Compartment Syndromes: Short-Term Outcomes

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17.1 Abdominal Compartment Syndrome

17.1.1 History and Definition

In the 1800s there began to emerge an understanding that elevated intra-abdominal pressure caused physiologic changes in several organ systems. Both Marey in 1863 and Paul Bert in 1870 demonstrated respiratory changes with rising intra-abdominal pressure. In 1890, Haase showed that intra-abdominal pressure increased with inspiratory effort. In 1890 also, Heinricius published that in cats and guinea pigs, intra-abdominal pressures of 27–46 cm of water caused death by interference of respiration [1].

By 1911, Emerson noted that elevated intra-abdominal pressure would "fatigue the diaphragm." Elevated intra-abdominal pressures would also cause "venous stagnation in the abdominal viscera" and thus diminish right-heart venous return. This resulted in "diminished [cardiac] output and fall in arterial pressure" [2].

In the modern era, Kron published in 1983 a series of postoperative patients that developed increased intra-abdominal pressure as well as new-onset renal failure.

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The patients' renal failure resolved with re-laparotomy and abdominal decompression [3].

We now recognize that abnormal abdominal pressures exist along a continuum, with maladaptive changes occurring at certain lower pressures and more severe ramifications at higher pressures. While pressures at which maladaptive changes can occur may vary from patient to patient, more significant than a single pressure value is the recognition of critical organ failure. Thus, with an improved understanding of intra-abdominal pressure, and standardization to measurements in mmHg instead of cm H_2O, the World Society of the Abdominal Compartment Syndrome (WSACS) has standardized the classification of intra-abdominal hypertension into [4]:

Grade I: IAP 12–15 mmHg
Grade II: IAP 16–20 mmHg
Grade III: IAP 21–25 mmHg
Grade IV: IAP >25 mmHg

Abdominal compartment syndrome itself is now defined as bladder-pressure measurements higher than 20 mmHg with new-organ failure.

17.1.2 Etiology and Pathophysiology

While elevated intra-abdominal pressure can have significant clinical repercussions, there were many direct causes that were quickly recognized to contribute to the phenomenon. Direct intra-abdominal pathologies include intraperitoneal causes, such as trauma, hemorrhage, and retained lap pads from intra-abdominal packing. Nontraumatic causes include malignancy, pancreatitis, ascites, ileus, bowel obstruction, and ruptured abdominal aortic aneurysm. It was quickly recognized that rapidly increasing space-occupying lesions within the abdomen can contribute to elevated intra-abdominal pressure.

It was recognized later that resuscitation can induce a "secondary abdominal compartment syndrome." In 1994, Greenhalgh and Warden published a series involving 30 children with burns [5]. Defining elevated intra-abdominal pressure as more than 30 mmHg, five patients had elevated intra-abdominal pressure during acute resuscitation, two of which required escharotomies. Seven patients developed elevated intra-abdominal pressures from sepsis, one of which required a laparotomy and another required decompression with an intra-abdominal catheter. In 1995, Burrows reported a trauma case in which intra-abdominal compartment syndrome resulted from management of an isolated extremity injury [6]. Fabian et al., in 1999, published the first large series of secondary compartment syndrome, finding that while 46 of 1216 ICU admissions required laparotomy for compartment syndrome, 6 of their patients had no intra-abdominal injuries [7]. Resuscitation in these six patients prior to decompressive laparotomy averaged 19 L of crystalloid and 29 units of packed red blood cells. They concluded that abdominal compartment syndrome

could occur without intra-abdominal injury. The World Society on Abdominal Compartment Syndrome recognized Secondary ACS in 2006 as abdominal compartment syndrome arising from conditions that do not originate in the abdominopelvic region [8]. Primary ACS, in contrast, was associated with injury or disease in the abdominopelvic region. They also recognized recurrent ACS as arising after previous surgical or medical treatment of primary or secondary abdominal compartment syndrome.

17.1.2.1 Cardiac Effects

The effect of intra-abdominal hypertension on cardiac function is multifactorial. Classically, it is accepted that increased abdominal pressure augments pressure on the inferior vena cava and reduces venous return to the heart; thus, cardiac output drops as predicted by the Starling curve dynamics. However, studies in patients as well as in an animal model have shown that as intra-abdominal pressure rises, central venous and wedge pressures rise [9, 10]. During this time, there is a decline in cardiac output paired with a rise in systemic vascular resistance. This suggests that many of the cardiac derangements and hemodynamic secondary to abdominal hypertension be more related to increased afterload. While decompressive laparotomy improves overall hemodynamics in patients with compartment syndrome, there are reports of hypotension in the immediate post-laparotomy period due to a rapid fall in systemic vascular resistance [11].

17.1.2.2 Renal Effects

As intra-abdominal hypertension progresses, there is decreased urine output and elevation in serum creatinine. Oliguria can be seen at intrabdominal pressures as low as 15 mmHg, while anuria can be seen at pressures of 30 mmHg. Timely decompressive laparotomy often results in rapid improvement in renal function. Several hypotheses have been proposed regarding the underlying pathophysiology for renal failure in this setting. One theory is that direct compression of the kidney causes a decrease in perfusion pressure and glomerular filtration rate. This was proposed in a 1982 study in dogs; intraperitoneal bags were inflated causing a decrease in cardiac output, renal blood flow, and glomerular filtration rate (GFR). Resuscitation improved cardiac output, but renal blood flow and GFR remained low, suggesting that the changes at the level of the kidney were not due to cardiac dysfunction [12]. This concept was challenged by another study showing that direct external renal compression did not affect GFR or renal artery blood flow [13]. Currently, the most commonly suspected mechanism is that compression of the renal vein reduces renal blood flow and induces renal failure. A study in a pig model showed that increasing pressure on the renal vein produced the expected decrease in GFR and renal arterial blood flow [14].

17.1.2.3 Pulmonary Effects

Elevated intra-abdominal pressure places resistance on the diaphragm and results in decreased pulmonary compliance. Elevated peak and plateau pressures are seen in patients undergoing volume-cycled mechanical ventilation. Patients undergoing

pressure control ventilation will exhibit decreased tidal volumes. With continued resistance from intra-abdominal pressure, hypercarbia and respiratory acidosis occur. While oxygenation may be better preserved than carbon dioxide exchange, many critically ill patients can also develop worsening hypoxia. Timely decompressive laparotomy results in improvement in lung mechanics, while extended exposure to barotrauma can result in acute respiratory distress syndrome.

17.1.3 Diagnosis

Early investigations into intra-abdominal pressure were confounded by different methods of obtaining an accurate pressure reading. Strictly speaking, true intraperitoneal pressure measurements would utilize intraperitoneal catheters, and such approaches were used by Winkler and Quirin [15]. While surrogate measures for intraperitoneal pressures include stomach, rectum, or IVC pressure, bladder pressure measurement has emerged as an effective, simple, and accurate way of indirectly measuring intra-abdominal pressure. This current methodology centers around the procedure described by Kron in 1983 [3]. After Foley catheter placement and drainage of the bladder, 50–100 mL of saline is instilled. A pressure monitor is then connected to the Foley catheter. Bladder pressure measurement has been validated in an animal study with a rabbit model, with intra-abdominal pressure modulated by a specifically placed intraperitoneal balloon. There was good correlation between intra-abdominal pressure and bladder pressure (correlation factor >+0.855 and $p < 0.001$) [16]. Later studies have demonstrated that over-instilling the bladder may produce inaccurate pressure values [17]. Current guidelines recommend instilling no more than 25 mL of saline in the bladder. In an effort to standardize measurements of bladder pressure, the World Society for Abdominal Compartment Syndrome (WSACS) also recommends expressing intra-abdominal pressure in mmHg, measuring at the end of expiration with the patient in the supine position, and zeroing the transducer to the level of the midaxillary line [4].

17.1.4 Current Treatment Recommendations

The World Society for Abdominal Compartment Syndrome recommends several maneuvers to treat intra-abdominal hypertension. Evacuation of intraluminal contents can be done with nasogastric tubes and rectal tubes. Patients with colonic pseudoobstruction can be considered for neostigmine. Patients with ascites as the main driver for their intra-abdominal hypertension can have drainage of their peritoneal fluid to reduce abdominal pressure. Abdominal wall compliance can potentially be improved with adequate sedation and analgesia, and if needed neuromuscular blockade. Optimal fluid balance should be achieved to minimize volume overloading [4].

Patients progressing to abdominal compartment syndrome, however, should be strongly considered for decompressive laparotomy, which remains the mainstay of

treatment for patients that develop abdominal compartment syndrome. Kron's 1983 seminal paper noted that all patients that underwent decompressive laparotomy had improvement in renal function. The four patients managed without surgery all continued in renal failure and ultimately died. Patients undergoing laparotomy for abdominal compartment syndrome invariably have improvement in their hemodynamic status and intra-abdominal pressure [3]. Promptness of decompression remains critical. In Fabian's 1999 series, it was noted that time to laparotomy was 3 h in survivors and 25 h in non-survivors. Thus, urgent decompressive laparotomy for abdominal compartment syndrome remains the rule with few exceptions [7].

17.1.5 Outcomes

Before a consensus approach was developed in 2006, the epidemiology of abdominal compartment syndrome was complicated by variable definitions of ACS. Despite a unifying clinical definition, ACS is a heterogenous inciting event, though most studies evaluating the incidence of ACS involve trauma patients. Furthermore, it appears that with changing philosophies and patterns in resuscitation, the incidence of ACS may be decreasing [18]. The prevalence of abdominal compartment syndrome in at-risk patients has ranged from 1 to 14%, with the incidence appearing to be lower in more recent studies [19–22]. It is now clear that when left untreated, abdominal compartment syndrome leads to tissue hypoperfusion, multisystem organ failure, and mortality. While intra-abdominal hypertension alone does not correlate with multiorgan failure, patients that progress to abdominal compartment syndrome can have mortality rates of 36% or more [23, 24]. Malbrain's 2004 multicenter trial showed that non-survivors had higher intrabdominal pressure, were older, and had worse APACHE 2 score [19]. Liver dysfunction and surgical disease process (as opposed to medical admissions) were more likely to be associated with mortality.

Surgical decompression of abdominal compartment syndrome is almost always effective, with improvement in hemodynamics, renal function, and pulmonary pressures. However, surgical decompression also carries a separate metabolic burden [25, 26] as well as reperfusion injury [27].

While many patients overcome these challenges, a recurring clinical goal is successful closure of the abdominal wound. Failure rates to close the abdomen can range from 20 to 78%, and many can develop severe morbidities [28–30]. One recent study showed that 24% of open-abdomen patients had one or more of a variety of complications, ranging from infection, recurrent abdominal compartment syndrome, entero-atmospheric fistula, and hernia [23]. In another multicenter trial from 2011, fistula formation was found in 7% of open-abdomen patients [31].

A concerted effort to close the abdomen as rapidly as possible reduces complications and mortality. In 2010, Cheatham described the change in approach within a single institution regarding the open abdomen [24]. Over the time period of the study, the center saw the adoption of WSACS guidelines and definitions for abdominal compartment syndrome, as well as a reduction in the threshold for laparotomy

in accordance with WSACS guidelines. Finding many patients with open abdomens, the institution developed a management algorithm with a focused effort to close the abdomen as early as possible. While severity of illness remained unchanged over the 6-year period, patient survival to hospital discharge increased from 50 to 72% ($p = 0.015$). There was also an improvement in primary fascial closure from 59 to 81%, and improvements in resource utilization. Chen's meta-analysis from 2014 confirmed that early fascial closure (defined as within 2–3 weeks) resulted in a reduction in mortality (12.3% vs. 24.8%, $p < 0.0001$) as well as complications (RR = 0.68, $p < 0.0001$) [32].

Early methods of temporary abdominal closure include sterilized intravenous fluid bags, PTFE sheets, Bogota bags, and gauze dressings [33]. All of these methods can be potentially augmented with negative dynamic retention sutures or Velcro-assisted closure ("artificial burr" or "Wittmann patch").

Since then, negative pressure wound therapy has arisen as a common method of managing the open abdomen. Barker in 2000 published a series of 112 trauma patients with open abdomens; the temporary closure method used a perforated polyethylene sheet as a barrier against the viscera, which was then covered in a moist towel and suction tubing, and finally covered by an iodophor-impregnated adhesive polyethylene sheet [34]. 55.4% of patients had primary closure, while 22.3% had a repair with absorbable mesh. Complication rates included 19.6% who died before abdominal closure was performed, 4.5% with enterocutaneous fistula, and 4.5% with intra-abdominal abscess formation. While the Barker wound closure or a modification of this method is utilized in many centers, now purpose-built commercial wound vacs such as the ABThera are widely available.

There have been many studies seeking to elucidate the best method of temporary abdominal closure. The data, however, are heterogenous. A 2008 meta-analysis suggested that high fascial closure rates were found with Velcro closure (90%), dynamic retention sutures (85%), and wound vac (60%) [35]. A 2012 meta-analysis suggested that the highest fascial closure rate was found with Velcro closure (78%), dynamic retention sutures (71%), and wound vac methods (61%) [36]. Another 2012 meta-analysis with differing methodology suggested that the use of sequential fascial closure to the abdominal wound vac had a higher fascial closure rate [37]. The most recent systematic review published in 2016 compared negative pressure therapy vs. standard temporary closure. There was no difference in fascial closure (63.5% vs. 69.5%, $p = 0.57$) and enterocutaneous fistula rate (2.1% vs. 5.8%, $p = 0.57$). However, the negative pressure wound therapy group did have reduced mortality (28.5% vs. 41.4$, $p = 0.03$) and decreased ICU length of stay [38].

Another technique to augment abdominal negative pressure therapy is direct peritoneal resuscitation (DPR). DPR techniques instill peritoneal dialysate solutions into the abdomen while a negative pressure dressing evacuates excess fluid. Animal studies demonstrated improved visceral blood flow even while visceral edema was reduced [39]. In 2010, Smith et al. published a series of open-abdomen trauma patients, with 20 patients utilizing DPR against 40 control patients (control patients did not have a standardized abdominal wound management technique) [40]. Time to definitive abdominal closure was improved with the DPR group (DPR

4.35 ± 1.6 days versus control 7.05 ± 3.31; $p = 0.003$). The DPR group also had a high rate of primary fascial closure and decreased 6-month ventral hernia rate. While abdominal closure method was not standardized within the control group, subgroup analysis comparing DPR against controls utilizing a Velcro closure technique confirmed decreased time to definitive closure (DPR 4.4 ± 1.7 days versus Velcro 6.4 ± 1.3, $p = 0.003$). While DPR shows promise in improving fascial closure rates and decreasing complications associated with the open abdomen, further prospective studies are needed.

Complications surrounding the open abdomen in abdominal compartment syndrome
Infection (12%)
Recurrent abdominal compartment syndrome (6%)
Bleeding (6%)
Entero-atmospheric fistula (3%)
Hernia (15%)
Mortality (36%)
Efforts to mitigate complications may include
Protocolized effort to close the abdomen as rapidly as possible
Use of negative pressure wound therapy
Velcro closure
Dynamic retention sutures
Direct peritoneal resuscitation

Complications adapted from De Waele JJ, Kimball E, Malbrain M, Nesbitt I, Cohen J, Kaloiani V, Ivatury R, Mone M, Debergh D, Björck M. Decompressive laparotomy for abdominal compartment syndrome. Br J Surg. 2016 May;103 (6):709–715. doi: https://doi.org/10.1002/bjs.10097

17.2 Muscle Compartment Syndrome

17.2.1 Pathophysiology and Diagnosis

Compartment syndrome occurs when swelling within a muscle compartment causes tissue ischemia. While commonly associated with bony injury or reperfusion injury after prolonged ischemia, there are a variety of other causes including crush injuries, burns, and electrocution. Many patients present with the syndrome in the lower leg, and in one series this was most associated with the lower leg [41]. However, virtually any muscular compartment of the body can be at risk, including the hands, forearm, upper arm, buttocks, and thighs.

Elevation in compartment pressures exceeds capillary filling pressure, resulting in muscle ischemia. While local muscle ischemia can carry immediate morbid complications, systemic illness can result from compartment syndrome. This includes rhabdomyolysis and renal failure. Renal failure in the critically ill patient is a significant marker for mortality and other complications.

Classically, the diagnosis of compartment syndrome is clinical. The historical six Ps of compartment syndrome are pain, pallor, pulselessness, paresthesia, paralysis, and poikilothermia [42]. While pain out of proportion is an early sign, and paresthesia can result from nerve compression as compartment edema increases, waiting for

all the signs to develop can result in irreversible muscle injury and systemic organ failure. Indeed, irreversible muscle ischemia is thought to occur 8 h after loss of perfusion [43].

While there is no consensus for a diagnostic algorithm for muscle compartment syndrome, clinical suspicion based on history and physical exam findings is usually used. To confirm clinical suspicion in borderline cases, compartment pressure measurements can be used. Most typically, a needle connected to a pressure transducer is used, with many considering a compartment pressure between 30 and 50 mmHg as critically high. Others advocate the use of compartment perfusion pressure (MAP—compartment pressure), with a delta-*P* of less than 30 mmHg thought to be concerning [42]. An adjunct under consideration to improve the rapid diagnosis of extremity compartment syndrome is near-infrared spectroscopy, which measures tissue oxygenation up to 3 cm deep from the skin [44]. Another option is measuring intramuscular glucose concentration, which diminishes as compartment syndrome develops [45]. The role of these adjuncts in the diagnosis of compartment syndrome, however, remains unclear.

17.2.2 Treatment

Treatment of compartment syndrome centers on fasciotomy, the surgical release of the affected muscle compartments. Timing is critical, as worsening time markers allow for increased muscle death and systemic complications.

In patients with rhabdomyolysis, medical therapy includes aggressive hydration to promote renal perfusion and to dilute myoglobin levels, prevention of myoglobin deposition in the renal tubule via alkalization of the urine, and intravenous mannitol for renal vasodilatation and free radical scavenging.

In the past, fluid resuscitation as high as 1.5 L NaCl 0.9% per hour has been advocated [46]. More recently, to prevent complications of overload such as abdominal compartment syndrome or respiratory failure, more modest goals often accepted are 3–6 L in the first 24 h with additional volume dependent on hemodynamic and urinary output parameters [46].

Mannitol is thought to protect against rhabdomyolysis by its free radical scavenging action and may also prevent renal failure by increasing the volume passing through the renal tubule [47]. Another adjunct is bicarbonate. Intravenous bicarbonate results in alkalization of the urine, a goal derived from laboratory data showing that only 4% of myoglobin aggregates at urine pH above 6.5.

17.2.3 Outcomes

Due to the time-critical nature of fasciotomy, one focus on improving outcomes is the rapid identification and rapid surgical decompression of affected muscle compartments. Diagnosis of compartment syndrome still hinges on clinical suspicion; measurement of spot compartment pressures can be used to

confirm clinical suspicion, but there is little data to support that it changes outcomes. Indeed, compartment pressures can have false negatives, or an incorrect compartment checked, giving a false sense of clinical stability. Adjuncts to diagnosis, including intramuscular glucose and near-infrared spectroscopy, remain unproven [48].

What is clear, however, is that there are numerous studies confirming that early fasciotomy is effective. In 1984, Rorabeck noted that if fasciotomies were done within 24 h, a good result was "almost always achieved" [49]. In 1976, Sheridan noted that in patients undergoing fasciotomy within 12 h, 68% had normal function compared to only 8% of those undergoing late fasciotomy [50].

More recent data still emphasizes the need for early fasciotomy. Hope noted in 2004 that in patients without fracture, the suspicion for compartment syndrome can be lower and the diagnosis can be delayed. In the setting of patients without fracture, there was greater delay and 20% of these patients had muscle necrosis requiring debridement. In comparison, in patients with a fracture, only 8% needed debridement [51]. In the military population, where medical evacuation can delay fasciotomy, significant morbidities were noted. In 2008, Ritenour noted that soldiers that underwent fasciotomy after medical evacuation (vs. fasciotomy in the combat theatre) had more instances of muscle debridement (25% vs. 11%), higher rates of amputation (31% vs. 15%), and elevated mortality (19% vs. 5%) than patients who had fasciotomies in the combat theatre ($p < 0.01$) [52].

While it is thought to be better to perform an unnecessary fasciotomy than a late fasciotomy, surgical decompression still carries its own risks such as chronic wounds, delayed healing, need for skin graft, pain, nerve injury, and muscle weakness [48]. Dermatotraction techniques such as the "Jacob's ladder" have been demonstrated to assist with wound closure and reduce need for skin grafting [53].

For patients with rhabdomyolysis, modulating outcomes with medical therapy remains unclear. While mannitol is commonly used by many clinicians for rhabdomyolysis in the setting of compartment syndrome, there are no randomized trials involving the use of mannitol. Similarly, the use of intravenous bicarbonate is controversial. In the largest study to date of compartment syndrome in trauma patients, bicarbonate and mannitol together was compared with patients who did not receive combination therapy. The combination did not prevent renal failure or dialysis or reduce mortality [54].

17.2.3.1 Hand

One of the main complications for hand compartment syndrome is the development of hand contracture as ischemic muscle becomes necrotic and eventually fibrotic. There is currently scarce literature regarding functional outcomes in these patients. In a retrospective review by Oulette in 1996, 4 of 19 patients were noted to have poor hand function. Time from diagnosis to treatment for these patients was more than 6 h. Two patients eventually required an amputation. Thirteen had normal function, but some required further surgery to facilitate wound healing and ameliorate nerve compression [55].

17.2.3.2 Forearm and Upper Arm

Forearm compartment syndrome requires a high clinical suspicion. Kalyani's systematic review from 2011 showed a 42% complication rate. Neurologic deficit was the most common complication (20.9%). Other complications were contracture (9.3%), crush syndrome (4.7%), gangrene (2.3%), Volkmann's ischemic contracture (2.3%), and Sudeck's algodystrophy (2.3%) [56]. Outcomes data is less forthcoming in upper arm compartment syndrome due to its exceeding rarity, but the principles of timely fasciotomy remain. A series by Duckworth in 2012 showed that patients with forearm fasciotomies delayed by more than 6 h were more likely to have complications (most commonly neurologic deficit and contractures) [57].

17.2.3.3 Gluteal Region

Gluteal compartment syndrome is exceedingly rare, often creating a delay in diagnosis. In addition to muscle necrosis and complex morbid wounds, sciatic nerve palsy can develop from untreated compartment syndrome in this area [58].

17.2.3.4 Foot

Management of foot compartment syndrome has some controversy, with nine different foot compartments and fasciotomy presenting potential morbidities [59, 60]. There is data that suggest no difference in motor, sensory defects, and pain between patients who undergo fasciotomy versus those that do not [61]. Despite this, fasciotomy remains a mainstay of treatment. Lokiec noted that the most common complications were neurologic defects (52%), toe contractures, (12%), and amputations (12%) [62]. A 2009 systematic review aggregating 39 patients showed that only 10% were able to return to work [63]. More recent data from 2015 showed that 79% were able to return to work [64].

17.2.3.5 Lower Extremity

Due to the relative prevalence of compartment syndrome of the lower extremity in comparison to other compartments, thigh and calf compartment syndrome remains the prototypical example of extremity compartment syndrome [41]. In a 2016 review, acute kidney injury was found in 2.4% of lower extremity compartment syndrome. 12.9% of patients required amputation. 10.2% had lower extremity pain, foot numbness was noted in 20.5%, and a foot drop was found in 1.2%. 69% of patients were able to return to employment [65].

Complications surrounding muscle compartment syndrome
Delay in diagnosis
Rhabdomyolysis with or without renal failure
Contracture
Nerve damage
Tissue necrosis
Mortality
Efforts to mitigate complications may include
Early diagnosis with assistance of pressure monitoring
Near-infrared spectroscopy (experimental)
Intramuscular glucose concentration (experimental)
Early fasciotomy
Adequate resuscitation with possible use of adjuncts such as mannitol and bicarbonate

17.3 Thoracic Compartment Syndrome

17.3.1 Pathophysiology

Thoracic compartment syndrome is an exceedingly rare condition that has been reported after pediatric and adult cardiac surgery, and less commonly seen after trauma. Postoperative myocardial edema or cardiac dilatation, combined with chest wall and mediastinal edema, leads to compression of the heart, diminished diastolic filling, and thus decreasing cardiac output [66]. The condition can occur hours to days after chest closure, and if left untreated can lead to cardiovascular collapse. Diagnosis relies on a high index of suspicion in the setting of cardiac tamponade like physiology.

17.3.2 Management

Temporary closure of the chest can be achieved with a synthetic material, such as polytetrafluoroethylene, which is sutured to the sternal and skin edge [67]. Another option is skin closure without sternal re-approximation [68]. In addition, chest tubes can be used to stent open the sternum and thus elevate the sternum above the heart [69]. In one case report, a sternal traction device was used to maintain the sternum in an open position [70].

In one study in neonates, successful closure of the sternum was still noted to cause some increase in pulmonary arterial pressure, left and right atrial pressure, and peak airway pressure [71].

17.3.3 Outcomes

In the pediatric patient population, a series of 113 patients with prolonged open sternotomy showed a 36.2% mortality rate, versus 5.4% in patients with primary sternal closure. Mortality was higher in patients with low cardiac output after cardiopulmonary bypass, in those that needed a circulatory assist device, and in those that developed postoperative tamponade requiring reopening of the sternum in the ICU. Primary cause of mortality was heart failure, illustrative of the patient population. Other causes of death were pulmonary hypertension, multiorgan failure, and intracranial hemorrhage. Only one patient had mediastinitis [69].

A 1996 series demonstrated a mortality rate of 36.6% in 123 adult patients who underwent cardiac surgery and required prolonged open sternotomy. Need for an intra-aortic balloon pump postoperatively was associated with greater mortality (46.3% vs. 16%, $p < 0.01$). Other reported complications included superficial sternal wound infection (1.6%), mediastinitis (0.8%), and sternal dehiscence (2.4%) [72].

An earlier series in 1992 had a higher rate of mediastinitis in the open chest group (4–6%), but this was still within the range of the overall cardiothoracic patient group (0.15–5%) [73].

Complications surrounding the open chest in thoracic compartment syndrome
Superficial sternal wound infection (1.6%)
Mediastinitis (0.8%)
Sternal dehiscence (2.4%)
Mortality (36.6%)
Efforts to mitigate complications may include
Temporary chest closure with synthetic material (such as polytetrafluoroethylene)
Chest wound skin closure without sternal re-approximation
Use of chest tube to stent open sternum above the heart

Complication rates adapted from Christenson JT, Maurice J, Simonet F, Velebit V, Schmuziger M. Open chest and delayed sternal closure after cardiac surgery. Eur J Cardiothorac Surg. 1996;10(5):305–11

17.4 Intracranial Hypertension

17.4.1 Pathophysiology

In the 1700s, Scottish physician Alexander Munro and Scottish surgeon George Kellie helped form the hypothesis that models intracranial pressure today. The skull is a fixed volume, and space must be shared by the brain parenchyma, blood, and cerebral spinal fluid. Any increase in any of these components results in elevated intracranial pressure [74]. The underlying disease process is broad, with the phenomenon of increased pressure described as intracranial hypertension. However, we will focus on medical therapies for intracranial hypertension in the setting of trauma.

17.4.2 Outcomes Based on ICP Monitoring

Intracranial pressure monitoring began with Lundberg in 1964, who implanted a ventricular catheter in 30 patients with traumatic brain injury [75]. They proposed that ICP values can be used to guide TBI management. In 1977, Miller's series of 160 patients identified that an ICP of more than 20 mmHg correlated with poor outcomes (defined as severely disabled, persistent vegetative state, or death) [76].

Today, medical options to modulate intracranial pressure include osmotic agents (hypertonic saline, mannitol), traditional sedatives (propofol, midazolam, dexmedetomidine), and barbiturates [77].

Hypertonic saline generates an osmotic gradient across the blood-brain barrier to decrease ICP. The agent demonstrates rapid onset and relatively long-lasting effects, as much as 12 h in some patients. In 2014, Colton showed that patients with ICP decreased for at least 2 h with the use of hypertonic saline had decreased mortality and improved functional outcomes [78]. Mannitol's use as an osmotic diuretic has a long-standing history in reducing ICP, though the agent can also cause hypotension. Studies comparing hypertonic saline to mannitol suggest that hypertonic saline may have a more dramatic and sustained reduction in ICP [77]. However, specific outcomes data is lacking.

Sedatives such as propofol and midazolam appear to decrease ICP similarly. Propofol may have added benefit against cerebral edema, while midazolam has anti-epileptic properties. Dexmedetomidine has been shown to reduce the amount of hypertonic saline or mannitol needed to maintain ICP within normal range [77].

Barbiturates are used as a second-line therapy in patients with refractory ICP. However, need for barbiturate use is associated with poorer outcomes, which is likely a reflection on the disease burden in patients with refractory intracranial hypertension [77].

Despite the ubiquity of ICP-guided management in the setting of TBI, its effectiveness remains unclear. Shafi's 2014 multicenter retrospective study on ICP therapy suggested that adherence to guidelines was associated with reduced mortality (OR 0.88; 95% CI 0.81–0.96, $p < 0.005$) [79]. In contrast, Cremer's 2005 study compared two well-matched trauma centers in the Netherlands: one that utilized ICP measurements, and the other that utilized CT scan and exam findings. The study found that while the ICP-focused center used more sedatives, barbiturates, vasopressors, and mannitol, there was no difference in mortality [80]. The only randomized trial data on ICP management comes from the 2012 Benchmark Evidence from South American Trials: Treatment of Intracranial Pressure (BEST:TRIP). Patients were randomized to ICP-guided management vs. treatment-guided CT imaging in conjunction with physical findings. Ultimately, 6-month mortality was similar in both groups (39% in ICP monitoring vs. 41% in imaging/exam, $p = 0.60$) [81].

It should be noted that these trials are not precisely ICP vs. no-ICP groups, but a comparison of TBI management guided by ICP vs. imaging and findings [74]. Ultimately, additional data are needed to accurately describe the optimal method of TBI management.

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