The interrelationship of sepsis resuscitation and intra-abdominal hypertension

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Introduction:

In the last 8 years an explosion in interest regarding interventional therapies for severe sepsis has occurred. The nidus for this interest was Dr. Rivers landmark article demonstrating that early goal directed therapy for sepsis provided a 16% improvement in survival.[1] Since that publication a worldwide effort, the Surviving Sepsis campaign, has been implemented to combine multiple therapeutic interventions into protocol driven "bundles" of care.[2] Bundling therapeutic interventions is felt to optimize outcome improvements for the severely septic patient compared to single interventions alone.

Vigilant application of sepsis care bundles yield impressive results: survival improvements often exceed 15-20% while resource utilization is simultaneously decreased.[3] However, it is clear that aggressive early intervention is critical to achieve these survival improvements.[4] Failure to optimize tissue perfusion at an early point may lead to irreversible cellular ischemia, continued inflammatory stimulation and coagulation cascading as well as multiple organ failure secondary to necrosis and/or cellular apoptosis from the profound oxygen debt that occurs.

A dramatic increase in the recognition, understanding and management of another syndrome related to critical illness – intraabdominal hypertension and the abdominal compartment syndrome (IAH/ACS) - occurred at the same time as the evolution of sepsis care.[5] Despite the increase in awareness regarding IAH/ACS, many clinicians continue to have little understanding regarding incidence, clinical presentation or interventional therapies available for IAH/ACS.[6] Perhaps even less recognized is the profound inter-relationship between IAH/ACS and severe sepsis:

- Intra-abdominal hypertension is intrinsically linked to aggressive fluid resuscitation in patients with capillary endothelial leak syndromes – exactly what occurs in severe sepsis resuscitation.[7-9]
- As volume resuscitation becomes a standard practice for sepsis care the prevalence of IAH/ACS may be increasing. In fact, Regueira et al recently reported that septic shock has the highest reported prevalence of IAH/ACS for any critically ill patient population.[10]

- IAH/ACS is a time dependent syndrome that if unrecognized and untreated leads to profound cellular ischemia and multiple organ failure mimicking that seen in severe sepsis but requiring additional interventions to prevent and/or avoid.[11-13]
- The pathophysiologic manifestations of sepsis and IAH/ACS are interrelated (Figure 1) and are so similar that IAH/ACS frequently goes unrecognized. Clinicians usually assume that the patients physiologic deteriorations are reflective of sepsis without recognizing that many of these complications may be compounded by sepsis induced IAH/ACS, a potentially preventable and treatable condition.[5, 14]
    - IAH/ACS presence has a direct impact on central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) that if unrecognized can lead to inadequate fluid resuscitation during sepsis care unless intra-abdominal pressure (IAP) is known and the CVP/PAOP are corrected for that influence.[15-18]

Figure 1 – Pathophysiologic inter-relationship of IAH and severe sepsis

This manuscript will review the inter-relationships between severe sepsis, sepsis resuscitation protocols and IAH/ACS. It will then discuss the clinical reasoning and outcomes data that suggest that patients who undergo sepsis resuscitation should have IAP monitoring started during the resuscitation phase and IAH/ACS interventions implemented, if necessary, during the management-bundle phase.

Sepsis and IAH – Inter-related pathophysiologic processes

Patients suffering septic shock and receiving early goal directed therapy are almost perfect candidates for developing intra-abdominal hypertension. The pathophysiologic events that occur in septic shock lead to severe (splanchnic) hypoperfusion while simultaneously causing a massive systemic inflammatory response with cytokine release and immune hyperresponsiveness. This results in an increase in capillary permeability, vascular volume leakage into the interstitium, hypovolemia and profound cellular ischemia. Early goal directed therapy provides some restoration of tissue blood flow through aggressive volume expansion; however, much of that fluid is eventually sequestered as edema in the extravascular (interstitial) spaces, which includes

the bowel wall and mesentery. As the edema progresses, the volume within the abdominal compartment expands until the compliance threshold of the abdominal cavity is crossed and the abdominal wall can no longer easily expand. At this point intra-abdominal pressure rapidly rises with any further fluid sequestration, leading to intra-abdominal hypertension.

The resulting intra-abdominal hypertension leads to intra- and extra-abdominal organ dysfunction via two mechanisms: pressure induced vascular compression/hypoperfusion and immune mediated organ dysfunction. During the early phases of IAH, organ dysfunction is primarily related to pressure induced reductions in cardiac output and organ perfusion.[15, 19, 20] Rising IAP compresses the venous structures within the peritoneal and retroperitoneal space including the vena cava.[15] It also pushes the diaphragms cephalad leading to reduced thoracic volumes, atelectasis and elevated intrathoracic pressure.[19] (See figure 3) The end result is reduced preload, reduced cardiac output, increased SVR, increased intrathoracic pressure with higher peak and plateau pressures, atelectasis, worsening oxygenation and ventilation, tissue hypoperfusion and worsening cellular hypoxia. These events begin with IAP levels as low as 10 mmHg and are profoundly worsened as the IAP continues to rise.

Figure 2 – Pathophysiologic impact of IAH on the bodies organ systems:

Elevated intraabdominal pressure also causes immune and inflammatory effects that can result in progressive organ failure. An emerging body of evidence suggests that untreated intra-abdominal hypertension may act as the second insult in the two-event model of multiple organ failure (MOF).[9, 12, 13] This model postulates that an initial insult causing cellular ischemia (such as what occurs in the syndrome of septic shock) primes the patients immune system for an exaggerated response to any secondary insult.[21] The second insult, in the form of IAH induced recurrent ischemia, can lead to immune hyperresponsiveness and increased inflammatory mediator release. Rezende-Neto el al found that IAH causes elevated levels of pro-inflammatory cytokines (tumor necrosis factor, and interleukins IL-6 and IL-1) as well a lung myeloperoxidase (MPO) and a 3-

fold increase in inflammatory neutrophil expression in an animal model.[12, 13] Other investigators also note increases in inflammatory mediators as well as bacterial translocation across the bowel wall during the ischemic insult that occurs with increased intra-abdominal pressure.[22, 23] Left unchecked and untreated this secondary insult (IAH) leads to a hyper-inflammatory immune response with a high incidence of multiple organ dysfunction and death. Figure 1 shows the pathophysiologic interrelationships of sepsis and of IAH/ACS.

Incidence of IAH/ACS in septic shock

The increasing focus on severe sepsis is also leading to more recognition of the complications of massive fluid resuscitation in the form of IAH/ACS. Regueira et al found a 51% incidence of intra-abdominal pressure over 20 mmHg in patients resuscitated from septic shock.[10] Daugherty et al noted an 85% incidence of IAH (IAP ≥ 12 mmHg) and a 30% incidence of abdominal compartment syndrome (defined as IAP ≥ 20 mmHg with organ dysfunction) in their aggressively fluid resuscitated septic shock population.[8] Other investigators report IAH/ACS rates of 40-50% in severe sepsis.[7, 24, 25]

IAH/ACS impact on septic shock monitoring endpoints: CVP, $ScvO_2$, Urine output

The frequency with which IAH/ACS develops in sepsis has important clinical ramifications. One concern regards the impact of IAH on sepsis resuscitation endpoints. Current surviving sepsis guidelines emphasize the importance of traditional end-expiratory CVP of 12 mmHg in combination with a mixed central venous oxygen saturation (ScvO2) of 70% or greater. Once the CVP level is achieved, fluid resuscitation is suspended. If the oxygenation endpoint has not been met, packed red blood cells or inotropic support is implemented.[1, 2] In addition, if the patient's mean arterial pressure is unacceptable (<65 mmHg) a vasopressor may be added to the mix. However, end-expiratory CVP overestimates fluid status in patients suffering from IAH because the intra-abdominal pressure is transduced across the diaphragm onto the catheter.[15-18] Failure to recognize the conductance of intra-abdominal pressure onto the CVP measurement may lead to under resuscitation with fluid and potentially worsen cellular ischemia. At the same time, over-vigorous fluid administration may complicate tissue

ischemia / reperfusion leading to more edema, ascites and higher levels of IAP. Multiple authors now recommend either that traditional end-expiratory CVP be corrected for IAP to reduce the possibility of inadequate fluid administration or that alternate measurements of fluid status such as volumetric indices be used in any patient with IAH.[15-18]

Failure to detect splanchnic ischemia (a complication of IAH) is also a problem when indirect measures of global oxygen transport such as $ScvO_2$ and serum lactate are used in sepsis evaluation. Because global oxygen delivery indicators reflect total body oxygen consumption, early ischemia may not dramatically affect the measurements. Regional hypoperfusion is often not recognized until very late in the disease state.[26-28] On the other hand, regional perfusion indicators such as tonometry or near infrared spectroscopy and broad splanchnic indicators of hypoperfusion such as IAP will detect ischemia (or the result of ischemia) in the splanchnic bed earlier in the clinical course- before it is detectable with global indicators.[28-31] These findings suggest that a very inexpensive, non-invasive piece of clinical data - intra-abdominal pressure monitoring - provides an earlier warning regarding splanchnic hypoperfusion risk before mixed central venous oxygen saturation, lactic acid and other global indicators are measurably altered. Until more direct methods such as tonometry and near infrared spectroscopy are perfected, IAP monitoring should be considered as an early indicator of splanchnic hypoperfusion in these high risk patients.[28, 30, 31]

Commonly utilized renal perfusion measurements such as urine output, blood urea nitrogen and creatinine are also adversely impacted by IAH.[20, 32] Abnormalities in these parameters might be misinterpreted as pre-renal azotemia when in reality they may occur due to direct visceral compression and reduced cardiac output due to IAH/ACS. In this situation, further fluid administration could worsen the problem while interventions directed towards reducing IAP may resolve or partially limit the issue.[5, 33-35]

This data is not meant to discourage the use of CVP, ScvO2 or urine output. Each piece of information when used with others, e.g. vital signs, urine output, CVP, ScvO2, lactate, and intra-abdominal pressure more adequately complete the big picture, improve patient care and support the clinician in their decision making process.

Sepsis and IAH: Resource consumption and outcomes

Too date there are no outcomes trials specifically looking at sepsis patients with IAH to determine whether interventions impact final outcome. However, there does exist a large body of data in broadly based critical care populations (including a substantial percentage of severe sepsis patients) which demonstrates that the presence of IAH worsens patients outcomes and increases resource consumption.[7, 8, 25, 32, 36] Additional data demonstrates that aggressive protocol driven interventions designed to treat IAH can improve outcomes without increasing resource utilization.[33, 37-40] Malbrain et al, found that even "mild" elevations of IAP (>12 mmHg) in a mixed population of ICU patients (1/3 suffered severe sepsis) was an independent risk factor for organ dysfunction and mortality (39% vs 22%).[7] Ejike et al found a similar but more pronounced mortality link in children (33% vs 2.4%) with an associated increase in ICU length of stay (13 days vs 6 days).[36] Sugrue et al showed elevated IAP (over 18 mm Hg) was an independent predictor of renal failure, ranking up with hypotension, age and sepsis in a large cohort of surgical ICU patients.[32] Even though their study was not powered to detect outcome differences, Daugherty et al noted that the majority of patients in their medical ICU population who developed IAH were patients with septic shock and that patients with IAH had a higher mortality (70% vs 50%, p=NS) and longer ICU length of stay (21 day vs 12 day, p=NS).[8] Reintam et al noted that 45% of their severe sepsis population developed IAH and that IAH presence was a marker for higher mortality compared to those septic patients who did not develop IAH (sepsis plus IAH mortality 50%, sepsis without IAH mortality 19%; OR 4.15, 95% CI 1.87-9.26). Multiple other studies show correlations between morbidity, mortality and resource utilization in patients with IAH/ACS.[41, 42]

Given the clear link between elevated IAP and mortality, the important question that remains is whether or not intervention will improve outcome for an acceptable cost. While urgent, aggressive treatment for other compartment syndromes (increased intra-cranial pressure, tension pneumothorax, pericardial tamponade, extremity compartment syndrome) is standard of care, most intensive care practitioners fail to treat intra-abdominal hypertension and the abdominal compartment syndrome with the same urgency if at all.[6] Because this syndrome leads to profound cellular ischemia and inflammation it is likely that optimal outcome improvements can only be recognized if

the problem is prevented or at the very least identified early by utilizing monitoring and proactive interventions. Emerging evidence suggests such an early, proactive approach does result in better patient outcomes. Almost a decade ago Ivatury et al showed that prophylactic interventions (temporary abdominal closure) to reduce IAH/ACS in major trauma cases led to dramatic outcome improvement (ACS reduction from 52% to 22%, death reduction from 36% to 11%).[38] The technique of temporary abdominal closure to prevent abdominal compartment syndrome is now a standard surgical approach and leads to clear outcome improvements in high risk trauma, pancreatitis and vascular surgical populations.[38, 39, 43] Joseph et al noted that IAP monitoring and treatment with laparotomy was instrumental in improving outcome in their neurotrauma/stroke patients with elevated intra-cranial pressure. Today, Joseph's group now advocates aggressive IAP monitoring and interventions to prevent IAP elevation over 20 mm Hg in all patients with elevated intra-cranial pressure.[44] In the severe pancreatitis population, Oda et al demonstrated that aggressive IAP monitoring with implementation of early renal replacement therapy once IAP increased to more than 15 mm Hg resulted in a reduction in their traditional mortality (>30% down to 6%).[33] Sun et al, in a prospective randomized trial, were able to cut hospital length of stay in half and reduced pancreatitis mortality from 20% to 10% using IAP monitoring to guide interventional strategies.[40] Cheatham et al noted that protocol driven interventions which combine both early medical therapy with aggressive surgical interventions result in improvements in outcomes without increasing hospital resource utilization.[37] Cheatham's group introduced an evidence-based algorithm to guide the care of their patients with severe IAH/ACS, demonstrating a mortality decrease from 49% to 29% and a reduction in hospital length of stay from 29 day to 18 days. They conclude that evidence based management strategies for IAH/ACS lead to significant improvements in survival with earlier return to normal function. They also point out that these outcome improvements come with no added resource utilization and recommend that all institutions adopt an evidence based treatment protocol (available at www.wsacs.org/allgorithms.php). In systems where ICU beds are at a premium, implementation of protocol driven IAP monitoring/intervention may provide a method of decreasing length of stay, assist in more rapid bed turnover and less expenditures.

Should intra-abdominal pressure monitoring occur in severe sepsis?

Though it is clear that severe sepsis and septic shock are major risk factors for IAH/ACS, the data evaluating interventional therapy to improve outcomes in sepsis are scarce. However, the preponderance of circumstantial evidence suggests it should be considered. On initial presentation septic shock patients are volume depleted and will be unlikely to have IAH/ACS. However, as EGDT is implemented and patients are fluid resuscitated their intra-vascular and interstitial fluids are in a state of rapid fluctuation creating an ever-changing hemodynamic picture. Because end-expiratory CVP is the primary measurement used in EGDT to assess fluid status the accuracy of CVP is critical to ensure adequate but not over-zealous fluid administration. Unfortunately, end-expiratory CVP overestimates fluid status in patients suffering from IAH.[15] Furthermore, IAH causes splanchnic hypoperfusion long before lactate elevation or ScvO2 changes are apparent and may further compromise renal function independent of the patients' fluid status.[28-31] Last but not least – IAH/ACS is very common in resuscitated sepsis patients. In fact, the highest reported incidence of IAH/ACS occurs in the septic shock population.[8, 10] For these reasons early IAP measurements and trending beginning during the resuscitation phase and continuing during the management phase of sepsis care may be beneficial. By utilizing IAP in conjunction with more traditional measures the clinician can more accurately interpret fluid status in relationship to the CVP, becoming aware of the confounding physiologic impact of IAP on potential organ dysfunctions and may be given an early warning that splanchnic perfusion is being compromised. This will allow for more timely medical interventions to temporize or eliminate IAH/ACS and facilitate a more aggressive resuscitation. Fortunately, multiple non-surgical interventions are available and their early implementation can reduce IAP and prevent progression to ACS.[5] Figure 4 & 5 provide a recommended therapeutic approach for IAH developed by an international consensus panel of experts on the disease.[5] By failing to monitor IAP early in the hospital course, profound and prolonged IAP elevations may occur causing delayed recognition of the problem, prolonging cellular ischemia and oxygen debt and possibly preventing the success of early interventions that may temporize or stop progression of IAH.

Figure 3 & 4: Recommended therapeutic approach for IAH (Provided by the World Society of Abdominal Compartment syndrome under license from Wolfe Tory Medical)

Summary:

Patients' resuscitated from severe sepsis and septic shock are at high risk for the development of IAH/ACS, resulting in a very high incidence of the syndrome. Furthermore, elevated intra-abdominal pressure profoundly influences commonly used septic shock resuscitation endpoints such as CVP (falsely elevated) and urine output (markedly decreased). Failure to adjust for the impact of IAP on these endpoints may negatively impact resuscitation decisions. Finally, in other critically ill patient populations, utilization of IAP to direct therapy early in their care results in improved outcomes and reduced resource consumption. This data combined with the well described organ dysfunction and tissue ischemia found in patients with IAH/ACS suggest that it is time to consider routine IAP monitoring as a primary measurement component of the bundled care in severely septic patients. At the very minimum, this information provides a hypothesis deserving of further investigation into volume resuscitation effects as well as the impact of early IAP monitoring and therapy on the outcome of severely septic patients.

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