Abstract
Acute compartment syndrome occurs when pressure within a fibro-osseous space increases to a level that results in a decreased perfusion gradient across tissue capillary beds. Compartment syndromes of the hand, forearm, and upper arm can result in tissue necrosis, which can lead to devastating loss of function. The etiology of acute compartment syndrome in the upper extremity is diverse, and a high index of suspicion must be maintained. Pain out of proportion to injury is the most reliable early symptom of impending compartment syndrome. Diagnosis is particularly difficult in obtunded patients and in young children. Early recognition and expeditious surgical treatment are essential to obtain a good clinical outcome and prevent permanent disability.

Compartment syndrome was first described in 1881 by Richard von Volkmann. The etiology, pathophysiology, and management of compartment syndrome and the associated complications have been extensively described. Acute compartment syndrome (ACS) occurs with elevation of interstitial pressure in a closed fascial compartment, resulting in microvascular compromise. This causes the perfusion gradient to fall below a critical value, leading to ischemia of the tissues within this confined space. In the upper extremity, the dorsal and volar compartments of the forearm are the most commonly affected. Upper extremity ACS can lead to devastating loss of function, including Volkmann ischemic contracture, neurologic deficit, infection, amputation, and death.

A wide range of causes of ACS in the upper extremity has been reported, and a high index of suspicion must be maintained. Distal radius fracture in adults and supracondylar humerus fracture in children are the most frequent causes of compartment syndrome in the upper extremity. Compartment syndrome is a clinical diagnosis, and it can be difficult to make in certain patient populations, such as persons who are obtunded and children. Emergent surgical treatment is required; the most important determinant of outcome is early recognition and expeditious surgical intervention. Reconstructive procedures can be performed to improve the function of the affected upper extremity in the patient with Volkmann contracture; however, return of normal function should not be expected.

Anatomy
The upper extremity contains 15 compartments. The upper arm consists of a flexor (ie, volar) compartment and an extensor (ie, dorsal) compartment. The forearm is divided into three compartments: volar, dorsal, and lateral (ie, mobile wad) (Figure 1). The hand has 10 compart-
ments: hypothenar, thenar, and adductor pollicis as well as four dorsal interosseous and three volar interosseous (Figure 2). The volar compartment contains the flexor muscles of the wrist and digits, including the flexor digitorum superficialis (FDS), flexor carpi radialis (FCR), flexor pollicis longus (FPL), flexor digitorum profundus (FDP), and flexor carpi ulnaris (FCU), as well as the ulnar nerve (UN), ulnar artery (UA), median nerve (MN), median artery (MA), radial artery (RA), superficial branch of the radial nerve (RN), anterior interosseous artery (AIA), and anterior interosseous nerve (AIN). The dorsal and volar compartments are separated by the interosseous membrane. The dorsal compartment contains the finger and thumb extensors and the long thumb abductor as well as the posterior interosseous artery (PIA), posterior interosseous nerve (PIN), extensor carpi ulnaris (ECU), extensor pollicis longus (EPL), and extensor digitorum communis (EDC). The mobile wad, which is often considered to be a third compartment, is composed of the extensor carpi radialis brevis and longus muscles (ECR) and the brachioradialis (BR). (Reproduced with permission from Ouellette EA: Compartment syndromes in obtunded patients. Hand Clin 1998;14[3]:431-450.)

Cross section at the junction of the proximal and middle thirds of the forearm demonstrating the compartments and the important neurovascular structures. The volar compartment contains the flexor muscles of the wrist and digits, including the flexor digitorum superficialis (FDS), flexor carpi radialis (FCR), flexor pollicis longus (FPL), flexor digitorum profundus (FDP), and flexor carpi ulnaris (FCU), as well as the ulnar nerve (UN), ulnar artery (UA), median nerve (MN), median artery (MA), radial artery (RA), superficial branch of the radial nerve (RN), anterior interosseous artery (AIA), and anterior interosseous nerve (AIN). The dorsal and volar compartments are separated by the interosseous membrane. The dorsal compartment contains the finger and thumb extensors and the long thumb abductor as well as the posterior interosseous artery (PIA), posterior interosseous nerve (PIN), extensor carpi ulnaris (ECU), extensor pollicis longus (EPL), and extensor digitorum communis (EDC). The mobile wad, which is often considered to be a third compartment, is composed of the extensor carpi radialis brevis and longus muscles (ECR) and the brachioradialis (BR). (Reproduced with permission from Ouellette EA: Compartment syndromes in obtunded patients. Hand Clin 1998;14[3]:431-450.)

Digital fasciotomy is necessary in some cases. In the upper arm, the medial and the lateral intermuscular septa separate the flexor compartment from the extensor compartment. The brachial fascia is a dense fibrous sheath that surrounds the muscles in each of the two compartments. In the forearm, the stiff interosseous membrane bridges the distance between the radius and the ulna. Just anterior to this membrane lie the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus muscles. Some consider the pronator quadratus to lie within an additional compartment of the distal volar forearm, separate from the flexor tendons at this level. These deep volar muscles are the most commonly damaged muscles in forearm compartment syndrome. The remaining, more superficial flexor muscles are less prone to ischemia than their deeper counterparts. The finger and wrist extensors lie on the posterior aspect of the forearm. Isolated exercise-induced compartment syndromes of the extensor carpi ulnaris muscle have been noted in case reports. The mobile wad, which consists of the brachioradialis, flexor carpi radialis longus, and flexor carpi radialis brevis tendons, is rarely involved. The antebrachial fascia, which is a continuation of the brachial fascia, envelops the compartments and muscles in the forearm.

The compartments of the hand are divided by the carpal bones, metacarpals, and individual investing fascial layers. Pressures within the carpal tunnel may become elevated in cases of hand or volar forearm compartment syndrome. Release of the transverse carpal ligament is often necessary in addition to release of the other involved compartments. The digital fascial compartments are bound by the Cleland ligament and the Grayson ligament, and elevated pressure may occur in individual digits (Figure 3). Compartment syndrome of the hand typically necessitates carpal tunnel release and dorsal interosseous compartment releases; however, any compartment may be involved and may require release. The decision regarding the number and location of incisions should be based on the clinical findings and on intraoperative pressure measurements.
Etiology

ACS of the upper extremity is caused by a myriad of factors, including fracture, bleeding disorders, constrictive dressing and casting, arterial injury, extravasation of intravenous or intraosseous infusion, and regional anesthesia. Anything that causes increased volume within the confined fascial space (eg, bleeding, edema, purulent material, extraneous fluids) or restricts the compartment from expanding (eg, burn, casting, dressing, tourniquet) can cause ACS.

It is difficult to estimate the true incidence of compartment syndrome of the upper extremity, but most cases occur in the setting of fracture. McQueen et al analyzed 164 patients with traumatic ACS. In 69% of cases, compartment syndrome was associated with fracture. The most frequent fracture types observed were of the tibial diaphysis (36%), the distal radius (9.8%), and the diaphysis of the forearm bones (7.9%). Only 2.5% occurred following hand fracture, and the incidence was 0.6% each for elbow fracture-dislocation and humeral fracture. Soft-tissue injury without fracture was the second most common cause of injury (23.2%). Incidence in the forearm and hand was 5.5% and 0.6%, respectively. Within this subgroup, 10.3% occurred in patients with bleeding disorders or who were taking anticoagulants.

Grottka et al reviewed a national trauma database and found the incidence of pediatric forearm compartment syndrome to be 1%. Ouellette and Kelly reported on 19 patients with compartment syndrome of the hand. Most cases were iatrogenic, resulting from complications related to intravenous or intra-arterial administration of drugs. The incidence of compartment syndrome in unstable fractures of the distal end of the radius is substantially higher with concomitant ipsilateral elbow injury than with distal radial fracture alone.

Pathophysiology

Compartment syndrome is caused by an elevation of pressure within a fibro-osseous space resulting in decreased tissue perfusion. The initial rise in intracompartmental pressure causes increased extravascular venous pressure. Because of lack of musculature in the wall media, this relatively small rise in pressure causes the venule walls to collapse. The resulting decrease in hydrostatic gradient causes reduced local perfusion and increased interstitial pressure. This increased interstitial pressure in turn causes increased edema within the compartment, and this cascade of events repeats itself perpetually.

Vasospasm and shock lead to decreased arteriolar pressure and possibly closure of end arterioles, leading to a further decrease in tissue perfusion (Figure 4).

Ischemia occurs when a critical threshold is reached in the local arteriovenous gradient and when circulation is compromised to the point that blood flow is insufficient to meet the metabolic demands of the tissue. This critical variable is represented by the absolute difference between compartment pressure and blood pressure. Heppenstall and colleagues prefer to compare intracompartmental pressure with mean arterial pressure, but diastolic blood pressure is easier to obtain and calculate. Experimental data in a canine model have shown that terminal arterial pressure is equal to diastolic blood pressure, thus, we use diastolic blood pressure to determine the critical threshold.

In a canine study, muscle necrosis occurred when the intracompartmental pressure reached 40 mm Hg, which is lower than the diastolic blood pressure, suggesting that the critical threshold is not reached. The critical threshold is reached when the intracompartmental pressure exceeds the diastolic blood pressure, which is approximately 50-60 mm Hg in a canine model. This suggests that the critical threshold is not reached when the intracompartmental pressure reaches 40 mm Hg, which is lower than the diastolic blood pressure.
Mental pressure rose to a level 20 mm Hg below diastolic blood pressure. Heckman et al demonstrated that irreversible changes to the peripheral nerves and skeletal muscle occur following 8 hours of complete ischemia. Following onset of cellular anoxia, affected cells begin anaerobic metabolism and may die. Devastating loss of function may occur following substantial loss of muscle and neural tissue in the upper extremity. Muscle ischemia can lead to release of myoglobin, which can cause myoglobinuria, metabolic acidosis, and hyperkalemia. Renal failure, cardiac arrhythmia, cardiac failure, and shock may then ensue. The magnitude of these systemic effects is dependent on the duration of ischemia and the size of the muscle compartments involved.

### Clinical Evaluation

Historically, the five Ps (pain, pallor, pulselessness, paralysis, paresthesia) were taught as the symptoms that herald compartment syndrome. Pulselessness is rare, occurring only after arterial injury. Most of these symptoms present only after a substantial amount of time has elapsed following the onset of compartment syndrome, and outcomes typically are poor even with expedient fasciotomy. Pain out of proportion to injury and pain with passive stretching of muscles in the involved compartment are the earliest indicators of compartment syndrome. Compartment syndrome also should be suspected with tense, swollen compartments. In a series of patients with compartment syndrome of the hand, Ouellette and Kelly demonstrated that the most consistent clinical finding was a tense, swollen hand in an intrinsic minus position (ie, extension of the metacarpophalangeal joints and flexion of the intercarpal joints) (Figure 5).

ACS is typically diagnosed clinically. However, it may be necessary to measure compartment pressure (Figure 6). Animal studies demonstrate that compartment syndrome is indicated in the presence of a difference between diastolic blood pressure and the compartment measuring.
≤20 mm Hg or a difference between mean arterial pressure and the compartment measuring ≤30 mm Hg. Fasciotomy is often performed in patients with absolute pressure measuring >30 mm Hg. We consider a differential between diastolic pressure and the compartment of ≤20 mm Hg to be an absolute indication for emergent fasciotomy. In the patient with swollen compartments and nearly normal pressures (<30 mm Hg absolute pressure), we advocate waiting and performing serial examinations and pressure measurements. Increasing swelling and rising pressures warrant decompression even when the pressure differential is ≤20 mm Hg. Emergent fasciotomy is also performed in the setting of a concerning physical examination and extremely elevated pressures, even with a pressure differential of >20 mm Hg. Compartment pressure should be measured when suspected in a patient who is uncooperative, who has an altered mental status, or whose young age renders the clinical examination inadequate. In pediatric patients, Bae et al demonstrated that an increased need for analgesics was most predictive of compartment syndrome.

**Compartment Pressure Measurement**

We typically measure compartment pressures in patients with equivocal
An equivocal examination is one in which compartment syndrome is suspected and uncertainty exists regarding the clinical examination. Measurements are also often taken in the operating theater to verify which compartment or compartments are affected and to ensure that they are fully decompressed following fasciotomy. Compartments that clinically appear to be involved are tested, and measurements are taken when the compartment appears to be the most tense and swollen. In the case of associated fracture, the catheter is inserted close to the fracture site. Otherwise, measurements are taken at the location that is most accessible to the compartment. Typically, a single measurement is taken for each suspected compartment. When in doubt regarding the resulting pressure value, repeat measurements should be obtained at other sites.

The two most common measurement techniques involve the use of a slit catheter or a side port needle. Straight needles have been shown to provide less accurate results. The catheter may be left in situ for repeat or continuous pressure measurements. In the past, we used an arterial line and a pressure transducer similar to the continuous-infusion technique described by Matsen, but we did not use the infusion pump. More recently we have begun using the Stryker Intra-Compartmental Pressure Monitor System (Stryker, Kalamazoo, MI), which is more convenient and requires no setup.

Ronel et al described a technique that allows decompression of the superficial muscles and the deep muscles of the volar forearm as well as release of the median nerve at possible sites of compression. We use this approach because it causes the least amount of iatrogenic injury to the superficial muscles, arteries, and nerves compared with other described approaches to the volar forearm. A longitudinal skin incision is made beginning just radial to the flexor carpi ulnaris at the wrist. The incision is extended proximally to the medial epicondyle, then curved radially over the antecubital fossa (Figure 7). Distally, the incision may be extended to the midline to incorporate open carpal tunnel release. The lacertus fibrosus and fascia over the flexor carpi ulnaris are opened. The flexor carpi ulnaris is retracted ulnarily, and the flexor digitorum superficialis is retracted radially to allow visualization and opening of the
fascia over the deep muscles of the forearm.

Release of the volar compartment often results in decompression of the dorsal compartment and the mobile wad, as well. Should the pressures remain elevated in the dorsal compartment or the mobile wad, a second fasciotomy is performed. We use a dorsal longitudinal incision extending from 2 cm distal to the lateral epicondyle of the humerus toward the midline of the wrist. Deep dissection is made through the interval between the extensor digitorum communis and the extensor carpi radialis brevis. This allows decompression of the musculature in the dorsal compartment and the mobile wad.

Release of the volar and dorsal interosseous compartments and the adductor compartment to the thumb is begun by making two longitudinal incisions dorsally over the 2nd and 4th metacarpals (Figure 2 and Figure 8). The fascia over the dorsal interosseous muscles is incised. To decompress the first volar interosseous and adductor pollicis muscles, blunt dissection is performed along the ulnar side of the 2nd metacarpal through the more radial incision. The second and third volar interosseous compartments are released by dissecting along the radial aspect of the 4th and 5th metacarpals through the more ulnar incision. To release the thenar and hypothenar compartments, longitudinal incisions are made at the junctions of the glabrous and nonglabrous skin over the radial side of the 1st metacarpal and the ulnar side of the 5th metacarpal.

Compartment syndrome of the upper arm is rare. The anterior and the posterior compartments can be decompressed through a single medial or lateral incision. Maginn and Elliot suggested that the creation of two separate midline incisions directly over the anterior and posterior compartments causes less disruption.

Figure 7

Surgical approaches to the volar compartment of the forearm. A, Illustration demonstrating the skin incision for the ulnar approach. B, The interval between the flexor carpi ulnaris muscle and the flexor digitorum superficialis muscle is entered. C, After the ulnar nerve and artery are identified and protected, the fascia over the deep compartment is opened completely. D, Incision for the Henry approach, which is performed through the interval between the flexor carpi radialis and the brachioradialis muscles. (Reproduced with permission from Ouellette EA: Compartment syndromes in obtunded patients. Hand Clin 1998;14[3]:431-450.)
to the perforating septal vessels, resulting in decreased iatrogenic ischemia to the muscles and flaps compared with a single medial incision. In our experience, compartment syndrome in the upper extremity is usually associated with humeral fracture, which requires open reduction and internal fixation. This cannot be done through a medial approach. It is rare that both compartments are involved. Thus, we make a longitudinal incision over the affected compartment to first perform thorough decompression. We use the same incision for open reduction and internal fixation of the fractured humerus. If the other compartment is also involved, a second fasciotomy is made over it.

**Wound Closure**

Wounds are typically left open and initially are covered with sterile wet-to-dry dressings. The skin should not be closed acutely following fasciotomy. We do not routinely use reten-tion sutures or rubber catheters to prevent wound edge retraction. In wounds with exposed tendon, bone, or neurovascular structures, a bovine collagen matrix wound dressing (Integra artificial dermis; Integra Life-Sciences, Plainsboro, NJ) is applied to prevent desiccation. This can be grafted over later. The patient is returned to the operating suite after 48 to 72 hours for irrigation and débridement. Delayed primary wound closure may be done at this time, as well. In the patient with muscle necrosis, all nonviable muscle is sharply débrided. When muscle viability is a concern, repeat débridement is done every 48 hours. Undermining the skin flaps often assists with tension-free closure. For wounds that cannot be closed, a vacuum-assisted wound closure system (VAC Therapy System; KCI, San Antonio, TX) is placed, and split-thickness skin grafting is performed at a later date. Potential advantages of a vacuum-assisted closure system include shorter hospital stays, reduced need for skin grafting, and a lower rate of nosocomial infection.

**Outcomes and Complications**

Outcomes following compartment syndrome are dependent on injury severity, duration of ischemia, and preinjury status and comorbidities. The amount of infarction varies considerably, as do outcomes. Time from diagnosis to fasciotomy is the most important predictor of outcome. The critical delay from diagnosis to treatment is reported to be 6 to 24 hours. Irreversible damage has been shown to occur after 8 hours in a canine model. Full recovery with minimal residual dysfunction can be expected with prompt diagnosis and intervention of compartment syndrome of the upper extremity. Delay beyond 6 to 24 hours may result in Volkmann ischemic contracture, neurologic deficit, infection, amputation, or death. How late is too late to perform surgical decompression? In most cases the exact time of onset of compartment syndrome cannot be determined; thus, it is impossible to determine its duration. In such instances, the clinical examination is relied on heavily. Voluntary muscle contraction in the setting of compression is indicative of the existence of viable muscle, and emergent fasciotomy should be performed. In the patient with no demonstrable muscle function, the best treatment may involve supportive care for myoglobinuria and aggressive splinting to maintain the arm and hand in a functional position.

Ouellette and Kelly noted poor results in 4 of 19 patients with hand compartment syndrome. All four patients were obtunded at the time of onset, and two required amputation. A high index of suspicion for compartment syndrome must be maintained in persons who are who are difficult to examine and who cannot communicate effectively. Gelberman et al reported the results of surgical decompression of compartment syndrome of the forearm in 10 patients. No patient developed Volkmann contracture, but only two patients had normal postoperative results. Six patients who underwent volar fasciotomy required supplemental skin grafting. The worst results were observed in patients with severe crush injuries and in another with arterial injury.
**Volkmann Contracture**

Irreversible tissue ischemia results in muscle necrosis within the affected compartment, which leads to contracture. In the forearm, the typical posture that develops includes elbow flexion, forearm pronation, wrist flexion, and thumb adduction with the metacarpophalangeal joints in extension and the interphalangeal joints in flexion. The median nerve is often affected to a greater degree than the ulnar nerve because the median nerve lies in the deeper zone that is more severely compromised by ischemia.

Tsuge\(^4\) classified contracture as mild, moderate, and severe. The mild type involves mild ischemic contracture primarily of the flexor digitorum profundus and no neurologic deficit. In the moderate type, the flexor digitorum profundus, flexor pollicis longus, and pronator teres are affected, and the flexor digitorum superficialis and flexor carpi ulnaris are affected to a lesser degree. Intrinsic muscle paralysis and sensory neuropathy of the median and ulnar nerves are also present. The severe type involves ischemic damage to all of the flexors in the forearm, variable involvement of the extensors, and severe neurologic injury.\(^2,4,8\)

The main goal in the management of Volkmann contracture is to restore function; however, normal function of the upper extremity should not be expected.\(^2,4,8\) Affected muscles are exposed, and fibrotic or necrotic tissue is débrided. Thorough neurolysis of the median and ulnar nerves should be performed as well as tenolysis of scarred tendons. Isolated tendon lengthening is not recommended because it typically results in recurrent contracture and loss of grip strength.\(^2,4,8\) In persons with mild contracture, the flexor/pronator slide may be performed to improve hand position as well as function of the forearm flexors. Tendon transfer may be performed to improve specific dysfunction in persons with moderate contracture. Usually this involves use of an extensor tendon transfer; however, flexor tendons can be transferred in the case of dorsal contracture. Free muscle transfer, typically using the gracilis muscle, is necessary to improve function in persons with severe contracture.\(^12,13,16\)

**Summary**

ACS of the upper extremity is a potentially devastating condition that can lead to substantial loss of function. Compartment syndrome has many causes, and a high index of suspicion must be maintained. The final common pathway is a vicious cycle of increased tissue pressure with resulting cellular ischemia. All affected compartments in the upper extremity must be recognized, and thorough decompression must be performed in an expedient manner. Compartment syndrome is difficult to diagnose in some cases, particularly in obtunded patients and young children. Prompt diagnosis and emergent surgical decompression are essential to obtain a good clinical outcome and preventing a catastrophic result.

**References**

*Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 2-6, 9, 10, 14-20, 28, 31, 42, 46, and 47 are level IV studies. References 11-13, 23, 37, 44, and 45 are level V expert opinion.*

Citation numbers printed in **bold** type indicate references published within the past 5 years.


