fingers and Wrist, Extensor Muscle Compartment, and Neurological Deficits in the Median and Ulnar Nerves, Affecting Hand Function)

Past treatment for severe contractures included bone and tendon procedures to address contractures but did little to improve function. Today, severe contractures are best treated with infarct excision and functional free muscle transfers in combination with adjunctive procedures including nerve reconstruction of nonfunctional portions of the involved nerves, bone procedures for stability, and tendon transfers if available.

The gracilis muscle is the most commonly used muscle for free functional muscle transfer; it has the best length and excursion to match the finger flexors and extensors. The estimated excursion of the gracilis is 12 cm (based on a 30-cm muscle length).[26] The estimated finger excursion needed for restoration of finger and wrist flexion is 9 cm (Figs. 3–5).[27] (**See Video 2 [online]**, which demonstrates independent thumb flexion and opposition.) (**See Video 3 [online]**, which demonstrates independent thumb flexion and opposition.) Other muscles (pectoralis major, medial gastrocnemius, and latissimus dorsi) have lesser excursion and do not produce good restoration of finger flexion. Both the gracilis and latissimus dorsi myocutaneous flaps can be transferred with a large skin envelope to address soft-tissue loss in the forearm. The myocutaneous gracilis flap in particular can be harvested with a broad area of fascia that includes the entire medial thigh.[28] This provides a broad area of tissue coverage and a robust fascial sleeve in which the muscle can glide. Appropriate marking of the muscle resting length and establishing a strong muscle origin and insertion are critical to achieving good functional results.[29,30] Separate motor functional units of the gracilis can be used to restore independent FDP and FPL motor function (Fig. 5).[31–34] Simultaneous median nerve reconstruction for necrotic or fibrotic nonfunctional nerve should be performed at the time of reconstruction. We prefer cabled sural nerve autograft for reconstruction. For severe-type contractures with extensive involvement of the extensor compartment, a double free muscle transfer should be considered. Therapy for muscle and joint mobilization, customized splinting, and muscle retraining remain an important part of patient care (Tables 2–4).

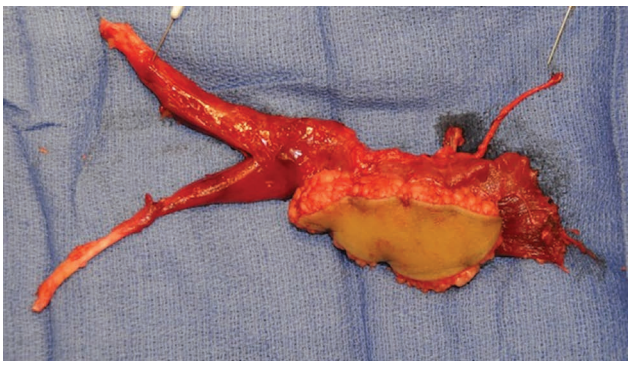

Fig. 5. Gracilis muscle shown after harvest from the medial thigh and separated into its two independent neuromuscular territories. There are two distinct fascicles forming the branch to the gracilis from the obturator nerve. These fascicles are separated and independently stimulated. The pattern of muscle contraction elicited by stimulation of each fascicle shows the plane of demarcation between the muscle territories, which allows the muscle to be separated.

Table 2. Technique Outline for Free Functional Muscle Transfer: Recipient Site

Technique Points	Pearls and Caveats
Recipient site	• The recipient site is addressed first to identify and confirm suitability of recipient artery and veins, and motor donor nerves
• For flexor reconstruction: • Longitudinal incision from the carpal tunnel to the antecubital fossa	
• Excision of infarcted and fibrotic muscles to allow correction of the contracture	• If there is no ability to passively flex the fingers and thumb by manual retraction on FDP and FPL, then the procedure should be aborted. Even with tenolysis, there is a high risk of recurrent scarring, making the procedure ineffective
• The superficialis tendons are excised at the level of the wrist • The FDP tendons are divided at the musculotendinous junction and sutured together with tensioning the reproduces the normal cascade of the fingers • The FPL tendon is separated at the musculotendinous junction	• The median and ulnar nerves should be inspected. Depending on preoperative function and appearance of the nerve, the nerves should be reconstructed at this time
• Recipient artery and veins and motor donor nerves are identified	• Recipient artery Radial or ulnar artery (end to end or end to side) end to side to brachial • Recipient veins Venae comitantes AND superficial veins (cephalic or basilic) • Motor nerve donors (most common) Anterior interosseous (FDP) Branch of median nerve to pronator (FPL)

Table 3. Technique Outline for Free Functional Muscle Transfer: Gracilis Harvest

Technique Points	Pearls and Caveats
Gracilis harvest	
• The gracilis is harvested with the patient supine and the leg in a frog lateral position • Care is taken to leave the fascial sleeve intact, and the subcutaneous fat is elevated anterior and posterior to the skin paddle and gracilis muscle	• The incision is started distally to identify the tendon of the gracilis Retraction on the tendon allows identification of the mid-axis of the gracilis • A skin paddle is designed centered over the gracilis muscle
• Harvesting of the gracilis muscle and the skin paddle should include a fascial skirt extending 4–6 cm anterior and posterior beyond the skin paddle	• The skin paddle is secured to the underlying fascia using several sutures to prevent shear injury to the perforating vessels
• Muscle resting length is marked using sutures placed at 5-cm intervals	• Resting length is measured with the thigh in abduction and the knee in extension
• The tendon is released at its tibial insertion from the pes anserine • The muscle is elevated from distal to proximal	• Care is taken to protect the deep fascia on the undersurface of the gracilis. This is confluent with the medial thigh fascia which is elevated anterior and posterior to the margins of the skin paddle
• The main pedicle is identified in the proximal third of the muscle • The pedicle is dissected as far proximally as possible to have the maximum length for insetting • The greater saphenous vein must be included in the skin flap dissection	• The dominant pedicle is usually 4–6 cm from the muscle origin and enters the muscle at a 90 degree angle. • The obturator nerve lies proximal to the dominant pedicle and enters the muscle at 45 degrees
• The obturator nerve is stimulated in situ • There are two fascicles which are separated and independently stimulated to identify the separate neuromuscular territories of the gracilis • The tendon is divided past the musculotendinous junction following the neuromuscular territories of the stimulated muscle	• If the muscle looks atrophic or does not stimulate (prior trauma of the obturator nerve), then it should not be harvested • Separation of the fascicles is easiest with the muscle in situ

Table 4. Technique Outline for Free Functional Muscle Transfer: Insetting

Technique Points	Pearls and Caveats
Insetting	

• Order of repair
  • Arterial
  • Venous (deep)
  • Nerves
  • Venous (superficial)
  • Origin
  • Insertion
	• The gracilis muscle is loosely stapled into position
	• The arterial repair is completed to allow revascularization of the muscle within 30–40 min of harvest
	• Venous repair is performed starting with the venae comitantes
	• The nerve is coapted as closely as possible to the muscle belly using separate motor donors for FDP and FPL reanimation
	• The greater saphenous vein is repaired to the cephalic or basilic vein

• Muscle origin • The gracilis muscle origin is secured to the medial epicondyle	• Several mattress sutures are used to secure the muscle of gracilis to the fascia overlying the medial epicondyle • 2-0 nonabsorbable suture is used
• The gracilis is then stretched to restore its preharvest resting length with the wrist and fingers in full extension and the thumb in full extension and abduction. • Tendon repairs are performed with nonabsorbable 2-0 or 3-0 suture using a Pulvertaft weave	• The larger of the neurovascular territory tendons is repaired to the FDP tendons • The smaller of the tendons is repaired to the FPL. • If following the tendon repair the muscle resting length has shortened, then proximal muscle is excised and the origin is resecured
• Sutures previously placed to secure the skin paddle and sutures placed to define the resting length should be removed during the insetting of the skin	• Wound closure is performed over a large bore drain, no matter how dry the tissue bed is at the time of closure

• Immobilization
  • Origin and insertion sites are protected for 8–10 wk
  • Passive flexion exercises at 2 wk
  • Full passive motion at 8–10 wk
  • Stimulation of the transferred muscle at 3–6 mo
	• Long arm posterior splint supplemented with a dorsal block splint
	  • Elbow at 45 degree flexion
	  • Wrist in neutral to 20 degree flexion
	  • Fingers in MCPJ flexion

MCPJ, metacarpophalangeal joint.

Acute Compartment Syndrome with Liquefactive Muscle Necrosis

Some cases of acute compartment syndrome should be considered for immediate reconstruction, specifically when there is extensive or liquefactive muscle necrosis. Early wide excision of necrotic muscle with immediate reconstruction using a functional free muscle transfer has the ability to prevent joint contracture, prevent sclerotic strangulation of intact nerves, and allow early restoration of finger flexion (and extension if necessary). There is no need in the setting of liquefactive necrosis to wait and see what function returns. Function will not return in the setting of dead and dissolved muscles. Waiting will only add to the disability of the limb, greater fibrosis around

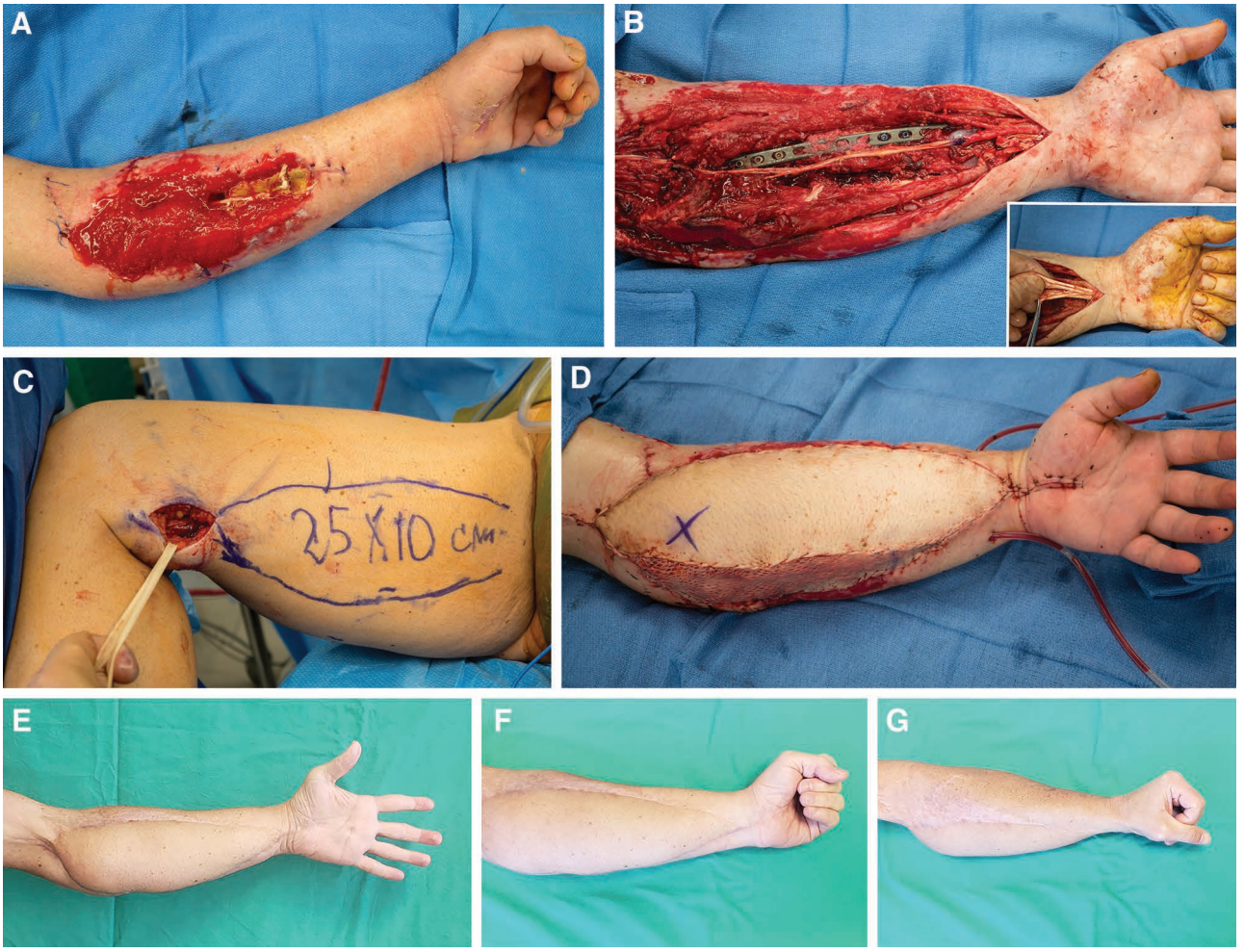

Fig. 6. Functional free muscle transfer for acute compartment syndrome with early liquefactive necrosis. This 35-year-old police officer was treated at an outside facility for an open both-bone forearm fracture with associated elbow dislocation resulting from an all-terrain-vehicle accident. His initial presentation at 6 weeks from injury demonstrated a subacute open wound with limited function of the hand and absent sensation in the median nerve distribution. He underwent serial debridement and revision bone stabilization. When the wound bed was clean, sural nerve autograft was used to reconstruct the median nerve, and a functional free gracilis muscle was used to provide soft-tissue coverage and to independently reconstruct finger and thumb flexion. A, Forearm appearance on presentation to the hand service. B, Wound appearance after several debridements. Dead flexor muscles have been removed, and the primary remaining tissues are the flexor tendons to the fingers and thumb. C, The gracilis muscle is harvested from the medial thigh with a large area of overlying skin. This donor site is closed primarily. D, The intraoperative appearance of the muscle once set into the forearm. The gracilis muscle is used to reconstruct the functional finger flexor deficits, and the overlying soft-tissue paddle is used to reconstruct the soft-tissue defect. Split thickness skin graft is used to supplement soft-tissue coverage. E, Finger extension at 14 months postoperatively. F, Finger flexion at 14 months postoperatively as seen in coronal plane. G, Finger flexion at 14 months postoperatively as seen in the sagittal plane. (**See Video 3 [online]**).

the nerves, and greater nerve dysfunction (Fig. 6 and **see Video 3 [online]**).

OUTCOMES

Outcomes are difficult to assess in this group of patients. Studies are limited by small patient numbers, variability in initial presentations, varied surgical techniques, and difficulty in following patients through to skeletal maturity.[8,9,11,35–37]

Sharma and Swam reported on outcomes of the flexor origin slide in 19 patients with moderate Volkmann contracture. Seventy-eight percent resulted in good outcomes with improvement in grip, dexterity, and appearance.[37] Griffart et al reported on outcomes in seven patients with mild contracture treated with flexor muscle origin slide. These patients had 72% of the grip strength of the contralateral hand. All patients were satisfied with surgery and would have the surgery repeated.[36]

Ultee and Hovius[38] reported on 25 patients, whose treatment varied based on the severity of involvement. 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 those who underwent functional free muscle transfer. Tendon lengthening alone often resulted in recurrent

contracture. Finally, in patients having sufficient remaining muscle, procedures which combined infarct excision, tenolysis, neurolysis, and tendon transfer when necessary produced good hand function.[38] Sundararaj and Mani noted improvement in sensory function in conjunction with neurolysis. Additional procedures were done simultaneously, and little analysis of outcomes of those other procedures was given.[39]

Sabapathy et al reported on 22 patients with Volkmann contracture treated with free functional muscle transfers for finger flexor reconstruction. In their series, four of 22 (18%) required nerve reconstruction. Twenty patients recovered M3 or M4 motor function. Ten patients who had measurement of grip strength achieved 24% of the strength of the contralateral hand.[40]

CONCLUSIONS

As with many diagnoses in medicine, the best treatment for Volkmann ischemic contracture is prevention. Early recognition and prompt treatment of compartment syndrome has decreased, but not eliminated, the incidence and severity of late contracture and hand dysfunction. We have found the flexor muscle slide is our best treatment option for mild and moderate deformity. This procedure can be combined with additional reconstructive procedures to maximize functional outcome. We believe this procedure results in the best preservation of the muscle resting length and limits the scarring around the adjacent muscles. The ulnar approach as opposed to the anterior approach avoids additional scarring within the flexor compartment. Refinements in the original technique described by Page in 1923 include the use of the extended ulnar approach with a longer proximal and distal incision that allows elevation and exposure along the full length of the elbow and forearm from the ulnar side, across the interosseous membrane to the radial side of the radius. This approach also allows access to the distal brachialis muscle which can be fractionally lengthened in the setting of elbow contracture. Ulnar nerve transposition reduces risk of tension on the ulnar nerve that may occur with restoration of wrist and finger extension (Table 5).

For severe cases, functional free muscle transfer has been transformative in offering functional recovery to patients with this devastating injury. Refinements in the techniques initially described include the ability to harvest a large and reliable skin paddle by including a large swathe of fascia in the harvest and the greater saphenous vein to augment outflow. Primary wound closure is often possible with this flap. The ability to provide independent thumb and finger flexions by separating the obturator fascicle of the gracilis provides more natural hand function. Preoperative identification of nerve deficits in the median (most common) and or ulnar nerves preoperatively prompts careful inspection of these nerves for signs of necrosis that would require simultaneous nerve reconstruction below the functional muscle transfer (Table 5).

We also advocate early wide excision and functional free muscle transfer for patients with severe compartment syndrome and liquefactive necrosis. This is done within the first 1–2 weeks when the tissue bed is clean. Early intervention may limit both joint contractures and injury to the nerves, decreasing the distal problems associated with motor and sensory impairment in the hand (Table 5).

Table 5. Summary of Refinements for Flexor Muscle Origin Slide and for Functional Free Muscle Transfer

Refinements in the flexor origin slide
• Extended ulnar approach from proximal to the elbow to the wrist flexion crease
• Allows exposure across the volar forearm to the radial side of the radius
• Provides access to the brachialis muscle which can be fractionally lengthened in the setting of an elbow contracture
• Ulnar nerve transposition in combination with flexor origin slide
• Extension to the carpal tunnel allowing tenolysis of the tendons within the carpal canal (a previously ignored area contributing to contracture
Refinements in functional free muscle transfer
• Harvest of a large skin paddle with the gracilis muscle which helps with muscle gliding and with soft-tissue coverage
• Augmenting venous outflow by including the saphenous vein in the harvest
• Separation of the gracilis muscle into independent neurovascular territories to allow separate flexion of the thumb and fingers
• Early nerve reconstruction in the setting of dysvascular nerves
• Early treatment of liquefactive muscle injury that may prevent contracture facilitate early recovery

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DISCLOSURE

The authors have no financial interest to declare in relation to the content of this article.

PATIENT CONSENT

Patients provided written consent for the use of their images.

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