The Compartment Syndrome of the Abdominal Cavity: A State of the Art Review

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Abdominal compartment syndrome gains increasing recognition. It impairs physiology and requires treatment. It occurs more commonly with acute rather than chronic abdominal hypertension. Functional impairments involve the cardiovascular system, respiratory system, hepatic, renal, and gastrointestinal function, and intracranial pressure. Abdominal hypertension decreases venous return, increases systemic vascular resistance and intrathoracic pressure, and therefore reduces cardiac output. It also adversely affects cardiovascular monitoring. In the presence of increased abdominal pressure, atelectasis and pneumonia are likely to develop and impaired ventilation may lead to respiratory failure. Also, blood flow to the liver and kidney may be reduced, resulting in functional impairment of both organs. The adverse effects on gastrointestinal function result from impairing lymphatic, venous, and arterial flow. Anastomotic healing may become a problem under these circumstances. Decreased venous return through the inferior vena cava in obese patients may lead to venous stasis ulcers and hemorrhage. The correlation of increased intracranial pressure and intra-abdominal pressure may be a problem for trauma patients with simultaneous injuries to the head and the abdomen. There are three severity grades of increased intra-abdominal pressure: Acute sustained elevation of intra-abdominal pressure above 20 mmHg is called mild abdominal hypertension. Physiologic effects are generally well compensated and usually clinically nonsignificant. Nonoperative therapy may be required. Moderate hypertension is defined as sustained elevation of 21–35 mmHg. Therapy is generally necessary. Surgical abdominal decompression may be critical. Severe hypertension or abdominal compartment syndrome is defined as sustained elevation above 35 mmHg. Operative decompression is always indicated. The gap between the abdominal wound edges must be temporarily covered to prevent fascia retraction and formation of a huge hernia. All detrimental effects of elevated intra-abdominal pressure and the methods and benefits of its decompression have been well studied, both in the laboratory and in clinical practice. Diagnostic suspicion may be confirmed with objective measurements of intra-abdominal pressure to select patients who may benefit from decompression. Operative decompression is achieved by abdominal fasciotomy and covering the fascial gap with mesh made of Marlex®, Gore-Tex®, silastic, or by a Velcro-like closure mesh (artificial bur). All meshes help to effectively decompress the abdomen. The artificial bur offers further advantages by permitting successive reapproximation of the fascia until final fascial closure, and avoiding the fistula and hernia formation seen with the other meshes.

Introduction and History

The abdominal cavity responds to a volume increase in any of its contents with abdominal hypertension. Elevated intra-abdominal pressure (IAP) may profoundly impair physiology and organ function because the abdominal wall’s compliance is limited. Once a critical threshold volume is reached, relatively small increments of volume are associated with relatively enormous pressure augmentations that quickly lead to decompression (Figs 1–4) [1]. Excessively increased IAP may result in total loss of function and may lead to death.

As early as 1876, E. C. Wendt noted the reduced urinary flow in the presence of abdominal hypertension [2]. When surgeons started treating peritonitis operatively, they were concerned about the “enormous pressure increase that often precluded abdominal closure” [3]. In 1911 Emerson introduced his readers to a series of elegant experiments with the statement that “pressure conditions which exist within the peritoneal cavity” had received insufficient attention [4]. Not much has changed since and the topic is not covered in current textbooks of surgery and critical care.

Nevertheless, abdominal hypertension was the subject to excellent reviews [5–10], and since 1959 numerous authors have described the negative effects of abdominal hypertension on the function of almost every organ system [4,6–9,11–15]. We learned that increased IAP may also deeply impair cardiovascular, pulmonary, hepatic, renal, and even central nervous system function and reduce perfusion to the gut and its organs [15–26]. Impaired intestinal perfusion from abdominal hypertension may be a critical factor in anastomotic healing and organ perfusion after transplant. It probably plays a role in many of the organ dysfunctions that are unsatisfactorily explained. Examples may be colon
ischemia, acalculous cholecystitis, or postoperative pancreatitis and some forms of ischemic bowel. In the 1980s we saw reporting of the benefits of abdominal decompression [27–29].

The first device that was specifically designed to temporarily decompress the abdominal cavity is the artificial bur. It became available in 1988 and was presented at the 1990 annual meeting of the Eastern Association for Trauma. Later various aspects of its use were published [30–37], while surgeons started recognizing abdominal hypertension and the abdominal compartment syndrome (ACS) as independent pathologic conditions from various causes that may lead to dysfunction of physiology [38–45]. This overview summarizes current knowledge about the causes, consequences, and treatment options of intra-abdominal hypertension.

**Definitions**

The current literature uses terms such as abdominal compartment syndrome (ACS) [29,38,40,41,46,47], abdominal hypertension (AH) [48,49], and increased intra-abdominal pressure. While the term “ACS” acknowledges the abdominal cavity as a closed space and “syndrome” addresses the associated pathology, the term “abdominal hypertension” is less precise and simply denotes pressure increases above normal. The definitions used are listed below. Of the various grading systems proposed, the simple grading system (see p. 203) is preferred because it may guide therapy.

**Compartment Syndromes in General.** A compartment syndrome is a condition in which increased pressure in a confined anatomic space adversely affects the function and viability of the

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Fig 1. Protruded bowel in abdominal compartment syndrome. There is massive abdominal edema in a trauma patient after laparotomy and fluid resuscitation for hemorrhagic shock. The abdominal content is covered with the artificial bur or STAR closure device. Although each sheet of the artificial bur is 20 cm wide, almost the width of both sheets combined was required to cover all the protruded bowel and omentum. The hook sheet consists of polypropylene micromushrooms (dark) that cling into a white loop sheet consisting of a meshwork of polyamide and polypropylene loops.

Fig 2. Relationship between intra-abdominal pressure and (A) abdominal wall compliance and (B) intra-abdominal volume increase [1].
and connected extraperitoneal organ systems. It may improve with nonoperative therapy.

**GRADES OF ABDOMINAL HYPERTENSION.** Pressures below 10 mmHg are normal. Short pressure increases with coughing, Valsalva maneuver, defecation, and weight lifting are functionally normal too. The patients’ premorbid physiologic reserves may severely compound the effects of pressure elevations on the above-mentioned functional impairments.

For practical purposes, one should differentiate between three intensity grades of increased IAP [46]. The crucial time frame to more precisely define the three stages is not yet known. Until further research is available, a pressure elevation will be called “sustained” if it is present for longer than 6 hours.

**Mild abdominal hypertension.** Sustained acute elevation of 10–20 mmHg. Physiologic effects are generally well compensated and thus are usually clinically nonsignificant. Nonoperative therapy may be required.

**Moderate abdominal hypertension.** Sustained acute elevation of 21–35 mmHg. Therapy is generally necessary. Intervention such as operative abdominal decompression may be critical.

**Severe abdominal hypertension.** Sustained acute elevation greater than 35 mmHg. Operative abdominal decompression is always indicated. Further pathologic conditions associated with increased IAP can be classified into acute and chronic abdominal hypertension.

**Forms of Abdominal Hypertension**

**Acute abdominal hypertension.** Acute abdominal hypertension is a pathologic condition of temporarily increased abdominal hypertension that may

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**Fig 3.** Increased intra-abdominal pressure in a patient with intra-abdominal infection. The abdomen was closed with three pairs of retention wires. Note the pressure necroses in the skin underneath the plates that anchor the retention wires. The bulging abdominal content provides further evidence for increased intra-abdominal pressure.

**Fig 4.** Hemodynamic changes seen with an increase in intra-abdominal pressure [60].
progress to ACS requiring operative decompression. Examples are diffuse peritoneal inflammation seen in diffuse peritonitis (Figs 3 and 13) [32,50], intestinal obstruction [51], ruptured abdominal aneurysm [29,40], and peritoneal edema following resuscitation for abdominal trauma; hepatic and retroperitoneal hemorrhage (Fig 1) [33,52–55]; and even extraperitoneal trauma requiring massive resuscitation [56]. A typical scenario occurs in a patient with diffuse peritonitis or in a multiple trauma patient who receives a large volume of fluid for resuscitation, causing an increase in interstitial fluid volume. The ensuing visceral and retroperitoneal edema is aggravated by shock-induced visceral ischemia. The closed abdominal wall initially contains the oncotic pressure of the edematous peritoneum. Once the abdomen is opened and counterpressure released, the edema may fully expand to grotesque clinical situations (Fig 1). Theoretically, 1 ml of peritoneal thickening may contain 15–18 L of fluid (see below). Closing the abdomen under these conditions becomes impossible.

Forcefully closing the edematous abdominal wall over the protruding abdominal contents will result in extreme tension, and already impaired functions will deteriorate. Positive pressure ventilation becomes necessary to maintain satisfactory oxygenation, but it further increases IAP [51]. Abdominal packing to control severe intra-abdominal hemorrhage may temporarily compress mesenteric veins, obstructing venous return and further increasing the edema [33,57].

Abdominal hypertension is further seen in 18% of elective laparotomies (exploratory laparotomies, upper and lower gastrointestinal, and aortic operations) and in up to 40% of emergency laparotomies [39,48].

**CHRONIC ABDOMINAL HYPERTENSION.** Chronic abdominal hypertension is a pathologic condition of lasting increased abdominal hypertension that may impair physiologic function and which may benefit from decompression. Examples are tense ascites [1,58] and congestive heart failure [1], large abdominal tumors, chronic ambulatory peritoneal dialysis, pregnancy [46], morbid obesity, and associated problems. Some complications that are ameliorated after successful therapy of morbid obesity may be directly related to normalization of IAP. Examples are hypertension, obesity–related hyperventilation syndrome, gastroesophageal reflux, stress overflow urinary incontinence, chronic vascular stasis ulcers, pseudotumor cerebri and cephalgia, and recurrent incisional hernia. Functional impairment from chronically increased IAP in pregnancy may be the pathogenic link for pregnancy-related diseases.

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**Measurement of IAP**

**Pressure Definition and Units.** Pressure is the force applied uniformly over a surface. It is measured as force per unit of area. At 0°C (32°F), standard sea-level pressure (1 standard atmosphere) is 1.030 kg/cm² (14.7 lb/in²), which is equivalent to a column of mercury (Hg) of 760 mm (29.92 in) in height. One mmHg then is 1/760 atm = 1.3335 mb = 1 torr = 1.36 cm H₂O, and 1 cm H₂O ≈ 0.74 mmHg. Although the modern SI definitions propose pascal as a standard measure of pressure in clinical practice, mmHg and torr are widely used. The torr is equal to approximately 1.316 × 10⁻³ atm or 1.333 Pa.

**Methods of Measuring IAP.** IAP can be measured by direct and indirect methods. In many earlier experiments, it was measured directly through a metal cannula or a wide bore needle inserted into the peritoneal cavity and attached to a saline manometer [4,6–9]. Indirect methods became popular when IAP was monitored clinically for treatment purposes and as a criterion for abdominal reexploration [27].

**Direct Methods.** The preferred method in numerous experimental studies has been direct measurement using an intraperitoneal catheter connected to a pressure transducer [22,23,59–62]. Occasionally an inflatable bag was placed into the abdominal cavity to produce and measure elevated pressure [63]. During laparoscopic procedures an automatic electronic insufflator provides continuous monitoring of pressure [64,65].

**Indirect Methods**

**INFERIOR VENA CAVA PRESSURE.** Transfemorally measured pressure in the infradiaphragmatic vena cava correlates directly with IAP [59,66]. Pressure changes in the supradiaphragmatic vena cava are less pronounced. As IAP increases to 40 torr, inferior vena cava (IVC) flow is reduced from more than 1,000 ml/min to about 500 ml/min [59].

**INTRAGASTRIC PRESSURE.** IAP can be measured by water manometry via a nasogastric or gastroscope tube [29,67,68]. Gastric pressure is determined by inhaling 50–100 ml of water through a nasogastric tube into the lumen of the stomach. The proximal end of the open tube is held perpendicular to the floor. The distance from the water level to the mid-axillary line is taken as the IAP in cm H₂O (1 cm H₂O = 0.74 mmHg). Pressure thus measured approximately correlates with pressure measured by
Transvesical Technique. A gastric tonometer with a balloon attached is introduced into the stomach via the oro- or nasopharynx. Correct intragastric position is confirmed by aspiration of gastric juice and auscultation of insufflated air, and confirmed by the increase in IAP after external epigastric pressure. Instillation of up to 3 ml of air allows the balloon to act as a pressure transducer. The transduced pressure is recorded, with the symphysis pubis or the midaxillary line in the supine patient used as the reference point. The tonometric balloon on the tube is usually employed for tonometry, a technique of indirect measurement of gastric pH. Sugrue et al. [68] found good correlation between gastric and urinary catheter measurements in a comparative investigation. The technique allows for continuous measurements. It may be too expensive, however, if used exclusively for pressure measurements and not in combination with pH assessments.

URINARY BLADDER PRESSURE. This simple, minimally invasive method can be easily performed at the bedside because the bladder behaves as a passive diaphragm when its volume is between 50 and 100 ml. Pressure measurements in animals recorded simultaneously through a urinary bladder catheter and directly via peritoneal catheters were equal for pressures ranging from 5 to 70 mmHg [27,60]. Simple bedside manometry provides an easy, rough estimate of IAP. The tubing of the closed urine bag collecting system is lifted and urine flows back into the bladder via the urinary catheter until equilibrium is reached. The height of the urine column in the tubing measured in centimeters above the symphysis corresponds roughly to IAP (1 cm H₂O = 0.74 mmHg). A neurogenic or small, contracted bladder may render the measurements invalid [52].

Transvesical Technique. IAP is best measured in the supine patient. The zero reference point is the symphysis pubis. Fifty to 100 ml of sterile saline is injected into the empty bladder through a Foley catheter. The tubing of the drainage bag is cross-clamped and a 16-gauge needle is inserted through the aspiration port and is connected to a water manometer or pressure transducer. Alternatively, a T connector or a three-way stopcock is inserted between the catheter and the drainage bag.

Method of Choice to Measure IAP for Clinical Use. The intravesical technique is reliable and easy to perform at bedside, even without a transducer. It is, however, time consuming and requires instillation of saline into the bladder and clamping. There may also be an increased risk for infection. The intragastric method provides continuous recording. It is much more expensive than transvesical measurements and realistically can only be used when the patient has a tonometer in place to measure intramucosal pH [68]. In patients without a bladder, it is the method of choice. Monitor readings or the height of the water column above the reference point represent IAP in mmHg or cm H₂O.

Normal and Increased IAP. Normal pressure depends on physiologic functional conditions such as coughing, defecation, and exercise. Normal mean IAP equals atmospheric pressure or less [4,6–9]. In normal subjects, IAP may increase for brief periods of isotonic or isometric contractions of abdominal wall muscles to 300 torr and more. There are no reports that such an increase is harmful. Maximal Valsalva maneuver in an upright person may increase IAP to 153–340 torr, a jump from a platform 40 cm high to 58–115 torr, maximal abdominal contraction with open glottis to 67–170 torr, and lifting 72.7±90.0 kg to 75±143 torr [69±71]. Operative laparoscopy is performed with a constant pneumoperitoneum at a pressure of 10–15 mmHg [64,65]. Elevation of IAP leads to gradual dysfunction of various systems [1,19,22,60,62,66,72–74]. Cardiac output increases first because abdominal veins are emptied upon sudden increase in IAP and then deteriorate [16,19,21,23,59]. Animals die from congestive heart failure. The magnitude of organ system dysfunction depends on premorbid physiologic reserves and various compounding factors. Conditions associated with elevation of IAP are listed in Table 1.

Anatomic Basis

Abdominal Structures. There are five anatomically distinct structures associated with the abdomen that may be subject to volume changes and modulate pressure.

1. In solid intra-abdominal organs such as the liver and spleen, changes are generally slow and may induce chronic abdominal hypertension.
2. Hollow viscus may increase in size acutely from traumatic or infectious inflammation, ileus, or bowel obstruction.
3. Blood and lymphatic vessels may contribute acutely to the development of abdominal hypertension when patient is fluid overloaded. This
Table 1. Causes of Acute Abdominal Hypertension

- Peritoneal tissue edema secondary to diffuse peritonitis
- Peritoneal tissue edema secondary to severe abdominal trauma
- Fluid overload secondary to hemorrhagic or septic shock
- Retroperitoneal hematoma secondary to trauma and aortic rupture
- Peritoneal trauma secondary to elective abdominal operations
- Peritoneal trauma secondary to emergency abdominal operations
- Reperfusion injury following bowel ischemia due to any cause
- Retroperitoneal and mesenteric inflammatory edema secondary to acute pancreatitis
- Ileus and bowel obstruction
- Intra-abdominal masses of any etiology
- Abdominal packing for control of hemorrhage
- Closure of the abdomen under undue tension
- All forms of ascites
- All forms of intra-abdominal fluid accumulations

is most likely to occur during crystalloid resuscitation for hemorrhagic shock and abdominal surgery (example shown in Fig 1).

4. The peritoneum itself may absorb huge amounts of fluid when inflamed. It consists of an outer single layer of mesothelial cells of varying architecture, a middle layer of highly vascularized loose connective tissue, and an inner layer of fascial structure (at certain locations described a Gerota’s fascia, Denonvilliers’ fascia, processus vaginalis, and phrenicoesophageal membrane) [75].

5. The peritoneal cleft (space between visceral and parietal peritoneum) may increase by accumulation of fluid because of either overproduction or reduced outflow via diaphragmatic lacunae. Also, this space my increase in volume iatrogenically when the abdominal cavity is packed with gauze for hemostasis.

In clinical reality it is difficult to attribute volume increases to any of these five structures specifically. Peritoneal edema, dilated bowel, tumors, or fluid accumulations are the main reasons for increased intra-abdominal volume.

The peritoneum comprises a total area of approximately 1.8 m², an area equal to that of the body surface. It covers all of the intestinal organs and the abdominal wall, the diaphragm, the retroperitoneum, and the pelvis. When with inflammation the peritoneum increases only 0.5 cm in thickness, the peritoneal inflammatory edema may absorb about 9 L of fluid (1.8 m² = 18,000 cm² × 0.5 cm thickening = 9,000 ml). Therefore an analogy can be drawn to the fluid shifts and associated systemic responses seen with burn injuries to the skin.

With its huge surface area, the peritoneum reacts quickly to irritations and injury, forming an inflammatory edema, as well as transudates and exudates, within a short time. This process further increases volume and pressure.

Abdominal Wall Compliance. Understanding the dynamic relationship between volume and pressure within the abdomen is important because after a relatively long period of compensation, deterioration is due to limited abdominal wall compliance (Fig 2). Compliance is structurally dependent on the stiffness of the peritoneum and its volume–pressure curve (i.e., compliance is not linear).

Upon IAP increase, abdominal wall fascias stretch and lose expandability. Progressively smaller volume increments are required to further elevate IAP [1]. Conversely, high IAP may be dramatically relieved by decompression [27–29,41,42,47,49,52,67,76–82]. Once a threshold volume is reached, the abdominal wall no longer buffers any further volume increases and thus translates them directly into pressure increases. Pressure elevations above 50 torr during contraction of the abdominal wall musculature are well tolerated for short periods (several seconds). Protracted abdominal hypertension above 50 torr is inconsistent with life [46].

Signs and Symptoms

Clinically abdominal hypertension with abdominal distention [29] and a tense abdominal wall presents with shallow respiration, an increased respiration rate, high diaphragms on percussion and auscultation, poor urinary output, and increased central venous pressure. Intubated patients require increased ventilatory pressure. Cardiovascular, respiratory, and renal dysfunction become progressively difficult to manage unless the IAP is reduced [10,38,41,52,67,68]. Abdominal decompression reverses all of the adverse effects of increased IAP.
Upon sudden release of IAP, cardiac output and IVC blood flow increases but is then promptly returned to the baseline value [22]. Recently radiologic studies of increased IAP using computed tomography yielded an increased ratio of anteroposterior to transverse abdominal diameter, direct renal compression or displacement, bowel wall thickening with enhancement, and bilateral inguinal herniation [83].

**Physiology**

The main physiologic consequences of increased IAP are summarized in Table 1. Changes in physiology that are seen with increasing pressure involve almost all systems and overlap. In this section, functional impairments are nosologically grouped.

**Cardiovascular System.** Emerson [4] was the first to note the impact of increased IAP on cardiac function. His animals died once the pressure had passed a certain threshold. It is now well documented that increased IAP significantly decreases cardiac output and left and right ventricular stroke work and increases central venous pressure, pulmonary artery wedge pressure, and systemic and pulmonary vascular resistance [1,16,17,19,21,22,49,51,56,60,63,66,67,72,73,77,84–86]. The relationship between decreased cardiac output and increased IAP is shown in Figure 5 [22], which illustrates the percentage change (normal pressure 0–10 torr versus >50 torr) of various hemodynamic parameters and the correlation of these parameters with pressure augmentations [60].

Adverse effects develop gradually and are seen with IAPs as low as 10–15 mmHg [22,60,72,86]. Cardiac output (and stroke volume) after a short initial rise due to squeezing the blood from abdominal veins [21] is then compromised through decreased venous return, elevated intrathoracic pressure, and increased systemic vascular resistance, resulting in decreased cardiac preload, increased cardiac afterload, and depressed ventricular function.

Decompression reverses all changes. Pre- and postdecompression hemodynamics in 11 trauma patients are shown in Table 2 [49].

**DECREASED VENOUS RETURN (PRELOAD).** Venous return (preload) is decreased through several mechanisms [13,16,17,21–23,49,56,59,61,86–89]. Elevated IAP is directly transmitted to large retroperitoneal veins, resulting in the caudal pooling of blood and decreased inferior vena cava flow [59] (Fig 6). In addition, functional narrowing of the inferior vena cava occurs as it leaves the abdomen at the diaphragm. The point of maximal narrowing of a tube always occurs at the transition site from an area of high external pressure (abdomen) into an area of low external pressure (thorax) [12,59]. Moreover, anatomic obstruction of the inferior vena cava can occur by the stretched diaphragmatic crura when increased IAP protrudes the diaphragm into the pleural cavities. Decreased venous outflow from the lower extremities during laparoscopic pneumoperitoneum has been observed using duplex ultrasound [90]. It is not known whether elevation of IAP induces deep vein thrombosis. Recently it was suggested that the reduced venous return from the lower extremities in morbidly obese patients and in pregnancy may be due to increased IAP [91–95].

**DECREASED CARDIAC OUTPUT AND INCREASED INTRATHORACIC PRESSURE.** IAP increases intrathoracic pressure by elevating the diaphragm [96]. Consequently, ventricular filling pressure increases and cardiac compliance decreases. With increased IAP, the cardiac output falls and systemic vascular resistance rises. The blood pressure usually remains unaffected [21,22,59], although it may fall [16,86] or rise [16,47,86]. The direction of response is influenced by the degree of the intra-abdominal hypertension and other compounding factors which are discussed below. Tachycardia is the common response to elevated IAP, compensating for the decrease in stroke volume in order to maintain cardiac output [16,60,72,86].

**INCREASED SYSTEMIC VASCULAR RESISTANCE.** The mechanisms of the increase in vascular resistance have not been elucidated but are likely to be due...
### Table 2. Decompression Physiology

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unit</th>
<th>Pre</th>
<th>Post</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>beats/min</td>
<td>124 ± 18</td>
<td>107 ± 15</td>
<td>0.005</td>
</tr>
<tr>
<td>Mean arterial pressure</td>
<td>mmHg (torr)</td>
<td>102 ± 18</td>
<td>104 ± 20</td>
<td>0.71</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure</td>
<td>mmHg</td>
<td>30 ± 11</td>
<td>24 ± 6.3</td>
<td>0.09</td>
</tr>
<tr>
<td>Central venous pressure</td>
<td>torr</td>
<td>29 ± 12</td>
<td>21 ± 7.2</td>
<td>0.06</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>L/min/m²</td>
<td>3.7 ± 0.6</td>
<td>3.9 ± 0.8</td>
<td>0.44</td>
</tr>
<tr>
<td>Stroke volume index</td>
<td>ml/m²</td>
<td>30 ± 8.0</td>
<td>37 ± 10</td>
<td>0.08</td>
</tr>
<tr>
<td>Right ventricular ejection fraction</td>
<td>%</td>
<td>37 ± 9.5</td>
<td>34 ± 7.3</td>
<td>0.48</td>
</tr>
<tr>
<td>Right ventricular end-diastolic volume</td>
<td>ml/m² torr</td>
<td>83 ± 18</td>
<td>110 ± 24</td>
<td>0.01</td>
</tr>
<tr>
<td>Right ventricular end-diastolic compliance</td>
<td>ml/m² torr</td>
<td>3.6 ± 2.1</td>
<td>5.9 ± 2.4</td>
<td>0.01</td>
</tr>
<tr>
<td>Systemic vascular resistance index</td>
<td>dyne/cm² sec/m²</td>
<td>1634 ± 474</td>
<td>1874 ± 863</td>
<td>0.23</td>
</tr>
</tbody>
</table>

Hemodynamic variables pre- (IAP = 49 ± 11 torr) and post-decompression (IAP = 19 ± 7 torr) in 11 trauma patients.

IAP = intra-abdominal pressure.

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**Fig 6.** Relationship of inferior vena cava flow to abdominal pressure increase in two dogs [59].

**Fig 7.** Influence of intra-abdominal pressure elevations on intra-abdominal blood flow. Relationship of inferior vena cava flow to abdominal pressure increase in two dogs [59].

EFFECTS ON CARDIOVASCULAR MONITORING. Increased IAP modifies various cardiovascular parameters that are commonly monitored. Femoral vein pressure, central venous pressure, pulmonary capillary wedge pressure, and right atrial pressure increase disproportionately with increasing IAP [1,16,19,21,22,27,51,56,60,63,66,67,77,85].

**Respiratory Function**

ATELECTASIS, PNEUMONIA. Both hemidiaphragms are pushed upward due to increased IAP, decreasing thoracic volume and compliance [21,67,96]. Decreased volume within the pleural cavities predisposes to atelectases and decreases alveolar clearance. Pulmonary infections may result. Pneumonia is a typical early complication in abdominal hypertension from diffuse peritonitis [50].

VENTILATION AND RESPIRATORY FAILURE. Ventilated patients with abdominal hypertension require increased airway pressure to deliver a fixed tidal volume [16,19,22,29,67]. Protrusion of the diaphragms into the pleural cavities raises intrathoracic pressure, depressing cardiac output and augmenting pulmonary vascular resistance [60]. Ventilation/perfusion abnormalities result, and arterial blood gas measurements demonstrate hypoxemia, hypercarbia, and acidosis [1,29,60,67]. Positive end-expiratory pressure (PEEP) ventilation increases intrathoracic pressure further and fosters the ill ef-
Table 3. Pulmonary Function

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unit</th>
<th>Pre</th>
<th>Post</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂ (partial pressure of oxygen in arterial blood)</td>
<td>torr</td>
<td>80 ± 36</td>
<td>124 ± 82</td>
<td>0.04</td>
</tr>
<tr>
<td>PaCO₂ (partial pressure of carbon dioxide in arterial blood)</td>
<td>torr</td>
<td>35 ± 8.3</td>
<td>34 ± 9.9</td>
<td>0.76</td>
</tr>
<tr>
<td>FiO₂ (fraction of inspired oxygen)</td>
<td>%</td>
<td>54 ± 24</td>
<td>53 ± 18</td>
<td>0.84</td>
</tr>
<tr>
<td>PaO₂/FiO₂</td>
<td></td>
<td>165 ± 78</td>
<td>236 ± 119</td>
<td>0.03</td>
</tr>
<tr>
<td>QI/QO (intrapulmonary shunt fraction)</td>
<td>%</td>
<td>33 ± 12</td>
<td>21 ± 12</td>
<td>0.04</td>
</tr>
<tr>
<td>PIP (peak inspiratory pressure)</td>
<td>cm H₂O</td>
<td>65 ± 7.5</td>
<td>46 ± 12</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>PEEP (positive end-expiratory pressure)</td>
<td>cm H₂O</td>
<td>20 ± 9.1</td>
<td>18 ± 9.5</td>
<td>0.6</td>
</tr>
<tr>
<td>TV (tidal volume)</td>
<td>ml</td>
<td>552 ± 171</td>
<td>638 ± 133</td>
<td>0.07</td>
</tr>
<tr>
<td>Cdyn (dynamic compliance)</td>
<td>ml/cm H₂O</td>
<td>13 ± 5.0</td>
<td>24 ± 6.8</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Pulmonary function variables pre- (IAP = 49 ± 11 torr) and postdecompression (IAP = 19 ± 7 torr) in 11 trauma patients. IAP = intra-abdominal pressure.

From [147] with permission.

...fected of abdominal hypertension [51,97]. Central venous pressure, pulmonary capillary wedge pressure, mean pulmonary artery pressure, and pulmonary vascular resistance are considerably increased, and venous return to the heart is impaired, compromising ventricular compliance [1,51,77,84]. Physiologic impairments are seen with IAPs as low as 20 torr. Pulmonary function variables normalize upon abdominal decompression (Table 3) [49].

Renal Function. Elevation of IAP causes renal dysfunction [2,67] and its decrease leads to reversal of renal impairment [85]. IAP of 15–20 mmHg may produce oliguria; anuria ensues with higher pressures [5,66,77]. There are no good data published that correlate abdominal hypertension with urinary output. To get an idea we compared data on seven patients [27] with controls (Fig 5). A urinary output of 5 ml/min has been observed with an IAP of 30 torr (after resection of a pelvic tumor), 34 torr (postgastrectomy), and 40 torr (after repair of a ruptured abdominal aneurysm and after a splenorenal shunt). The decrease in renal blood flow, glomerular filtration rate, urine output, and various specific tubular functions associated with elevated IAP is of multifactorial etiology [13,17,52,61,66,76]. Improved cardiac output plays a role in diminished renal perfusion, but even when cardiac output is maintained at normal or supernormal values by blood volume expansion, impairment of renal function persists [66]. Renal dysfunction is also caused by compression of the renal vein, which causes partial renal blood outflow obstruction [13,17]. Compression of the abdominal aorta and renal arteries contributes to increased renal vascular resistance [66]. Furthermore, direct compression of the kidneys elevates cortical pressures, leading to “renal compartment syndrome” [52,77]. Elevation of plasma antidiuretic hormone may represent another etiologic factor [98]. Ureteral compression can be excluded as the cause of diminished urine production with elevated IAP since oliguria was not prevented by placing ureteral stents [66,76]. Direct interaction with glomerular filtration pressure has not been investigated. After decompression, significant improvements of urinary output, creatinine clearance, and osmolar clearance have been observed (Table 4) [85].

Effects on Liver Function. Increased IAP is associated with a reduction in hepatic blood flow [15,56,63,74,99]. Hepatic arterial, portal, and microvascular blood flow are all affected (Fig 8) [74]. Trauma patients may be particularly susceptible because shock-induced changes of intestinal vascular resistance are an important determinant of portal...
Table 4. Renal Function

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unit</th>
<th>Before Paracentesis (51 Studies)</th>
<th>After Paracentesis (51 Studies)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Intra-abdominal pressure</td>
<td>(cm H2O)</td>
<td>33.5</td>
<td>9.1</td>
</tr>
<tr>
<td>Serum osmolality</td>
<td>(MOSM)</td>
<td>289</td>
<td>9.8</td>
</tr>
<tr>
<td>Urine osmolality</td>
<td>(MOSM)</td>
<td>447</td>
<td>131</td>
</tr>
<tr>
<td>Serum sodium</td>
<td>(mEq/L)</td>
<td>135</td>
<td>4.5</td>
</tr>
<tr>
<td>Urine sodium</td>
<td>(mEq/L)</td>
<td>51.2</td>
<td>33.4</td>
</tr>
<tr>
<td>Blood urea nitrogen</td>
<td>(mg/dl)</td>
<td>27</td>
<td>9</td>
</tr>
<tr>
<td>Urine volume</td>
<td>(cc/h)</td>
<td>47</td>
<td>27</td>
</tr>
<tr>
<td>Serum creatinine</td>
<td>(mg/dl)</td>
<td>1.37</td>
<td>0.49</td>
</tr>
<tr>
<td>Urine creatinine</td>
<td>(mg/dl)</td>
<td>85.5</td>
<td>54.2</td>
</tr>
<tr>
<td>Creatinine clearance</td>
<td>(cc/min)</td>
<td>46.2</td>
<td>18.2</td>
</tr>
<tr>
<td>Osmolar clearance</td>
<td>(cc/min)</td>
<td>71.4</td>
<td>35.4</td>
</tr>
<tr>
<td>Free water clearance</td>
<td>(cc/min)</td>
<td>0.71</td>
<td>0.26</td>
</tr>
</tbody>
</table>

* = Significant at p < 0.01.
† = Not significant.
From [85] with permission.

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**Fig 9.** Intra-abdominal pressure and intestinal perfusion: as intra-abdominal pressure increases, mesenteric and mucosal blood flow decreases [62].

Blood flow and may be complemented by abdominal hypertension. It may be assumed that hepatic synthesis of acute-phase protein, immunoglobulin, and factors of the other host defense systems will be impaired by reduced hepatic flow, further compromising response to massive trauma and diffuse peritonitis [50]. Detailed studies addressing the issue of reduced hepatic protein synthesis have not yet been published nor is there information on the impact on wound healing.

The impairment of healing of the abdominal wound after laparotomy (wound dehiscence and wound infection, fascial necrosis leading to necrotizing fasciitis) has been attributed to the reduced blood flow to the abdominal wall and fascia in the presence of abdominal hypertension (Diebel LN, personal communication). The application of abdominal binder may further compromise abdominal wall perfusion by sandwiching the structure between increased abdominal volume and the binder. This should be avoided.

**Gastrointestinal Function.** Besides impairment of liver function from abdominal hypertension, other gastrointestinal functions may be compromised by increased pressure. Splanchnic hypoperfusion may start at IAPs as low as 15 mmHg. Reduced perfusion of intra-abdominal arteries, veins, and lymphatics is well documented. Secondary effects such as changes in mucosal pH, bacterial translocation, bowel motility, production of gastrointestinal hormones, and exocrine and hormonal alteration deserve more focused research. The effects of abdominal hypertension on the spleen, pancreas, adrenals, and reproductive organs are not yet known.

**Impairment of Arterial Flow.** Abdominal hypertension impairs intestinal blood flow (Figs 7–9) [1,58]. Elevation in IAP results in decreased mesenteric arterial blood flow, intestinal mucosal blood flow [62], and arterial perfusion of the stomach, duodenum, intestine, pancreas, and spleen [63]. As IAP increases, mucosal pH falls, indicating severe ischemia or necrotizing pancreatitis [62]. Compartment-induced impaired intestinal perfusion may be a critical factor in anastomotic healing. Abdominal hypertension probably plays a role in many of the organ dysfunctions of currently questionable etiol-
ology. Examples may be ischemic gastritis, acalculous cholecystitis or pancreatitis, colon ischemia, and some forms of bowel ischemia.

These changes are greater than can be accounted for by the alterations in cardiac output [63] and also occur when cardiac output and systemic blood pressure are maintained at normal levels [16,62].

EFFECTS ON ABDOMINAL VEINS. IAP is transmitted to all abdominal and retroperitoneal veins (Figs 8 and 9). Brief elevations of IAP in cirrhotic patients cause increases in free and wedged hepatic venous pressures and increased azygous blood flow. Opposite changes occur with reductions in IAP [56,74]. Whether increased IAP precipitates the rupture of esophageal varices remains controversial [11,18,56,58].

EFFECTS ON LYMPH FLOW. Lymphatic flow in the thoracic duct significantly decreases when IAP is elevated and promptly increases after abdominal decompression [100]. Stretching of the diaphragm decreases the volume of the diaphragmatic lymphatic lacunae, thus reducing transport of peritoneal fluid into the thoracic lymphatics [101].

TRANSLOCATION. High rates of translocation of bacteria to the regional lymph nodes have been observed when increased IAP reduces intestinal perfusion (Diebel LN, annual meeting of the Western Trauma Association, 1997). This factor may be significant in the development of infections and sepsis in patients with abdominal hypertension and may contribute to further septic complications, fueling ongoing infection.

Intracranial Pressure. Idiopathic intracranial hypertension is increased in chronic abdominal hypertension. It decreases when IAP is reduced in morbidly obese patients [24–26,47,91,102,103]. Abdominal hypertension significantly increases intracranial pressure at pressures routinely used during laparoscopy [103]. The mean intracranial pressure at baseline was 13.4 ± 771.0 torr. It rose to 18.7 ± 1.5 torr (p = 0.0001) during pneumoperitoneum of 10–15 mmHg. When the intracranial pressure was increased, as seen with head injuries, it rose from 22 ± 1.8 torr to 27.4 ± 0.9 torr (p < 0.001).

These increases were independent of changes in arterial pCO₂ or arterial pH. Bloomfield et al. [47] found that elevated IAP increased intracranial pressure (7.6 ± 1.2 to 21.4 ± 1.0) and abdominal decompression returned intracranial pressure toward baseline. Abdominal trauma in head-injured patients contributes to intracranial hypertension. Data support the notion that it is better to have a low threshold for abdominal decompression in patients with combined injuries. Twenty percent of patients with severe abdominal injuries and 40% with severe head injuries were documented as having an associated head or abdominal injury of the same magnitude [104]. Figure 1 shows a trauma patient whose abdomen was decompressed using the artificial bur. Diagnostic laparoscopy may increase intracranial
to both increased myocardial oxygen consumption and myocardial ischemia or congestive heart failure in patients who are susceptible [86]. Similarly a moderate increase in IAP may suffice to cause anuria in a patient in hemorrhagic shock or when superimposed on chronic renal failure [17].

The intravascular volume status of the patient is crucial; hypovolemia aggravates the effects of increased IAP, whereas volume expansion with intravenous fluids tends to compensate for the decreased venous return, maintaining cardiac output [21,61,66,67,74,77,86]. A similar effect is achieved by the Trendelenburg position. The additive consequences of PEEP ventilation were mentioned earlier [16,19,86,97]. Cardiovascular disturbances causing a specific injury such as diaphragmatic rupture are more profound when combined with elevated IAP [106].

![Image](image_url)

**Fig 11.** STAR closure device covering protruding bowel. Cross section of the open abdominal wound and the STAR closure device in place. The gap between the fascias can be adjusted to allow optimal decompression and still prevent the fascias from sideward retraction. As the pathology is reversed and the intra-abdominal pressure normalizes, the two bur sheets may be reapproximated by trimming excessive material off the sheet that carries the “hooks.” Note that the space between the subcutaneous tissue above the bur is filled with Kerlex gauze surrounding a drain that is connected to negative suction. A self-adhering drape covers the entire wound and surrounding skin to seal the wound and to prevent contamination with microorganisms.

Therapeutic Decompression

Therapeutic decompression may be indicated in severe abdominal hypertension. Nonoperative and operative methods are available. Decompression may reverse all of the adverse effects of increased IAP [1,17,19,21,22,31,32,42,48,66,72,107]. The changes in hemodynamic, pulmonary, and renal function parameters before and after decompression are listed in Tables 2–6. Immediately after decompression, a paradoxical response may be seen with a brief elevation of cardiac output and IVC blood flow, followed by a prompt return to baseline values [21].

Operative and nonoperative decompression is addressed directly in a small number of anecdotal reports (summarized in references 39 and 46), but there is a huge body of literature on the “open abdomen technique,” indirectly dealing with abdominal decompression. While these studies focus on operative management of diffuse peritonitis, the importance of decompression as part of the technique is not appropriately acknowledged. Recent publications analyzed 1,983 such cases [108–110].

**Nonoperative Decompression.** Nonoperative decompression has been reported mainly for cirrhotic patients with ascites. Removal of ascites in cirrhotic patients to decrease IAP has been associated with a dramatic improvement in renal function [28,85,89], cardiac performance, and hepatic perfusion [14,56,88]. Sudden removal of a large volume of peritoneal fluid is hemodynamically safe in patients who are not volume depleted [111]. Bloomfield et al. [112] recently presented a sophisticated device...
Table 5. Tissue Perfusion

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unit</th>
<th>Pre</th>
<th>Post</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial lactate</td>
<td>mmol/L</td>
<td>4.4 ± 2.3</td>
<td>3.9 ± 1.5</td>
<td>0.35</td>
</tr>
<tr>
<td>Arterial base deficit</td>
<td>mEq/L</td>
<td>11 ± 5.4</td>
<td>8.5 ± 5.0</td>
<td>0.04</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>g/dl</td>
<td>11.8 ± 1.5</td>
<td>12.7 ± 2.0</td>
<td>0.17</td>
</tr>
<tr>
<td>Oxygen delivery (DO₂)</td>
<td>ml/min/m²</td>
<td>570 ± 115</td>
<td>663 ± 189</td>
<td>0.08</td>
</tr>
<tr>
<td>Oxygen consumption (VO₂)</td>
<td>ml/min/m²</td>
<td>124 ± 44</td>
<td>142 ± 43</td>
<td>0.16</td>
</tr>
<tr>
<td>Arterial oxygen saturation</td>
<td>%</td>
<td>96 ± 3</td>
<td>98 ± 3</td>
<td>0.18</td>
</tr>
<tr>
<td>Mixed venous oxygen saturation</td>
<td>%</td>
<td>75 ± 10</td>
<td>77 ± 10</td>
<td>0.14</td>
</tr>
<tr>
<td>Arterial pH</td>
<td></td>
<td>7.26 ± 0.14</td>
<td>7.32 ± 0.08</td>
<td>0.22</td>
</tr>
<tr>
<td>Gastric intramucosal pH</td>
<td></td>
<td>7.15 ± 0.13</td>
<td>7.20 ± 0.14</td>
<td>0.01</td>
</tr>
<tr>
<td>Urine output</td>
<td>ml/h</td>
<td>105 ± 85</td>
<td>188 ± 127</td>
<td>0.007</td>
</tr>
</tbody>
</table>

Systemic and regional perfusion variables pre- and postdecompression.
* Indexed to body surface area.
† For the 4 hours before and after decompression.
From [147] with permission.

Table 6. Effects of Abdominal Decompression in Trauma Patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preoperative</th>
<th>Postoperative</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Bladder pressure (cm H₂O)</td>
<td>40</td>
<td>1.1</td>
<td>22</td>
</tr>
<tr>
<td>Tidal volume (ml)</td>
<td>830</td>
<td>140</td>
<td>850</td>
</tr>
<tr>
<td>Peak inspiratory pressure (cm H₂O)</td>
<td>52</td>
<td>1.2</td>
<td>44</td>
</tr>
<tr>
<td>Mean airway pressure (cm H₂O)</td>
<td>27</td>
<td>1.3</td>
<td>23</td>
</tr>
<tr>
<td>PaO₂/FiO₂ ratio (mm Hg)</td>
<td>159</td>
<td>105</td>
<td>217</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>86</td>
<td>1.8</td>
<td>92</td>
</tr>
<tr>
<td>Cardiac index (L/min/m)</td>
<td>4.8</td>
<td>1.5</td>
<td>4.9</td>
</tr>
<tr>
<td>Urine output (ml/h)</td>
<td>79</td>
<td>55</td>
<td>123</td>
</tr>
<tr>
<td>Fluid balance (L/24h)</td>
<td>5.4</td>
<td>4.6</td>
<td>3.2</td>
</tr>
</tbody>
</table>

Effect of abdominal decompression on various parameters in 46 trauma patients (35 men, 11 women).
* Paired, two-tailed t-test.
NS = not significant.
From [42] with permission.

that noninvasively reduced intra-abdominal pressure.

Operative Decompression. Abdominal operative decompression is the method of choice in patients with severe abdominal hypertension from peritoneal edema and large tumors. There is published clinical experience with decompression of posttraumatic or postoperative ACS in patients in whom ACS was recognized and treated [32,33,35,39,82,110]. Postdecompression improvement of hemodynamics, pulmonary function variables, tissue perfusion, and renal function parameters are listed in Tables 2–6. After decompression, cardiac, respiratory, and renal function is immediately improved, followed occasionally by transient episodes of hypotension [41,42,47–49,67,77,81]. Immediate postdecompression asystole has been reported in four cases (three fatal). It has been suggested that after decompression, cardiac output increases while systemic vascular resistance decreases; hypotension occurs because the dilation of peripheral vessels is more profound [77]. The hypothesis that postdecompression cardiovascular collapse results from reperfusion injury because of release of acid metabolites from the reperfused ischemic viscera and lower extremities is not well documented [67].

Technique of Operative Abdominal Decompression. To prevent hemodynamic decompensation during decompression, intravascular volume should be restored, oxygen delivery maximized, and hypothermia and coagulation defects corrected. The abdomen should be opened under optimal conditions in the operating room, including hemodynamic monitoring with adequate venous access and controlled ventilation. As a measure to combat the expected reperfusion washout of by-products of anaerobic metabolism, prophylactic volume loading with a crystalloid solution containing mannitol and sodium bicarbonate may be of benefit [113]. Use of vasoconstrictor agents during decompression to prevent the sudden drop in blood pressure has been suggested [77]. After decompression, the abdomen
and the fascial gap is left open using one of the temporary abdominal closure methods mentioned below (Figs 10 and 11). The postdecompressed open abdominal wound must be hermetically sealed using the hypobaric wound shield, a self-adhering drape, under negative suction pressure, to prevent contamination of the abdominal cavity and to allow for measuring fluid and protein loss replacement (Fig 12) (see the next section) [114].

**Bridging the Abdominal Gap with the Bur Closure.** Leaving the fascia open and closing only the skin with sutures or towel clips to protect the bulging viscera has been recommended [115,116]. Occasionally, however, closing the skin only may not result in sufficient decompression. Instances of IAP of 50 mmHg or more have been reported [117].

Certainly leaving both fascia and skin unsutured (open abdomen technique) offers maximal reduction in IAP, but it may result in high rates of fistula and evisceration [108,109]. Bridging the fascial gap with prosthesis circumvents these problems. Absorbable and nonabsorbable, and porous and nonporous prostheses have been recommended in this situation with variable success [118]. Use of absorbable prostheses has been associated with very high rates of intestinal fistula formation and ventral hernia formation, while the use of Gore-Tex® patch (Flagstaff, AZ), Marlex® mesh (Cranston, RI), and silastic mesh in patients who received high-volume resuscitation after massive abdominopelvic trauma or emergent repair of a ruptured abdominal aortic aneurysm resulted in acceptable outcome [119]. Gore-Tex in particular minimized the risk of gastrointestinal fistulization associated with other techniques [120].

The best option may be the artificial bur (STAR-Patch®, www.starsurgical.com, Burlington, WI), a Velcro®-like device that has recently been approved by the U.S. Food and Drug Administration (FDA) (Figs 1, 4, and 10). This device has also been approved by the European Union (EC Mark) and is available at www.HIDIH.com, Dörrbach, Germany [32,35,121]. The artificial bur closure consists of two adherent sheets of knitted synthetic fibers, each with clinging elements on one surface. When applied against each other, the sheets adhere together. At the end of the first operation (or the decompressive procedure) the two sheets are sutured with a running 0-nylon suture to the two edges of the abdominal fascia. The sheets are then applied against each other to effect temporary closure. The tension of the closure can be adjusted by increasing or decreasing the contact area between the two sheets (Fig 13). During routine use of the artificial bur in more than 200 cases, we have never encountered an instance of “decubitus-exposed” intestinal fistula. Once the underlying pathology of abdominal hypertension is controlled, the abdomen can be closed fascia to fascia the same way as with a single noncomplicated laparotomy.

**Reclosure of the Abdomen.** Abdominal reclosure should be attempted only in well-resuscitated patients after tissue oxygenation has been restored and hypovolemia, hypothermia, and coagulopathy have been corrected. Ideally, in the absence of pressing indications for early reexploration (e.g., after packing or damage control) [55,122–124], the reoperation should be scheduled when the probability of achieving complete fascial closure is the highest. This occurs usually 3–4 days after the primary abdominal entry, when brisk diuresis, nega-
Primary fascial closure during the initial hospitalization, however, may circumvent the above-mentioned problem. At reoperation, the two sheets of the artificial bur are peeled from each other and the abdominal cavity is thoroughly explored. Fascial edges are approximated provisionally and, if still under tension, IAP is measured. When excessive tension, documented by increased pressure greater than 20 torr, exists, the two sheets of bur are reapplied against each other. Usually, during reexploration, the contact surfaces of the two sheets can be decreased and the overlapping bur trimmed off, achieving a gradual decrease of the abdominal defect, with final fascial closure during the next planned procedure. When the abdominal pressure is less than 15–20 torr with the fascias reapplied, final closure can be attempted. Final closure assumes that the surgeon is satisfied with hemostasis, viability of the bowel, adequacy of necrotic tissue debridement, and the condition of suture lines, and that he is reasonably sure that a further laparotomy will not be necessary in the near future.

ACS and Peritonitis

Increased IAP as a side-effect of infectious abdominal catastrophes identifies a subset of patients with a grave prognosis [35]. Surgeons treating intra-abdominal infection respected the “enormous increases of IAP” as early as 100 years ago (Körte, 1897 cited in [3]). Although effective treatment for ACS became available with the introduction of the open abdomen techniques [50,108], few authors realized the impact of their methods on impaired physiology that resulted from abdominal hypertension [76,126–131]. The advocates of planned relaparotomy for severe intra-abdominal infections or trauma did not account for the adverse effects of increased IAP initially as they closed the abdomen with retention sutures (Fig 3) [132–135] or simple zippers [132–134]. Because the retention sutures left severe pressure marks on the abdominal wall underneath the plastic plates that held the sutures (Fig 3), the impact of increased IAP on physiology was gradually more appreciated, and broad devices for temporary abdominal closure were used [32,136]. The final development was the procedure we have termed STAR abdominostomy, where STAR stands for staged abdominal repair and abdominostomy for the open abdomen. The abdominal cavity is closed using the STAR closure device as described above [110].

A closer look at the various open abdomen techniques, however, reveals no visible improvement. The mortality rate for 869 cases of open abdomi-
A possible explanation for the lack of improvement may be that too many additional complications impaired the outcome of open abdominostomies. Intestinal fistulas formed in more than 16% of the open abdominostomy procedures and in more than 10% of the mesh abdominostomy operations [137–142].

The use of a mesh device for temporary abdominal closure in combination with planned relaparotomy (STAR abdominostomy) may circumvent the problems that were encountered by simply leaving the abdomen open. The mortality rate of 385 cases enrolled in 11 studies dealing with some sort of STAR abdominostomy was 28.1%, whereas in the conventionally operated control groups, the mortality was 44.2% [108,109]. The studies analyzed did not give an answer to what would be the best device to cover the gap between the opened abdominal fascial borders. Ethizip is easily pulled apart and may open spontaneously in the ICU. The use of Marlex mesh with a zipper requires frequent resuturing of the zipper to the Marlex as abdominal edema decreases and fascial edges need to be reapproximated. Sheets of Gore-Tex and similar material are too small and not designed to be used for only a few days. Also, Ethizip, the zippers, and the plastic infusion bags that are used in some institutions have not been approved by the FDA for medical use. The artificial bur, on the other hand, which was recently approved by the FDA, resists pulling forces of more than 100 lb and has never opened spontaneously in our hands. Trimming off to adjust for decreasing abdominal girth is very easy. It has been our device of choice [32,35,121].

ACS and Trauma

Patients with multiple injuries or hemorrhagic shock from penetrating abdominal trauma are particularly susceptible to developing ACS. There are numerous reports in the literature addressing the issue and its therapeutic decompression [33,55,122–124,143]. The compartment syndrome may be further aggravated if there is a need for immediate laparotomy to control hemorrhage. Once the abdomen is opened in these patients resuscitation fluid sequesters into the peritoneal loose connective tissue and bowel wall, leading to enormous protrusion of the intestines through the abdominal wound, as demonstrated in Figure 1, where the protrusion is covered with the artificial bur. It is then impossible to close the abdomen under these circumstances, and forceful closure of the abdomen in patients having massive retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hematoma and visceral edema, or a large retroperitoneal hem

Fig 14. (A) Appendectomy incision and midline incision before opening, (B) after index STAR opening, and (C) same patient at discharge from hospital, 12 days later. There was primary wound healing. No hernia developed.
need for homeostatic packing, may be detrimental [46]. Multiple methods for temporary closure have been advocated; most of which have been unsatisfactory [116]. Such procedures have been advocated as damage control operations [37,122,144–146]. With the artificial bur, the abdominal fascia can be reapproximated as the abdominal edema subsides, and final closure may be accomplished by fascia-to-fascia closure (Figs 4, 6, 13, and 14). Formation of huge abdominal hernias is avoided. The STAR procedure [35] thus combines the concept of damage control operations with definitive repair in the acute trauma setting.

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