The American Journal of Surgery (2012) 204, 826–835

The American Journal of Surgery®

The Southwestern Surgical Congress: Edgar J. Poth Memorial Lecture

The open abdomen: practical implications for the practicing surgeon

Clay Cothren Burlew, M.D.*

Department of Surgery, Denver Health Medical Center and the University of Colorado School of Medicine, Denver, CO, USA

KEYWORDS:
Open abdomen;
Damage control surgery;
Abdominal compartment syndrome;
Enteral nutrition;
Bowel injury;
Trauma

Abstract. The open abdomen is a necessary sequela after damage-control surgery or abdominal compartment syndrome. Management of the patient in the intensive care unit continues to evolve, with considerations of fluid resuscitation, enteral nutrition, and supportive care. Management of the abdominal contents incorporates several basic techniques and considerations: appropriate temporary covering, enteric injury repair in most patients, placement of an anastomosis in an area of the abdomen with minimal manipulation without exposure to the atmosphere, acquiring enteral access for initiation of enteral nutrition, and ultimate abdominal closure. An understanding of these complex factors is instrumental for the practicing surgeon.

It is truly an honor to give the Edgar J. Poth Memorial Lecture, so named after the 15th president of the Southwestern Surgical Congress. Although the recognition of the utility of open-abdomen management occurred after his period of scientific investigation, I would like to think that this topic might have been one of interest to Dr. Poth. I begin by acknowledging that when most people hear the phrase "open abdomen," they think that this happens only in the realm of trauma. Because many practicing surgeons try to avoid trauma call after completing their residencies, they feel they will never encounter this entity. These patients, in fact, are seen by the general surgeon: the medical intensive care unit (ICU) consult for abdominal compartment syndrome (ACS) in the pancreatitis patient after large volume resuscitation, the patient with perforated diverticulitis and feculent peritonitis who is in septic shock, or the gunshot wound to the superior mesenteric artery requiring complex reconstruction.

Presented as the Edgar J. Poth Memorial Lecture at the 2012 Southwestern Surgical Congress, Rancho Palos Verdes, California.
* Corresponding author. Tel.: 303-436-6558; fax: 303-436-6572.
E-mail address: clay.cothren@dhha.org
Manuscript received April 10, 2012; revised manuscript April 23, 2012

0002-9610/$ - see front matter © 2012 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.amjsurg.2012.04.013

The following is a brief overview of my address this morning. In discussing the open abdomen, the first question is, How did we arrive here? The most common etiologies resulting in an open abdomen are ACS and damage-control surgery (DCS), whether DCS for trauma or general surgery. I will discuss these etiologies and the indication for leaving the abdomen open for each. The next issue of concern is the temporary closure options that are available to enable transport to the ICU. In the ICU, there are some management considerations that are unique to the open-abdomen patient. Next, how should one prepare for repeat laparotomy, including intraoperative questions and the plan for definitive abdominal closure? Finally, what are some of the complications you might encounter during the hospital course of the patient with an open abdomen?

Abdominal Compartment Syndrome

The ACS is typified by intra-abdominal hypertension due to either intra-abdominal injury (primary) or following massive resuscitation (secondary) (Fig. 1).[1–8] Some causes of primary ACS include solid-organ injuries, bowel perfora-

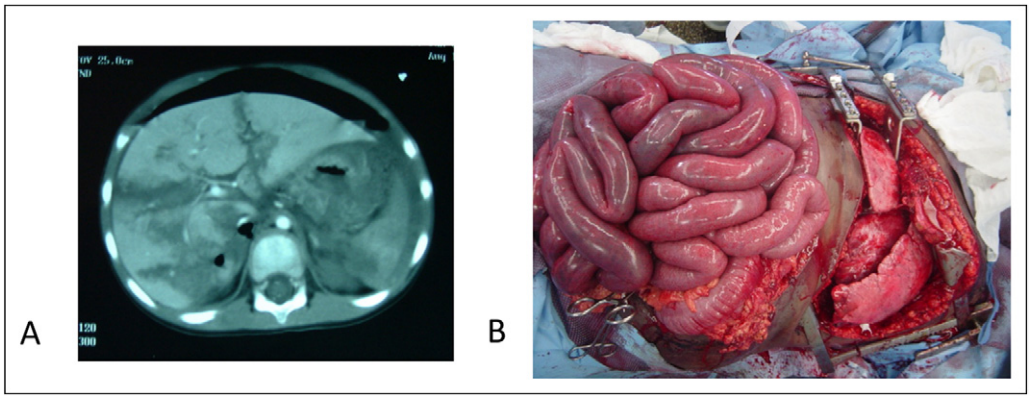

Figure 1 The ACS can be due to a primary intra-abdominal process such as a liver injury (A) or resuscitation-associated visceral and retroperitoneal edema such as that seen in a patient undergoing massive resuscitation for a gunshot wound to the heart (B).

tions or obstruction, ruptured vasculature, and postoperative hemorrhage. Secondary ACS may be due to any etiology requiring large-volume resuscitation, including both crystalloid and blood products. This includes patients with extremity trauma, isolated head or chest trauma, pancreatitis, liver failure, and overt sepsis. In these cases, intra-abdominal hypertension is due to resuscitation-associated bowel edema, retroperitoneal edema, and large quantities of ascitic fluid.

Increased abdominal pressure affects multiple organ systems (Fig. 2). The ACS, however, is defined by intra-abdominal hypertension causing such end-organ sequelae as decreased urine output, increased pulmonary pressures, decreased preload and subsequent cardiac dysfunction, and even elevated intracranial pressure.[1] The first key is that you have to think of the diagnosis of ACS. In any critically ill or injured patient, there are many etiologies that could cause low urine output and cardiopulmonary woes. But if intra-abdominal hypertension does not occur to you as a potential cause of the patient's low urine output or cardiovascular collapse, rather than the patient's associated hypovolemic shock or sepsis or cardiac contusion, you will miss the diagnosis of ACS and the window for intervention. The pitfall to avoid is that physical examination cannot definitively diagnose intra-abdominal hypertension. Although the patient may have a markedly distended abdomen that is suggestive of the diagnosis, your examination may reliable only about 40% of the time. A diagnosis of intra-abdominal hypertension is obtained by measuring the patient's bladder pressure. To measure a patient's bladder pressure, 50 mL of saline is instilled into the bladder via the aspiration port of a 3-way Foley catheter with the drainage tube clamped; after waiting for 30 to 60 seconds to allow the detrusor musculature to relax, pressure measurement with a manometer at the pubic symphysis is performed.[9] There are several conditions in which the bladder pressure may not be reflective of the intra-abdominal pressure. This includes patients with external compression on the bladder such as pelvic packing, those with bladder rupture, marked adhesive disease, and patients with a neurogenic bladder.

Although we measure the actual pressure within the abdominal cavity, the real question is, When does intra-abdominal hypertension become ACS? ACS, by definition, is intra-abdominal hypertension causing end-organ sequelae. So it

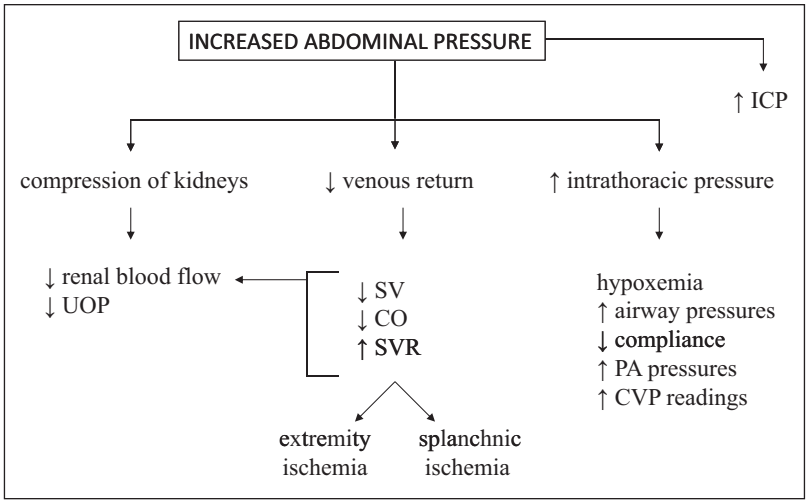

Figure 2 Increased abdominal pressure affects multiple organ systems.