

published about 5 years ago.² The authors did not refer to our article, but the observations in the 2 studies are remarkably similar.

The authors emphasize the differences between IAH and ACS and make the observation that untreated IAH may lead to full-blown ACS. In addition, they mention a bladder pressure of 25 mm Hg as the critical pressure beyond which the undesirable sequelae of ACS appear to set in—points similar to those we made in a group of critically injured patients with penetrating abdominal trauma who developed primary IAH. As in the present article, our patients who had higher levels of bladder pressure also had evidence of gastric mucosal acidosis as measured with gastric mucosal pH, an observation also made by other authors.³

The authors' proposed cause for secondary ACS is similar to what we hypothesized for primary ACS resulting from catastrophic abdominal injuries and coagulopathy. Of particular interest is our observation, in concordance with findings in the present study, that in patients who have had sequential hits of hemorrhagic shock and subsequent IAH, the respiratory and splanchnic effects of IAH may be more severe than with either insult alone. The increased frequency of IAH associated with increased multiple organ failure and mortality was observed in our clinical study² and in the laboratory.⁴⁻⁷ In our article,² we also suggested that these serious complications should be anticipated in patients who are critically injured and/or undergoing damage-control procedures. An attempt must be made to prevent IAH and ACS by leaving the abdomen open, as has been suggested by other authors.⁸ Such an approach may actually decrease multiple organ failure and mortality.

This is an important study that establishes supranormal resuscitation as a cause of secondary ACS, advances the concept that IAH and ACS are gradations of the same disease, and reaffirms the splanchnic basis of multiple organ failure associated with these complications. Because the splanchnic bed seems to be adversely affected by both inadequate and overzealous resuscitation, what methods do the authors propose to prevent ACS caused by either of these 2 mechanisms?

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In reply

We are honored that Dr Ivatury has commented on our work.¹ His seminal observations related to ACS are well known to our group, and we referenced 2 of his articles. We believe that ACS is in part an iatrogenic complication of current management of traumatic shock (ie, damage-control surgery combined with goal-oriented resuscitation in the intensive care unit). With ongoing analysis of severely injured patients who require aggressive resuscitation, our concepts of ACS pathogenesis have evolved.¹⁻⁷

We first noted the iatrogenic nature of overzealous perihaptic packing. As a result, we minimize intraperitoneal packing and practice early pack removal. We next began to use presumptive Bogota bag (an opened, empty, sterile intravenous fluid bag) closure after damage-control laparotomy but soon recognized that this does not eliminate the problem. We therefore use routine urinary bladder pressure (UBP) monitoring.

However, UBP has limitations. Patients who develop ACS typically require emergency operative or interventional radiological procedures and arrive in the intensive care unit on average 6 hours after admission with established IAH (mean UBP of 19 mm Hg). Conventional wisdom has been IAH falsely elevates commonly measured indexes of preload (eg, central venous pressure and pulmonary capillary wedge pressure), and, therefore, patients with low cardiac output should be volume loaded. We have observed that volume loading sets up a futile cycle of crystalloid preloading. Initial volume loading may increase cardiac output, but it also increases intra-abdominal pressure. Increased intra-abdominal pressure compresses the inferior vena cava, which adversely affects cardiac preload and promotes intestinal edema. With continued preloading, UBP increases into the low 20s. At this point, the abdomen is on the steep portion of its compliance curve. A small increase in abdominal content results in a large increase in intra-abdominal pressure, which is often detected too late with intermittent UBP measurements to avoid full-blown ACS.

In regard to the question posed by Dr Ivatury, we believe that fundamental changes need to occur in management before patients reach the intensive care unit. From our models, we can accurately predict the clinical trajectory of ACS within 3 to 6 hours. These patients arrive in obvious shock, but their sources of bleeding are not always readily apparent. This is especially true in patients with blunt trauma who have pelvic and extremity fractures. Early volume loading during the ABCs (airway, breathing, circulation) of advanced trauma life support is valuable in defining hemodynamic stability, but continuing aggressive volume loading during uncontrolled bleeding sets the stage for ACS.

Failure to promptly recognize the severity of ongoing blood loss is a recurring problem in the emergency department. Early base deficit determination and central venous pressure monitoring are valuable adjuncts. Blood and fresh frozen plasma should be used liberally, and indiscriminate crystalloid infusions should be minimized. Hemorrhage con-

control is paramount. Novel hemorrhage-control techniques such as application of topical fibrin sealant materials or infusion of recombinant activated factor VII are attractive concepts that need to be tested.

Current resuscitation standard of care (ie, early volume loading with lactated Ringer solution and packed red blood cell transfusion) is directed at minimizing the severity of the ischemic insult. Resuscitation standard of care is not directed at optimizing splanchnic perfusion or at minimizing inflammation induced by ischemic reperfusion. Recent laboratory data indicate that resuscitation standard of care is detrimental in these regards. We share Dr Ivatury's belief that mesenteric ischemic reperfusion is a pivotal mechanism in the multiple organ failure cascade and that decompression of full-blown ACS may serve as a second insult.

In the laboratory, we are testing alternative resuscitation fluids. Hypertonic saline (HTS) is an attractive adjunct that requires much less volume than does lactated Ringer solution, and it increases mesenteric blood flow. The argument favoring HTS is even more compelling with the recent recognition that HTS markedly reduces shock-induced systemic neutrophil cytotoxicity and acute lung injury.^{8,9} Results of more recent studies^{10,11} have linked these beneficial effects of HTS shock resuscitation to the gut. Using a standard superior mesenteric artery occlusion model, we demonstrated that HTS resuscitation markedly reduces intestinal inflammation, injury, and dysfunction by the local induction of heme oxygenase-1.¹² Using a similar model, Sims et al¹³ have shown that Ringer ethyl pyruvate ameliorates intestinal injury induced by ischemic reperfusion.

We do not believe the colloid vs crystalloid debate is resolved, and we share the belief that colloids might reduce the incidence of ACS. However, the potential benefits must be weighed against known detrimental effects. Newer colloid solutions are now available but should be tested in clinically relevant models before they can be endorsed for preventing ACS.

Early presumptive decompressive laparotomy is an alternative strategy to prevent massive intestinal edema and full-blown ACS. Trauma surgeons, however, are reluctant to perform this surgery because it creates a second problem—the open abdomen. Our treatment of the open abdomen, however, has evolved. To date, we have described on 45 patients in whom we could not approximate the abdominal fascia at secondary laparotomy.^{14,15} We treated their open abdomens with a vacuum-assisted wound closure device. In 38 (84%) of these patients, we achieved early fascial closure with minimal complications (2 wound infections, 2 enteric fistulas, and no abscesses) at a mean of hospital day 7. With vacuum-assisted wound closure and validated pre-

diction models, we believe presumptive decompressive laparotomy will become a rational strategy.

In summary, current management of traumatic shock has saved the lives of severely injured patients who would previously have died but has also created a virtual epidemic of ACS and open abdomens worldwide. These problems will not likely go away, but we believe that the incidence can be minimized by identifying the patients at high risk in the emergency department and managing their care with alternative strategies. We look forward to future studies from Dr Ivatury and his colleagues who have clearly been leaders in defining the cause and clinical consequences of ACS.

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