Pressure, perfusion, and compartments: Challenges for the acute care surgeon

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I would like to thank President Mackersie and the American Association for the Surgery of Trauma (AAST) for this great privilege of giving a master surgeon lecture. It is a great honor and a humbling experience.

DEDICATION

Dr. William Stahl, my mentor at the Lincoln Medical & Mental Health Center in New York, who introduced me to the intricacies of trauma and surgical critical care and shaped my career, passed on at the end of last year. I gratefully dedicate this lecture to his memory.

The intricate relationships of pressure and perfusion occur on a regular basis in the many compartments, ubiquitously found in the human body. The ultimate sequels of an acute increase in pressure in these rigid compartments may range from limb loss to organ failures and death. The pathophysiology, however, is unclear, the diagnosis is uncertain, and the treatment is often delayed and incomplete.1-3 This area is of the greatest challenge to the acute care surgeon as a clinician to improve the current management principles. This is also a fertile field for game-changing research. This review will briefly cover some aspects of the more relevant and important compartments.

INTRACRANIAL COMPARTMENT SYNDROME

The rigid intracranial compartment and the effects of intracranial pressure (ICP) elevation are very familiar to the trauma surgeon. Simplistically, the outcomes, both short and long term, are poor when the ICP is elevated, the cerebral perfusion pressure (CPP) is either too low or too high. In a British study of 429 patients with head injury, mortality was significantly higher with ICP greater than 20 mm Hg and CPP lower than 55 mm Hg. Interestingly, severe disability increased even with elevated CPP of greater than 95 mm Hg. There was no difference between mean ICP and CPP in good/moderate and severe disability outcome groups.4 More recent studies are questioning whether the prognosis of traumatic brain injury is totally dependent on ICP and CPP.5-7 In an analysis of 20 patients with severe brain injury with real-time monitoring of focal cerebral blood flow (fCBF) and pressures, seven patients died. Five had technically adequate recordings before and as death supervened. Three patients had a pressure death (CPP decreased before fCBF decreased), and two patients had a nonpressure death (fCBF decreased before CPP decreased). The authors concluded that there are pathologic processes in the injured brain that do not directly involve increases in ICP and decreases in CPP. These authors, in a previous study, investigated the relationship between perfusion and fCBF in 23 patients. They noted that CPP did not have a linear relationship with fCBF over a range of 50 mm Hg to 150 mm Hg in patients who survived. In those who died, four of seven showed some indication of linearity. They concluded that in a normal brain, autoregulation of cerebral blood flow predominates, leading to the lack of an obvious relationship between perfusion pressure and flow. Interestingly, the onset of a pressure-flow correlation may be an early warning of this autoregulation and a clinical deterioration. A similar concept of looking beyond ICP and CPP was proposed by a Swiss group of investigators. They suggested that secondary brain injury may be better managed by multimodal brain monitoring, including brain tissue oxygen (PbtO2), cerebral microdialysis, and transcranial Doppler, to optimize cerebral blood flow and the delivery of oxygen/energy substrate.6,7 There, evidently, is much more to be discovered in the pressure-perfusion tale of the injured brain.

ORBital COMPARTMENT SYNDrome

The orbit is another closed compartment, bound anteriorly by the eyelids and orbital septum and posteriorly by the bony orbital walls. “Orbital compartment syndrome” with the characteristic protrusion of the eye and the limitation of lateral gaze is not so well known but can have disastrous consequences in a short time because of the occlusion of central retinal artery and the resultant ischemia of optic nerve (Fig. 1). It not only can be caused by retrobulbar trauma or hemorrhage but also can occur in patients with severe burns who are receiving massive resuscitation volumes. Of 13 patients with burns greater than 25% body surface area in one burn center, 5 had intraocular pressures greater than 30 mm Hg and required lateral canthotomy for decompression.8 Singh et al.9 reported similar results in a group of 29 patients with burns. They noted a 4.4% relative risk of the need for orbital decompression in patients receiving more than 8 mL of fluids per body surface area of burns. In such patients, it is imperative that ocular pressure be monitored. It is important to note that 60 minutes to 100 minutes of elevated intraocular pressure can result in orbital nerve ischemia and permanent...
blindness. A bedside lateral canthotomy is indicated for high pressures. An excellent review was published in 2009.10

THORACIC AND MEDIASTINAL COMPARTMENT SYNDROME

This is well known after prolonged cardiac surgery, again the effects of excessive volume infusion, leading to cardiac dilatation. The resulting reduction in cardiac output and subendocardial ischemia cause further reduction in cardiac output, and the vicious cycle continues.11 Any attempts at sternal closure causes a reduction of cardiac output and a drop in systolic blood pressure. In these cases, leaving the sternum unclosed for a delayed closure after a few days is the recommended approach. On some occasions, the syndrome may manifest itself a few days after the primary sternal closure. Increasing peak airway pressures and increasing hemodynamic instability should suggest the diagnosis. This phenomenon is also seen after trauma. For example, a young patient who had a gunshot wound of the chest causing a tangential laceration of the heart with cardiac tamponade had a prompt thoracotomy, relief of tamponade, and a suture of the laceration (Fig. 2). The resuscitation should have ended then. Overzealous fluid administration, however, contributed to a tremendous cardiac dilatation so that the pericardium and the chest could not be closed without compressing the heart and causing hypotension. The chest incision had to be left open with a temporary closure, using an Esmarch bandage sutured to the skin edges. It was closed subsequently, after a few days of diuresis. Three other cases were reported in the literature after chest trauma.12

ABDOMINAL COMPARTMENT SYNDROME

The abdomen as a rigid compartment and the pathophysiology of increased intra-abdominal pressure (IAP) were appreciated and well described approximately 150 years ago. Soon forgotten, the clinical challenges of intra-abdominal hypertension (IAH) confronted us with the advent of abdominal packing, damage-control surgery, and large-volume resuscitation of massively injured patients. It is now clear that the established abdominal compartment syndrome (ACS) and the associated organ failure are a late stage of unrecognized, untreated IAH (Fig. 3).13 16

Judicious use of the open abdomen, IAP measurement, attention to end points of optimal resuscitation, and monitoring of organ function have become the mainstays in the management of the sickest patients having “damage-control” laparotomy. Many lessons about the open abdomen have been learned in the last decade and, together with the advent of such innovations as negative-pressure therapy, it is now possible to accrue all the benefits and minimize the complications of an open abdomen approach.13 16

Many concepts were refined by the World Society of Abdominal Compartment Syndrome (WSACS), a group of international collaborators interested in the challenges of IAH and ACS. The society summarized the current knowledge in a monograph,13 developed consensus definitions, and made recommendations for medical and surgical interventions.14 Although the WSACS was denigrated at times as redundant and aggrandizing, it was very effective in raising awareness of the pathophysiology of IAH and ACS throughout the world, in all specialties of medicine and across patients of all age groups. These initiatives did produce spectacular results. Routine IAP monitoring and sagacious use of the open abdomen were shown

Figure 1. Orbital compartment syndrome from retro-orbital hemorrhage after trauma. Note the unilateral proptosis, limitation of downward gaze, and conjunctival edema (reproduced with permission from C. Robert Bernardino, MD, Review of Ophthalmology). Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

Figure 2. Thoracic compartment syndrome after a small, tangential gunshot wound of the heart (arrow). Overzealous resuscitation with crystalloids after pericardiomectomy and relief of tamponade caused severe cardiac dilatation. The chest could not be closed without compromising hemodynamics. A temporary closure with Esmarch bandage was necessary.
to lead to improved fascial closure rates, reduced prevalence of the full-blown syndrome of ACS, and enhanced survival, while minimizing such dreaded complications as enteroatmospheric fistulas. Furthermore, Balogh et al. reported that in severely injured patients, while IAH may be prevalent, ACS and organ failures could be prevented. The prospect of eliminating the morbid ACS is suddenly on the horizon! A more recent undertaking of WSACS, accomplished under the leadership of Kirkpatrick, is a systematic grading of all available evidence on the subject of IAH and ACS. Along with the 2013 update of consensus definitions and recommendations, this is presented in a massive document on the Web. An abridged version is published in Intensive Care Medicine.

POLYCOMPARTMENT SYNDROME

It is fascinating to note that all the compartments, namely, intracranial, intrathoracic and intra-abdominal, have an interplay, in some cases leading to a polycompartment syndrome. Scalea et al. noted that our therapeutic efforts to increase CPP in patients with concomitant head and multisystem injury may have adverse effects on pulmonary function. This will necessitate efforts to improve lung function by increasing ventilator support. The result may be a further increase in IAP and ICP. In some patients, this intracranial hypertension may be resistant to all conventional interventions, until abdominal decompression is performed to reduce IAP and, secondarily, the ICP. This clinical scenario has been dubbed the polycompartment syndrome (Fig. 4). Further support for the interplay between the compartments was shown by a porcine study from Greece. IAH significantly reduced cerebral and spinal perfusion pressures, concomitantly increasing interleukin 6, lactate, and tumor necrosis factor α in cerebrospinal fluid, suggesting the development of central nervous system ischemia. This effect was transient and reversible when perfusion pressures were restored to a level greater than 60 mm Hg, irrespective of the level of IAH, an alternative to abdominal decompression as a therapeutic option.

EXTREMITY COMPARTMENT SYNDROME

Compartment syndrome in the extremities is a particularly frequent and complex clinical entity. It can result from an increase in the contents of the many rigid compartments prevalent in the extremities. Examples include fractures, hematomata, soft tissue injury, crush syndrome, revascularization, exercise, fluid infusion, snake bite, nephrotic syndrome, myositis, and osteomyelitis. It is also caused by a myriad of causes of reduced compartmental volume such as after burns. Pathophysiology

Just as in other compartments, a small increase in volume can have a profound effect on the intracompartmental pressure. This increase in interstitial tissue pressure, exceeding the perfusion pressure, results in a narrowed A-V gradient, capillary collapse, as well as muscle and tissue ischemia. Ischemic skeletal muscle responds by the secretion of histamine-like substances. The resultant increased vascular permeability leads to leakage of plasma, sludging of capillary flow, and worsening of muscle ischemia. Myocytes undergo lysis; myofibrillar proteins escape into the interstitium, osmotically extract fluid, and increase the compartmental pressure further. Muscle ischemia worsens and perpetuates this vicious cycle (Fig. 5). Other scientists questioned this concept. They postulate the presence of a critical closing pressure (termed the \( P_{crit} \)), approximately 30 mm Hg, within the muscle compartments, which determines the level at which muscle blood flow decreases. This theory proposes that arterioles require high arteriole-tissue gradient to maintain patency and conditions causing extremity compartment syndrome (ECS) cause a reduction or removal of this gradient, leading to arteriolar collapse.

Regardless of the mechanism, the effect is ischemia-induced cellular destruction and alterations in muscle cell function.
membrane, leading to the release of myoglobin into the circulation. This rhabdomyolysis results in renal injury. The pathophysiology of ECS following vascular occlusion may differ slightly. Ischemia-reperfusion injury plays a dominant role. We will return to this later in the discussion of the role (or absence of it) of fasciotomy in vascular occlusion.

Prevalence
Tibial fracture accounts for approximately 10% of all cases of ECS and seems to be more common in open fractures. Vascular injury is the other frequent area. Feliciano et al. reported that 19% of patients with vascular injury required fasciotomy for ECS. Secondary ECS describes the syndrome developing without significant injury to the extremity itself and is an iatrogenic phenomenon occurring in massively injured patients receiving large-volume resuscitation (Fig. 6). In the first series from Grady reported by Tremblay et al., 10 of 11,996 patients and a mean of 3.1 extremities developed this complication in 10 upper and 12 lower extremities. Mortality in the group was 70%. In their follow-up series, these authors presented a similar group of very severely injured patients who received massive resuscitation but who were carefully monitored for compartment syndrome. The incidence of renal failure and death was reduced but was still substantial at 35%. Of the 46 extremities with ECS, 7 required amputation.

Diagnosis
The familiar cardinal p’s of ECS, namely, pallor, pulselessness, paraesthesia, pressure, poikilothermia, and palsy are more specific than sensitive. They are poor indicators of the syndrome, unless there are multiple symptoms present, especially in the appropriate clinical setting. Laboratory tests are often unhelpful. However, the relevant tests for rhabdomyolysis (Creatine phosphokinase levels, renal function, urine myoglobin levels, and urinalysis) may raise suspicion for ECS. Conventional radiographs, computed tomographic scans, and magnetic resonance imaging studies may be indicated in selected patients with compartment syndrome of the pelvis, thigh, and so on.

Compartment pressure (CP) monitoring is the most useful test for diagnosis. This can be performed bedside by several techniques, including the Stryker Quick Pressure Monitor Instrument, manometric IV pump method, and the slit catheter technique. The Stryker instrument has the advantage of simplicity. Normal resting CP is approximately 8 mm Hg in adults and slightly higher (13–16 mm Hg) in children. CP varies by location both within normal compartments and in the injured limb and with distance from the site of fracture, reasons for measuring at multiple sites within multiple compartments.

Spectroscopy
Other useful diagnostic techniques for the determination of ECS include methods to detect its sequelae of tissue ischemia. Near-infrared spectroscopy (NIRS) is a technique to determine tissue oxygen tension by a bedside monitor both noninvasively and continuously. An inverse correlation between CP and oxygenation and a correlation between perfusion pressure and oxygenation were demonstrated in an animal model. A human model of compartment syndrome showed that both tissue oxygenation and CP significantly correlated with a decrease in deep peroneal conduction, cutaneous peroneal sensitivity, and pain. NIRS was shown to be at least as good as CP monitoring for detecting developing ECS. Giannotti et al. published a series of case reports on patients who underwent fasciotomy, who showed that tissue oxygenation levels in patients with compartment syndrome were significantly lower than a matched control group with lower extremity injuries and no compartment syndrome. The measured tissue perfusion of the compartment syndrome group was also significantly lower than postfasciotomy values.

Multiple case reports have been published detailing the use of NIRS as an adjunct for continuous monitoring of lower extremity perfusion. Shuler et al. published a series of patients who underwent fasciotomies based on ICP monitoring with simultaneous NIRS monitoring. All compartments showed decreasing tissue oxygenation with decreasing perfusion pressure. NIRS has also been used to characterize changes in perfusion following tibial fracture in patients without compartment syndrome, as compared with subjects’ contralateral extremity and uninjured extremities in other controls. Of note, a lack of posttraumatic hyperemia (15% increase in tissue
oxygenation seen in injured extremities) was felt to actually represent a compartment syndrome even if oxygenation in the injured leg is equivalent to that in the uninjured extremity.41

Tissue oxygenation, as determined by NIRS, may help avoid fasciotomy. In a study of patients with clinical signs of compartment syndrome after revascularization surgery for lower limb ischemia, Arató et al.43 reported that measurement of compartmental pressure and NIRS-measured tissue oxygenation could be used to determine whether fasciotomy was needed. Patients with pressure less than 40 mm Hg and normal tissue oxygen saturation were treated conservatively.

Recently, resonance Raman spectroscopy, using excitation by a Krypton ion laser, was tested in our institution in animal models of hemorrhagic shock and resuscitation.44 Raman spectroscopy–derived sublingual oxygen tension (SmO2) was calculated as the ratio of the oxygenated heme spectral peak height to the sum of the oxymyoglobin and deoxyhemoglobin spectral peak heights. Measurements were compared with central venous oxygen tension (Scvo2) as well as with other indicators of oxygenation. SmO2 was significantly (p < 0.0001) correlated with Scvo2 (r = 0.80), lactate (r = −0.78), base excess (r = 0.80), and shed blood volume (r = −0.75). The technique was thought to show promise as a method to noninvasively monitor tissue oxygenation. In a subsequent study,45 the ability of resonance Raman spectroscopy to monitor tissue hemoglobin oxygenation (RRS-StO2) during hemorrhage was studied and compared with conventional invasive mixed venous (SmvO2) and central venous (ScvO2) hemoglobin oxygen saturation and NIRS tissue hemoglobin oxygenation (NIRS-StO2). The following receiver operating characteristic areas under the curve were obtained: SmvO2 (1.0), ScvO2 (0.994), RRS-StO2 (0.972), and NIRS-StO2 (0.611). RRS-StO2 seemed to have significantly better ability to predict shock based on elevated lactate levels when compared with NIRS-StO2. Future investigations should focus on the ability of RRC-StO2 to diagnose critical muscle ischemia from evolving ECS. Continuous monitoring of StO2 and CP with careful serial clinical examination in high-risk clinical situations must be studied in the future to allow early diagnosis of ECS.

Timely Fasciotomy

Unfortunately, none of these methods tell us (yet) what the critical CP is, which requires compartmental decompression, while all agree that early diagnosis for a prompt fasciotomy is important. The indication for fasciotomy was also an area clouded with lack of clarity. Some investigators recommend the so-called Delta P (diastolic blood pressure − compartmental pressure) with a critical range of 10 mm Hg to 30 mm Hg.33−35 Others use an absolute threshold of 30 mm Hg. Mubarak and Hargens46 argue for the consideration of the total clinical picture: they recommend fasciotomy in patients who are normotensive with positive clinical findings and CP greater than 30 mm Hg, when the duration of increased pressure is unknown or thought to be longer than 8 hours, in those who are uncooperative or unconscious with a CP greater than 30 mm Hg, and those with low blood pressure and a CP greater than 20 mm Hg. The timeliness of fasciotomy obviously is terribly important. Matsen and Krugmire20 suggest 2 hours to 4 hours of ischemia as causing functional impairment of muscles, which becomes irreversible after 4 hours to 12 hours. For nerve tissue, these critical times are 30 minutes to 45 minutes and 12 hours to 24 hours.

There are high-risk groups of patients and high-risk situations when critical muscle ischemia either goes undetected or occurs very early. Tissue that has been previously subjected to intervals of ischemia, patients under general anesthesia, sedated, or obtunded, and those receiving postoperative epidurals after tibial fracture fixation are especially susceptible to an insidious onset of critical ECS.2,3

The importance of timeliness of fasciotomy in vascular injuries was investigated by Farber et al.47 analyzing National Trauma Data Bank data of 612 patients who underwent fasciotomies, 543 early and 69 late (before 8 hours and after 8 hours of vascular repair). Patients in the early fasciotomy group had lower amputation rate and shorter total hospital stay compared with those in the late fasciotomy group. On multivariable analysis, early fasciotomy was associated with a fourfold lower risk of amputation and 23% shorter hospital stay. The role of fasciotomy in crush injuries of extremities was emphasized by two recent articles from the Middle East wars. In the first series,48 a total of 336 patients underwent 643 fasciotomies. The majority were to the lower leg (49%) and forearm (23%). Patients who underwent a fasciotomy revision had higher rates of muscle excision and mortality compared with those who did not receive a revision. Patients who underwent fasciotomy after evacuation had higher rates of muscle excision (25% vs. 11%), amputation (31% vs. 15%), and mortality (19% vs. 5%) compared with patients who received their fasciotomies in the combat theater, suggesting that revisions and delayed fasciotomies result in increased complications and mortality. The army, of course, went into a correction mode and did an education program designed to improve surgeon knowledge. Subsequent analysis49 showed that this resulted in higher fasciotomy rates, reduced revisions, and improved survival.

In the civilian setting, conditions obviously are much more favorable than the battlefield, and timely fasciotomy may be expected to be the rule rather than the exception. Several series, however, document the opposite. Feliciano et al.50 reported in 1988 on 125 fasciotomies (25 upper and 100 lower). Nineteen percent of lower extremity fasciotomies for vascular injuries were not performed at the primary operation, and 75% of amputations were from delayed or incomplete fasciotomies. In a review of 81 popliteal artery injuries (39 arterial and 42 combined), Fainzilber et al.50 performed primary fasciotomy on 53% of their patients. Of the 35 patients who did not have a primary fasciotomy, 6 required an amputation. In a retrospective review, Abouezzi et al.51 noted two amputations for extensive popliteal vascular injury despite primary fasciotomy. One amputation was the result of a delayed fasciotomy and failure of vascular repair. The overall amputation rate for popliteal injuries was 9%. They also observed that four of nine patients who had neurologic deficit in the extremity also had failure of vascular repair and delayed fasciotomy. Another multicenter civilian series from Canada52 documented significant delays in diagnosis and treatment in both traumatic and nontraumatic cases of ECS.
A decade later, a report\(^5\) from, arguably, the most distinguished trauma center in the country identified 83 cases of compartment syndrome over 7 years. Six percent had amputations, and 8% died, after predominantly orthopedic injuries. Eight percent had considerable morbidity from repeated debridement of dead muscle due to delayed fasciotomy. Of note, none had pressure monitoring. Another 7% had iatrogenic transection of superficial peroneal nerve during the performance of fasciotomy. The authors concluded that compartment syndrome remained a significant diagnostic and management challenge with great morbidity in terms of limb loss and neurologic outcome. They proposed a key pathway with a multidisciplinary approach for optimal outcome in these patients. Figure 7 is based on their recommendation.

Such honest reports of suboptimal therapy of ECS, presented from distinguished institutions without adornment or embellishment, are not frequent in the surgical literature. An accurate estimation of avoidable amputations and dysfunctional limbs may, hence, be an unattainable goal. Personal experience and privileged communications from around the country, however, substantiate delays in fasciotomy and the tragedy of limb amputations and organ failures in both orthopedic and vascular cases.

For vascular injuries in high-risk patients (those with prolonged ischemia time, significant preoperative hypotension, associated crush injury, combined arterial and venous injury, or the need for a major venous ligation in the lower limb), an early fasciotomy is recommended before arterial exploration.\(^5\)\(^4\) The role of fasciotomy in cases of ECS caused by vascular occlusion is even less clear.\(^5\)\(^5\)\(^–\)\(^5\)\(^7\) If ECS is diagnosed late, fasciotomy is of no benefit. In fact, fasciotomy probably is contraindicated after the third or fourth day following the onset of the syndrome. When fasciotomy is performed late, severe infection usually develops in the necrotic muscle. However, if the necrotic muscle is left alone and the compartment is not opened, it can heal with scar tissue. This may result in a more functional extremity with fewer complications.\(^5\)\(^5\)

This notion of delayed fasciotomy causing more harm than good was supported by this 1989 editorial from the distinguished Blaisdell,\(^5\)\(^6\) AAST past president, who was called the father of modern trauma care: “The question is, what factor is responsible for the increased compartmental pressure? When there has been trauma to a compartment and the cause of increased compartmental pressure is hemorrhage either into muscle or into a compartmental space, then few could argue the rationale for fasciotomy. When a limb has been revascularized after many hours of ischemia, the reason for the swelling is not quite so clear… I contend that we still do not have a clear understanding of the reason for the high compartmental pressures nor do we have the precise point at which fasciotomy will be more beneficial than harmful.” In a more recent communication elucidating the pathophysiology of skeletal muscle ischemia and reperfusion, Blaisdell\(^5\)\(^7\) expounds on the relative merits of fasciotomy versus anticoagulation in the ischemic extremity. “Only in this region will therapy be of any benefit, whether fasciotomy to prevent pressure occlusion of the microcirculation, or anticoagulation to prevent further microvascular thrombosis… In instances in which the process involves the bulk of the lower extremity, amputation rather than attempts at revascularization may be the most prudent course to prevent the toxic product in the ischemic limb from entering the systemic circulation.”

Despite these hazy areas, many other issues have clarity. At a critical pressure, perfusion goes down. This happens at much lower pressures and to a greater extent by previous shock and resuscitation. A vicious cycle of ischemia and end-organ damage is set in motion to culminate in ECS. We do not have a reliable test to diagnose ECS: clinical signs are late, and compartmental pressures and muscle oxygenation are useful but not specific. The result is a delayed diagnosis and treatment of ECS. Inevitably, the outcomes are poor, including neurodeficits, muscle necrosis, organ failures, death, and amputations. Limb loss comes at a great cost to the unfortunate patient. The projected lifetime health care cost for the patients who had undergone amputation had been noted to be three times higher than when treated with reconstruction ($509,275 and $163,282, respectively).\(^5\)\(^8\)

The goal of the acute care surgeon, therefore, should be first to prevent ECS by avoiding secondary syndromes. Aggressive crystalloid overloading or “fluid-creeper”\(^5\)\(^9\) should be avoided, since that seems to be the common denominator for many compartment syndromes. The next goal should be to pursue methods to diagnose compartment syndrome at its onset, so that compartmental decompression can be timely and reap the maximum benefit. Fasciotomy must be technically exquisite with long incisions and decompression of all four compartments in the leg. Iatrogenic nerve injury must be avoided.

It is perhaps time to borrow the graph (Fig. 3) from the story of ACS: define and emphasize the stage of compartmental hypertension, and pursue its diagnosis and treatment (Fig. 8) to eliminate progression of pathology to the established ECS and the potential for the loss of limbs, organs, and life.

In summary, the field of pressure, perfusion, and compartments has many gaps in knowledge. The current state of our management is tentative, with huge implications for patient safety. It is time to accept our imperfection, rise to the challenge, and generate innovative concepts in basic as well as clinical research. The acute care surgeon should intervene
timely and competently in compartment syndromes, paying attention and worrying constantly about the diagnosis.

DISCLOSURE
The author declares no conflict of interest.

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