Role of the Open Abdomen in Critically Ill Patients

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KEYWORDS

• Open abdomen • Abdominal compartment syndrome
• Temporary abdominal closure • Negative pressure wound therapy
• Retroperitoneal Hemorrhage • Intra-abdominal infections • Damage control surgery

KEY POINTS

- An open abdomen is commonly used in critically ill patients to temporize permanent abdominal closure for clinical reasons.
- The most common reason for leaving the abdomen open by reopening a laparotomy, not closing, or creating a fresh laparotomy is the abdominal compartment syndrome.
- The open abdomen technique is also used in damage control operations and intra-abdominal sepsis.
- Negative pressure wound therapy may be associated with better outcomes than other temporary abdominal closure techniques.
- The open abdomen is associated with many early and late complications, including infections, gastrointestinal fistulas, and ventral hernias. Clinicians should be vigilant regarding the development of these complications.

An open abdomen is defined as purposely foregoing fascial closure of the abdomen after the cavity is opened. It is most commonly used after a midline laparotomy or celiotomy. The goal is to temporize abdominal closure for a clinical reason and return in a short period of time to effect complete fascial closure. While the abdomen is open, the patient's disease or condition is stabilized or preferably improved. The patient is returned to the operating room and complete fascial closure is achieved under semi-elective conditions.[1]

The technique was first used by Ogilvie to treat intra-abdominal sepsis more than 75 years ago.[2] His initial intent was to leave the abdomen open, allowing the intra-abdominal infection to drain like any other infected wound, which would achieve the

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principle of source control.[3] Vaseline-impregnated gauze was used to protect the exposed bowel. The infection was treated, it was hoped that the patient improved, and then fascial closure was achieved 1 to 4 days later. An open abdomen continues to be used for severe peritonitis usually complicated by septic shock, but it is most commonly used as treatment of intra-abdominal hypertension causing the abdominal compartment syndrome (ACS).

ABDOMINAL COMPARTMENT SYNDROME

One of the most common indications for not closing or reopening a laparotomy incision is the ACS. ACS represents abdominal hypertension that produces organ dysfunction secondary to the increased intra-abdominal pressure. Intra-abdominal hypertension (IAH) is defined as a bladder pressure greater than 12 to 15 mm Hg in adults. When IAH reaches 20 to 25 mm Hg and organ dysfunction is recognized, ACS is considered to be present. These pressures are relative but have been identified by the World Society of the Abdominal Compartment Syndrome.[4] The causes of IAH are considered primary or secondary. Primary IAH/ACS is associated with intraperitoneal or retroperitoneal processes such as bleeding or infection. Secondary IAH/ACS is associated with extra-abdominal processes such as bowel edema precipitated by massive fluid resuscitation of shock, which causes bowel edema or intraperitoneal fluid accumulation. Differentiation of the two types is important because the treatment steps are different.[4]

The organ dysfunction with ACS is usually recognized by changes in lung and renal function. Pulmonary dynamics change as ACS develops. Tidal volumes decrease or, if mechanical ventilation is being used, an increase in peak pressure is noted with similar tidal volumes. This process occurs as the IAH pushes the diaphragm up, decreasing the intrathoracic volume. The increased peak pressures increase the chances of barotrauma and possibly cause acute lung injury. Renal dysfunction is reflected by a decreasing urine output caused by decreased renal perfusion as the renal vein is compressed from the increased abdominal pressure. Renal blood flow is decreased as renal vascular resistance increases. Other organs that can manifest changes with ACS include cardiac and cerebral organs. As IAH increases, venous return is reduced, which causes a decrease in cardiac output despite what appear to be increased intravascular pressures. Cerebral changes are possible when the intrathoracic pressures reduce venous return from the brain. Intracranial pressure increases, which may result in cerebral edema. IAH and the ACS can produce changes in other intra-abdominal organs.[5] The bowel mucosa becomes ischemic and, after release of the IAH, a reperfusion injury can occur. Abnormal hepatic metabolism is also observed.

DIAGNOSIS OF ABDOMINAL COMPARTMENT SYNDROME

When ACS is suspected, bladder pressures can be measured. This measurement is commonly accomplished by instilling a small amount of sterile saline into the bladder and attaching the Foley to a pressure transducer. The symphysis pubis is used as the zero point. A grading system is used for the pressure measurements. Grade I is 12 to 15 mm Hg; grade II is 16 to 20 mm Hg; grade III is 21 to 25 mm Hg; and grade IV is greater than 25 mm Hg.[6–8] Although a single bladder pressure is commonly used to detect and grade IAH, a calculation of an abdominal perfusion pressure (APP; mean arterial pressure – intra-abdominal pressure) is suggested as a better method to detect ACS. An APP greater than 60 mm Hg is suggested as an adequate goal for resuscitation when IAH is present, although this value has not been subjected to prospective trials.[9] Although abnormal physiology can be detected at all levels of IAH,