COMPARTMENT SYNDROME

ABDOMİNAL COMPARTMENT SYNDROME

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DEFINITIONS

- **Intra-abdominal pressure;** it is the **steady-state** pressure concealed within the abdominal cavity.
- Normal IAP ranges from sub-atmospheric to 0 mmHg , 5–7 mm Hg in critically ill patients.
- Intra-abdominal hypertension; In healthy individuals, normal IAP is <5–7 mmHg, The upper limit of IAP is generally accepted to be 12 mmHg.
- Pathologically elevated intraabdominal compartment pressures exceeding 12 mmHg define **intraabdominal hypertension (IAH)**
- **Abdominal Compartment Syndrome** in adults is defined as an intraabdominal pressure of **>20 mmHg** with evidence of organ dysfunction.

	Normal	IAH	ACS
IAP*	5-7mmHg	≥12 mmHg Grade I: 12-15 mmHg Grade II: 16-20 mmHg Grade III: 21-25 mmHg Grade IV: > 25 mmHg	≥ 20 mmHg
Organ dysfunction**	No	No	Yes

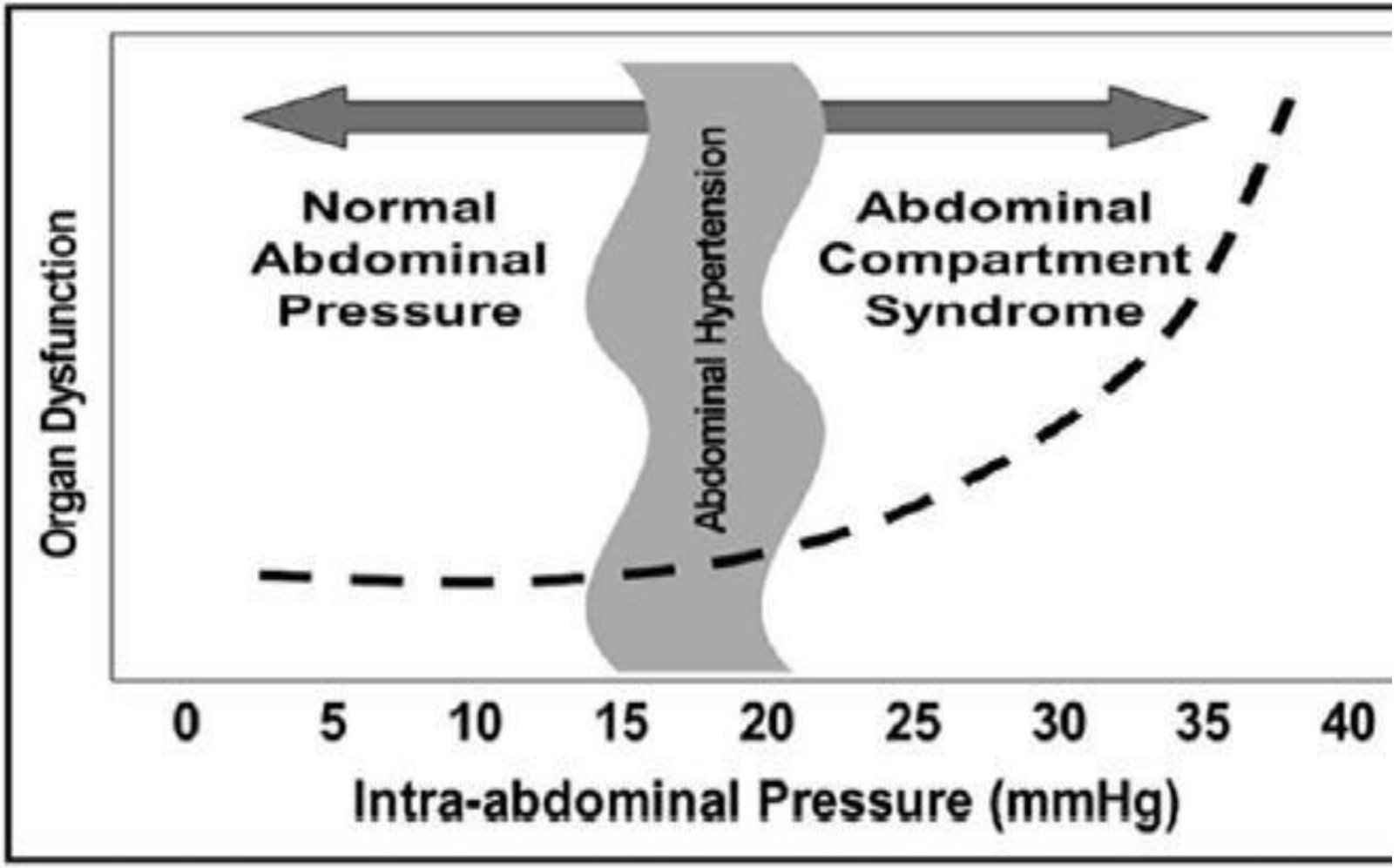
Epidemiology

- Historically, the prevalence of ACS was most frequently studied in the context of trauma, where it has been shown to occur in 1% of all general trauma admissions, and 5% to 15% of trauma ICU admissions.

- **Currently**, in patients with known risk factors, intra-abdominal hypertension can be found in approximately 25% of ICU admissions with almost 3% having ACS.

- Mortality rate is high, ranging from 25% to 75%, due to the presence of multi-organ failure and severe underlying injuries.

Figure 2. Distinctions between normal intra-abdominal pressure (IAP), intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS)



Physiological variations of IAP

- **IAP increases with inspiration** (diaphragmatic contraction) and **decreases with expiration** (diaphragmatic relaxation).
- It is also directly affected by the **volume of the solid organs or hollow viscera**(which may be either empty or filled with air,liquid or fecal matter), the presence of **ascites**, **blood** or other **space-occupying lesions** (such as tumors or gravid uterus), and the presence of **conditions that limit expansion of the abdominal wall** (such as burn or third-space edema)

Etiology

Primary causes

- Are due to decreased abdominal compliance, presence of an intra-abdominal or retro-peritoneal injury, or a pathological process.
- **Decreased abdominal compliance**: (i.e., the elasticity of the abdominal wall and diaphragm), such as severe obesity, burns with abdominal wall eschars
- **Intra-abdominal infection/inflammation.**
- **Ileus** :any process that decreases or impairs the normal transit of bowel contents can produce accumulation of luminal contents leading to bowel distension and an **increase in IAP**.
- **Liver cirrhosis**: patients with higher amounts of ascites at baseline are at higher risk of developing ACS.
- **Hemoperitoneum**.
- **Pneumoperitoneum** can arise from progression of a pathophysiological process, such as peptic ulcer disease or diverticulitis, that leads to a perforated viscus.

Secondary causes

- **Are due to tense ascites or oedema of an otherwise normal bowel.**

- **Excess fluid resuscitation (more than 3 L within a 24-hour period) is the most common cause of ACS.**

- **Massive blood transfusion (>10 units in 24 hours) are also common cause; these are usually given to patients with severe traumatic injury or post-traumatic coagulopathy.**

- **In practice, many primary causes require fluid resuscitation and/or massive transfusion; these patients will have mixed primary and secondary causes for increased IAP.**

RISK FACTORS

Strong :

- Excessive fluid resuscitation (>3 L in 24 hours)
- Massive blood transfusion (>10 units in 24 hours)
- Decreased abdominal compliance
- Intra-abdominal infection/inflammation
- Haemoperitoneum

❑Weak :

- Ileus
- Pneumoperitoneum
- Loss of abdominal domain
- Comorbid cirrhosis
- Retroperitoneal haematoma

CLINICAL MANIFESTATIONS & DIAGNOSIS

- **Cardiovascular**

1. Impaired cardiac function.

2. Reduced venous return.

- **Pulmonary**

1. These effects are likely due to elevation of the diaphragm causing extrinsic compression of the lung.

2. also have reduced chest wall compliance and spontaneous tidal volumes.

Renal

1.Renal vein compression increases venous resistance.

2.Renal artery vasoconstriction.

3. Oliguria generally develops at an intra-abdominal pressure of approximately 15 mmHg, while anuria usually develops at an intra-abdominal pressure of approximately 30 mmHg.

- **Gastrointestinal**

1.Intestinal mucosal perfusion is decreased at an intra-abdominal pressure of approximately 20 mmHg.

2.Celiac artery and superior mesenteric artery blood flow are decreased at an intra-abdominal pressure of approximately 40 mmHg.

3.**Hepatic** :The liver's ability to remove lactic acid is impaired by increases of intra-abdominal pressure as small as 10 mmHg.

- **Central nervous system**

1. Intracranial pressure (ICP) transiently increases during the short-lived elevation of intra-abdominal pressure that occurs with coughing, defecating, or emesis.
2. critical decrease in cerebral perfusion and progressive cerebral ischemia.

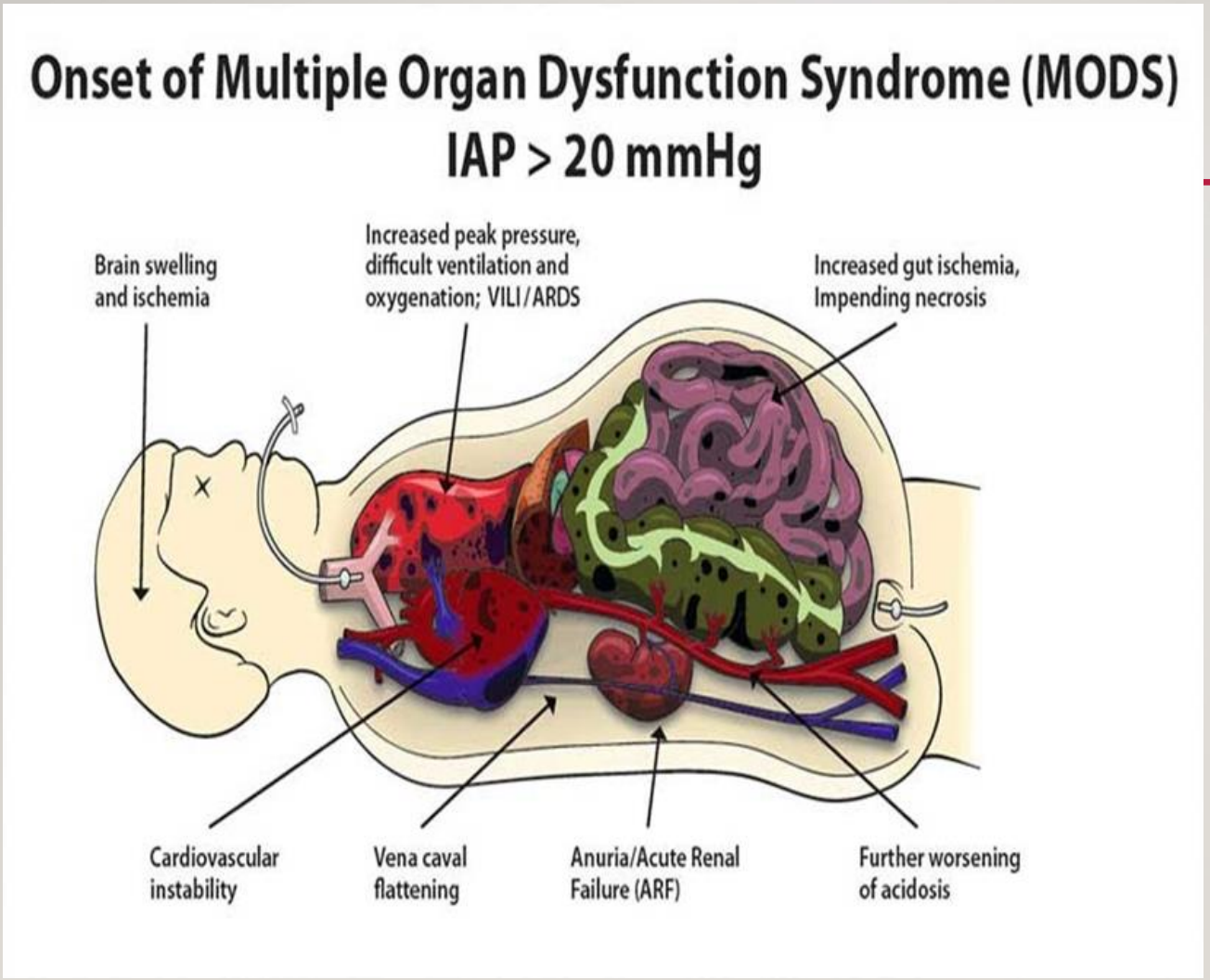
CLINICAL PRESENTATION

- **Symptoms:**
  - Malaise, weakness
  - Light-headedness
  - Dyspnea
  - Abdominal bloating, or abdominal pain

- **Signs:**
  - Progressive oliguria
  - Increased ventilatory requirements
  - Hypotension, tachycardia
  - Elevated jugular venous pressure, jugular venous distension, peripheral edema
  - Abdominal tenderness
  - Hypoperfusion; including cool skin, restlessness, or lactic acidosis
  - A previous history of pancreatitis

IMAGING FINDINGS

- A **chest radiograph** may show decreased lung volumes, atelectasis, or elevated hemidiaphragms.
- Chest **computed tomography (CT)** may demonstrate tense infiltration of the retroperitoneum that is out of proportion to peritoneal disease, extrinsic compression of the inferior vena cava, massive abdominal distention, direct renal compression or displacement.


Onset of Multiple Organ Dysfunction Syndrome (MODS)
IAP > 20 mmHg
Brain swelling and ischemia
Increased peak pressure, difficult ventilation and oxygenation; VILI/ARDS
Increased gut ischemia, Impending necrosis
Cardiovascular instability
Vena caval flattening
Anuria/Acute Renal Failure (ARF)
Further worsening of acidosis

ACS

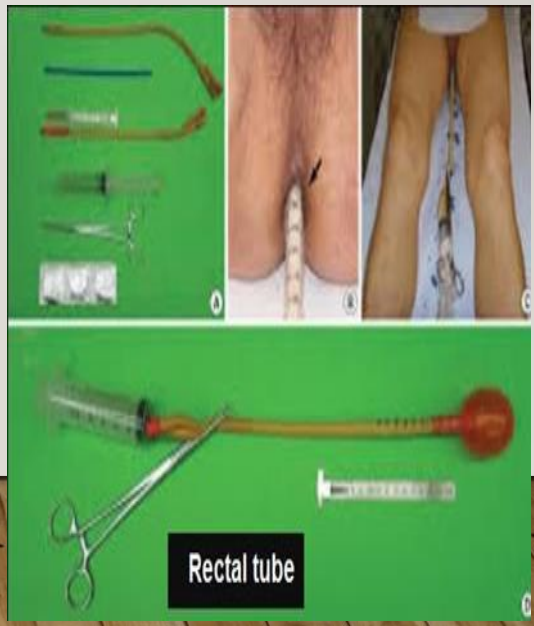
DIAGNOSTIC EVALUATION

- Definitive diagnosis of ACS requires measurement of the intra-abdominal pressure.
- **Measurement of intra-abdominal pressure:** Intra-abdominal pressure can be measured indirectly using intragastric, intracolonic, intravesical (bladder), or inferior vena cava catheters.
- Measurement of bladder (intravesical) pressure is the **standard method** to screen for intra-abdominal hypertension (IAH) and ACS .

MANAGEMENT

CONSERVATIVE (NON-SURGICAL) MANAGEMENT

- The early use of non-surgical interventions may prevent the progression of IAH to ACS.
- The **neuromuscular blockade** is used for relaxation of the **abdominal** musculature, leading to a decrease in pressures. These patients must be put on a **ventilator**.
- Gastric decompression is done with **NG tube**. Colonic decompression is done with **rectal tube**. These are done to evacuate the Lumen.
- The head of the patient's bed should not be raised for more than 30°.



- **Percutaneous drainage** is a decompressive procedure used for; abscesses, ascites, or any free fluid in the abdominal compartment.

  -These patients are identified by bedside **ultrasound**.

  -The morbidity of a laparotomy is avoided.

- **Positive fluid balance (Hypervolemia)** is associated with high mortality rates. And it is managed by; **dialysis**. It is usually accompanied by **CHF.** Furthermore, on the occurrence of **Pulmonary Edema**, ventilation must be supported.

- The relief of any concomitant **pain** is **essential**.

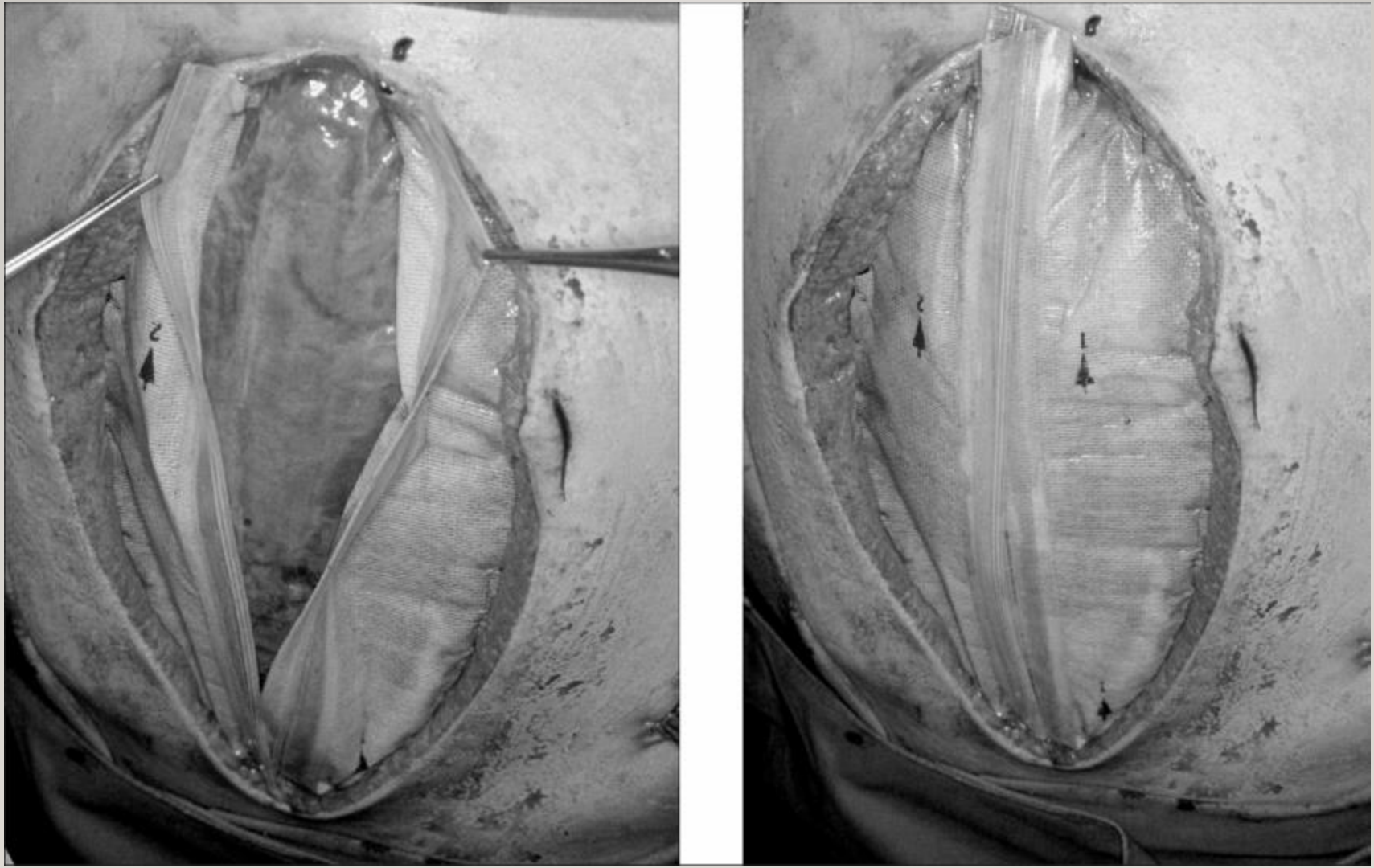
SURGICAL MANAGEMENT

- It is the Primary treatment of ACS; If conservative management does not resolve the IAH, or end-organ damage is seen.
- The treatment of ACS is to **release the abdominal fascia**.
- The abdominal fascia should be **left open** and **covered under sterile conditions,** with **delayed fascial closure**.
- **Laparotomy** is considered;

- After surgical laparotomy, the **abdominal** fascia can be **temporarily closed** (fascia approximation), by devices such as (VACs, meshes, and zippers).

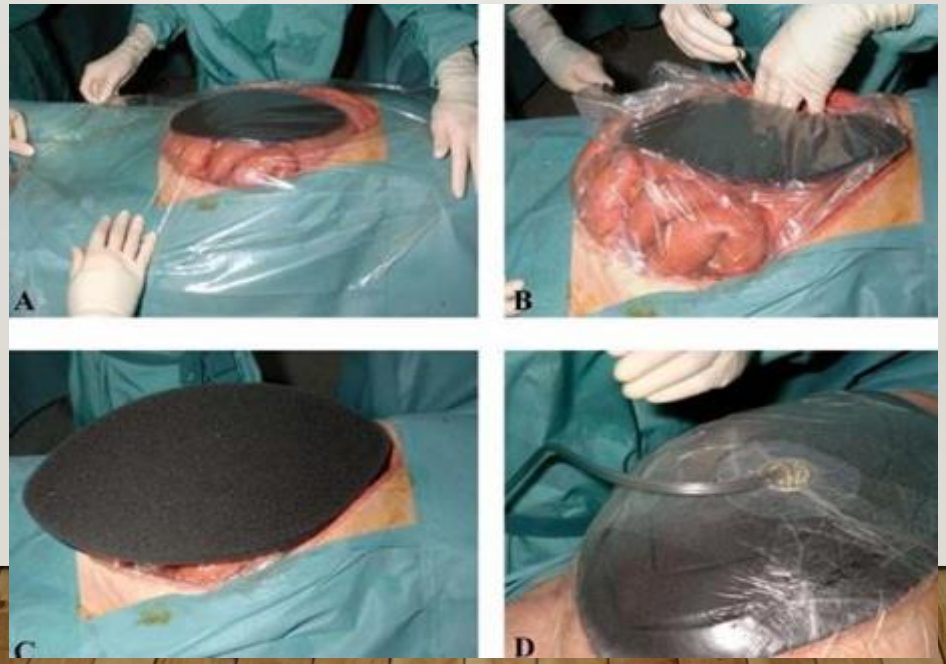
- The fascia can be **completely closed** after 5-7 days, if there is a pressure decrease.

-Laparostomy with Zipper in place.



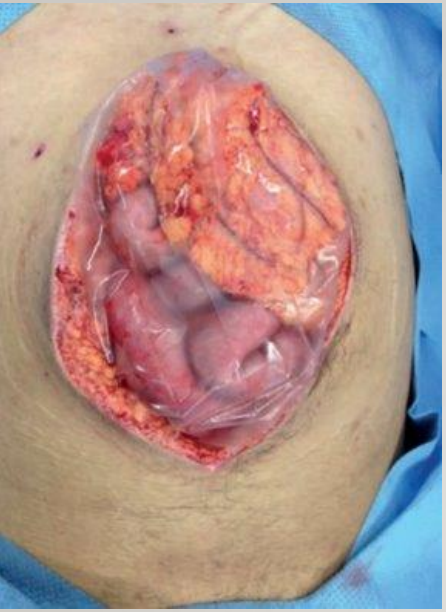
- Left untreated, ACS may lead to multiple system end-organ dysfunction or failure and has a high mortality.
- **Abdominal wall closure** should be attempted **every 48 to 72 hours** until the fascia can be reapproximated.
- If the abdomen cannot be closed **within 5 to 7 days** , a large **incisional hernia** is the net result.
- The goal of the operative team is **to close the abdomen as quickly as possible,** to minimize morbidity and cost of care.

- **Timing of decompression** is crucial; as **70%** mortality in patients with a **delay** in decompression, and a uniform mortality in those **not** undergoing decompression.
- Decompression is usually performed operatively, either in the **ICU** if the patient is hemodynamically unstable, or in the **OR** if they were stable.
- **ICU bedside laparotomy** is easily accomplished, avoids transport of hemodynamically compromised patients, and requires **minimal equipment** (e.g., scalpel, suction device, cautery, and dressings for temporary abdominal closure)

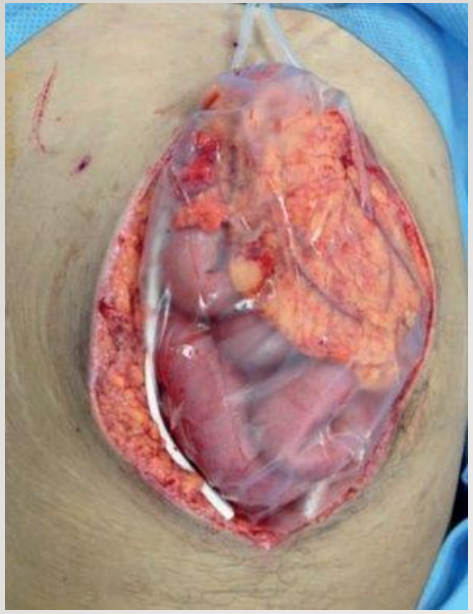


OPERATIVE DECOMPRESSION

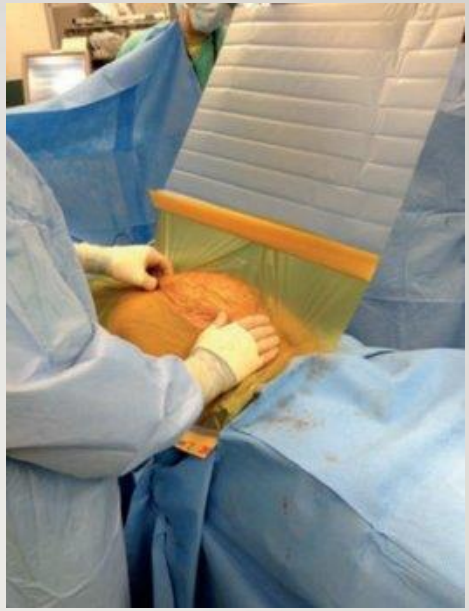
- Damage control surgery ,and the recognition of abdominal compartment syndrome have dramatically improved patient survival, but at the risk of an open abdomen.
- Despite having a widely open abdomen; patients can develop **recurrent abdominal compartment syndrome**. Therefore, **bladder pressure should be monitored every 4 hours.**
- Patients with an open abdomen lose between **500** and **2500 mL** per day of abdominal effluent (peritoneal fluid); thus, volume compensation with albumin-rich fluid must be carried out.

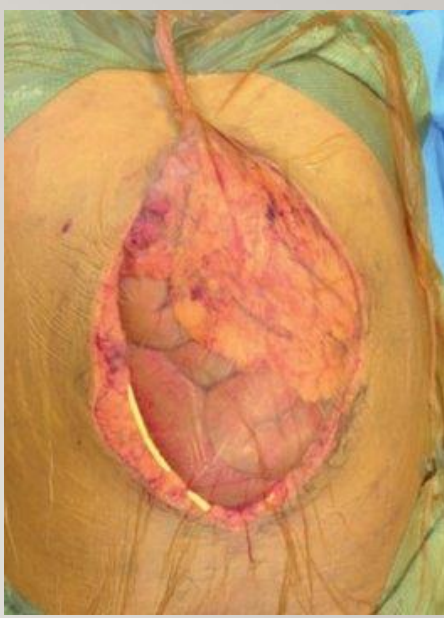


1- Temporary closure of the abdomen entails covering the bowel with a fenestrated subfascial 45 × 60 cm sterile drape.



2- Placing Jackson-Pratt drains along the fascial edge.



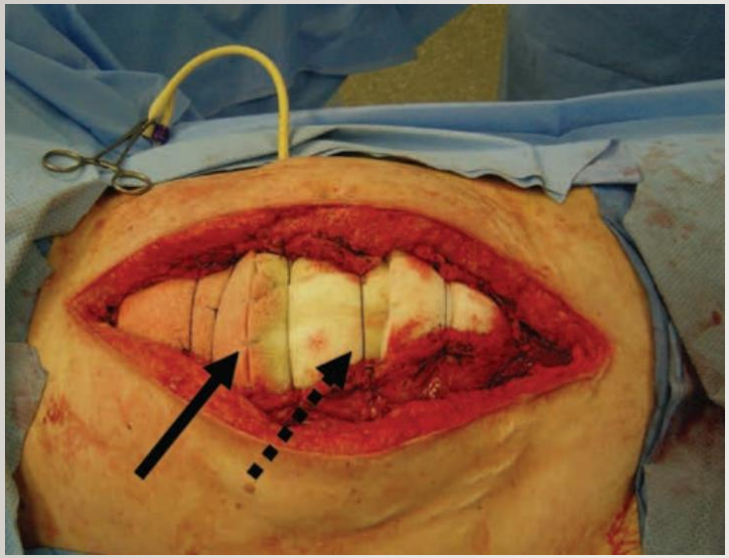


3&4 - Occluding with an ioban drape to prevent bacterial wound contamination.

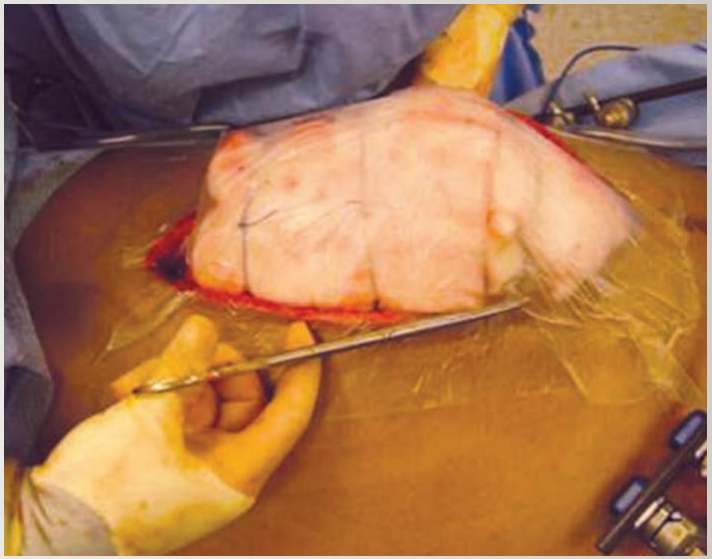
SEQUENTIAL CLOSURE TECHNIQUE

- **Sequential closure technique** is **currently used.**
- Recurrent return to the OR every 48 hours **until closure is complete**.
- Success rate with this approach exceeds 95% .
- Patients not attaining fascial closure; **20%** suffer GI tract complications that prolong their hospital stays. These include; abscesses, enteric fistulae, and bowel perforations.

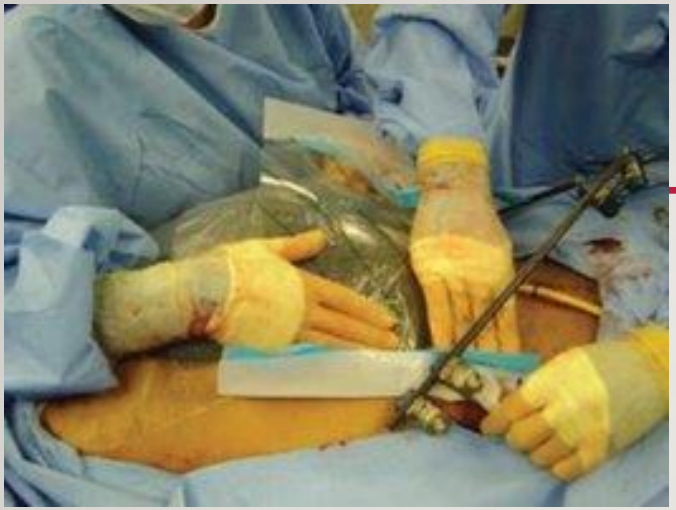
Sequential Closure Technique

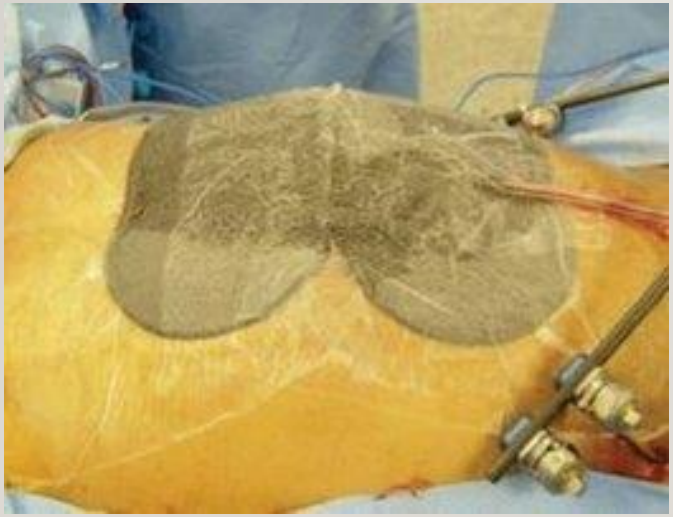


1- Multiple white sponges (solid arrow), stapled together, are placed on top of the bowel underneath the fascia. Interrupted sutures are placed approximately 5 cm apart (dashed arrow).

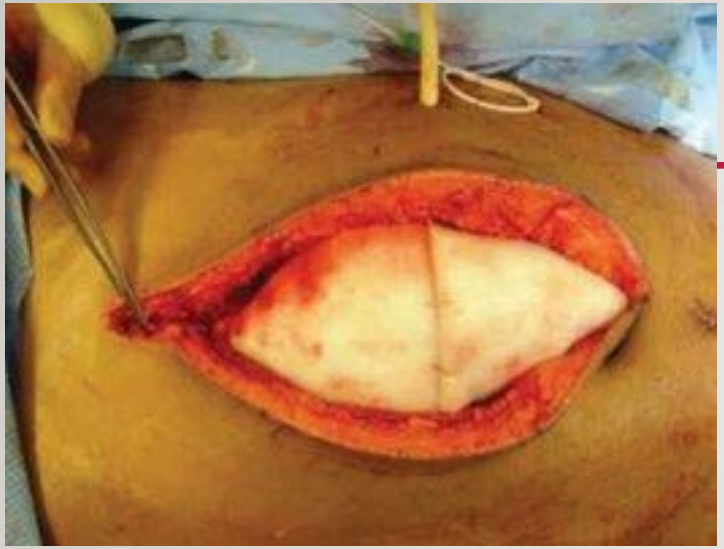


2- After the placement of the sticky clear plastic vacuum-assisted closure (VAC) dressing over the white sponges and adjacent 5 cm of skin, the central portion is removed by cutting along the wound edges.

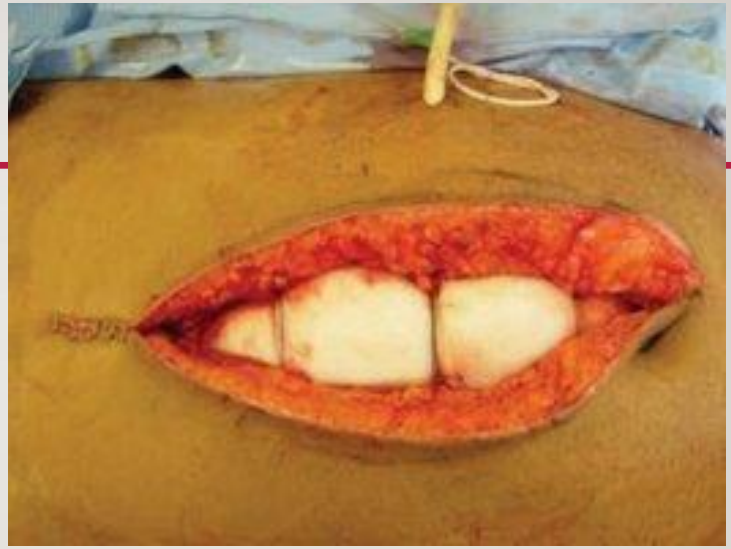




3&4 - **BlackVAC sponges** are placed on top of the white sponges and plastic-protected skin with standard occlusive dressing and suction



5- On **return to the OR** 48 hours later, fascial sutures are placed from both the superior and inferior directions until tension prevent further closure; skin is closed over the fascial closure with **skin staples**.



6- White sponges (fewer in number) are again applied, and fascial retention sutures are placed with planned return to the OR in **48 hours**.

BURN INJURY MANAGEMENT

- Massive resuscitation of burned patients may lead to an abdominal compartment syndrome.
- Decompressive **laparotomy** is the standard of care, but it carries **poor prognosis** in burn patients.
- **Adjunctive measures** should be initiated **before resorting to decompressive laparotomy**, and these include;

  -minimizing given fluid

  -performing torso escharotomies

  -decreasing tidal volumes (Low Tidal Volume Ventilation)

  -chemical paralysis (neuro-muscular blockade)

PROGNOSIS

- **Abdominal compartment syndrome** is a fatal complication, If it is left untreated or was delayed in treatment.
- Risk factors for mortality include a history of **diabetes** and the **transfusion of a high volumes of blood products**.
- Many reports state that even with treatment, multiorgan failure can delay recovery for weeks or months. These patients are usually characterized by; a **prolonged need for mechanical ventilation**, **need for dialysis**, and **prolonged hospital stays**.

Thank You