

Managing exsanguination: what we know about damage control/bailout is not enough

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Exsanguination awaits better definition, not only clinically but also physiologically and biochemically. Asensio (1–4) has described it as the most extreme form of hemorrhage, with an initial blood loss of >40% and ongoing bleeding that, if not surgically controlled, will lead to death. Although trauma surgeons recognize exsanguination as a syndrome (1–19), its multifactorial effects on the cell, microcirculation, inflammatory cascades, and temperature-dependent enzymatic functions of both platelets and the coagulation pathways remain to be defined (1–7). Shock-causing cardiopulmonary arrest or massive injuries responsible for blood loss exceeding >40% of total blood volume with ongoing hemorrhage/exsanguination set in motion poorly understood cellular and subcellular derangements manifested by the cycle of core hypothermia, unrelenting acidosis, and coagulopathy, which if uninterrupted rapidly leads to uncontrollable dysrhythmia, “the declamping syndrome,” and death (1–19).

Profound shock and/or massive injuries responsible for large blood losses quickly initiate the cycle of hypothermia, acidosis, and coagulopathy (1, 8–13) described by Moore (11) as the “bloody vicious cycle.” A fourth component of this cycle is dysrhythmia, which usually heralds the patient’s demise and has been described by Asensio as the “lethal tetrad” (1, 14–16). Recognizing that the bloody vicious cycle must be interrupted, Stone et al described the “bailout” approach, ushering in the era of staged surgical procedures for the management of severely injured patients (8). This approach was later refined by Rotondo et al with the goal of returning the patient to the operating room after all physiological derangements had been corrected (9).

Stone et al’s hallmark work describing the bailout approach did not, however, provide intraoperative parameters, other than observation of clinical coagulopathy, to select patients for this approach (8). Postoperatively, his protocol focused on coagulation measurements—prothrombin time (PT), partial thromboplastin time (PTT), clotting time, platelet count, and fibrinogen levels—as the only predictive factor of when to return the patient to the operating room. In addition to considering coagulation derangements, Phillips identified patients who sustained massive transfusion exceeding 2 times their estimated blood volume as patients at risk of the development of organ failure, focusing on the volume of blood transfusion of 25 units (17).

After studying a series of 200 patients treated with unorthodox techniques to interrupt laparotomy and the triad of hypothermia, acidosis, and coagulopathy, Burch et al proposed a model

based on clinical and laboratory parameters including core temperature of $\leq 32^{\circ}\text{C}$, $\text{pH} \leq 7.09$, and a mean volume of packed red blood cell (PRBC) transfusions of 22 units (10). They postulated that this model could predict 48-hour survival in critically injured patients based on a linear regression model that identified PRBC transfusion rates of approximately 12.5 units per hour and pH. Furthermore, they advanced the concept of abbreviated laparotomy as a rational approach to an apparently hopeless situation. Sharp and Locicero reviewed results in 39 patients (including 31 who sustained massive hepatic injury) and proposed a model consisting of objective parameters such as temperature $\leq 33^{\circ}\text{C}$, $\text{pH} \leq 7.18$, $\text{PT} \geq 16$ seconds, $\text{PTT} \geq 50$ seconds, and mean transfusion volume ≥ 10 units of blood to indicate the need for early packing (18).

Rotondo et al described a multiphase approach to the management of exsanguinating patients sustaining abdominal injury. However, in their group of 46 patients, they could not identify any statistical differences between the 22 patients subjected to definitive laparotomy and the 24 patients subjected to damage control laparotomy. The authors then identified a maximum-injury subset consisting of 22 patients, 9 of whom were subjected to definitive laparotomy and 13 of whom underwent damage control laparotomy. In this group of patients, the survival rate in the damage control group was 77% vs 11% in the definitive laparotomy group (9).

On the basis of their findings, Rotondo et al recommended 3 phases for patient management: 1) interruption of laparotomy, 2) return to the intensive care unit for volume resuscitation, with particular attention to the resolution of acidosis and coagulopathy, and 3) return to the operating room for removal of packing and completion of definitive surgical repairs. They concluded that damage control was a promising approach to increasing survival in exsanguinating patients (9).

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Interestingly, the authors did not identify any objective parameters during the intraoperative phase of damage control. They simply described their approach: "When in the judgment of the senior surgeon, signs of intraoperative coagulopathy developed, intraperitoneal packing was applied to sites of nonsurgical bleeding as well as persistently bleeding visceral injuries. The procedure was terminated and remaining definitive repairs were deferred" (9). Recently, Johnson et al confirmed Rotondo's original findings and recommendations based on a series of 24 patients from the same institution (19).

Morris et al described a series of 107 patients who underwent staged celiotomy with abdominal packing and focused on the indications and timing of reconstruction, criteria for emergency return to the operating room, complications after reconstruction, and the abdominal compartment syndrome (12). They proposed proceeding with damage control celiotomy early in the course of the operation based on a patient's temperature $<35^{\circ}\text{C}$, a base deficit <14 , and the presence of medical bleeding.

Moore described progressive coagulopathy as the most compelling reason for staged laparotomy and analyzed factors predictive of a severe coagulopathic state: intraoperatively measured PT and PTT >2 times normal, massive and rapid blood transfusion exceeding 10 units in 4 hours, persistent cellular shock defined as an oxygen consumption index (VO_2I) <110 mL/min/ m^2 , lactic acid level >5 mmol/L, pH <7.2 , base deficit >14 , and core hypothermia $<34^{\circ}\text{C}$.

Subsequently, Cosgriff et al postulated that the ability to predict the onset of coagulopathy, one of the very important components of the "bloody vicious cycle," would have significant decision-making implications with regards to the institution of damage control (13). Their predictive model for life-threatening coagulopathy included a systolic blood pressure <70 mm Hg, temperature $<34^{\circ}\text{C}$, pH <7.10 , and Injury Severity Score (ISS) ≥ 25 .

Clearly, no single model can accurately predict the timing for institution of damage control (1, 8–13, 17–19). A pH of <7.09 or 7.10 or a core temperature of $<33^{\circ}\text{C}$ may indicate that the "bloody vicious cycle" is too far advanced to be interrupted. Similarly, intraoperatively measured PT, PTT, fibrinogen, and lactic acid levels are difficult to obtain. Results are not returned quickly enough, and the laboratory studies are unavailable in the operating rooms of some of America's busiest trauma centers. ISS, which was proposed in some models (11), is clearly not a usable parameter intraoperatively.

Recently, Asensio and colleagues reported a 6-year retrospective study involving 548 patients admitted with the diagnosis of exsanguination (1). Criteria for inclusion in this study were minimal estimated blood loss ≥ 2000 mL during a trauma operation, minimal administration requirements of ≥ 1500 mL of PRBCs during initial resuscitation, and the diagnosis of exsanguination. Data collected included demographics; prehospital and admission vital signs and physiologic predictors of outcome; Revised Trauma Score, Glasgow Coma Scale score, and ISS; volume of resuscitative fluids and the need for thoracotomy in the emergency department; volume of fluids administered in the operating room and the need for thoracotomy in the operating room; organs injured; and intraoperative complications. Eighty-two percent of injuries were penetrating, with the majority of those (78%) being gunshot wounds. For all the patients, the mean

Revised Trauma Score was 4.38, and the mean ISS was 32, denoting a physiologically compromised and severely injured patient population. In this series, patients had a mean pH of 7.15 and mean temperature of 34.3°C in the operating room and received an average of 14,165 mL of crystalloids, blood, and blood products (1).

Of the 548 patients, 449 arrived at the operating room alive but in critical condition. Of those, 281 died, for a mortality rate of 63%. Alternatively, it could be stated that 37% of these patients survived who might not have otherwise lived without institution of damage control. Unusual survivors included a patient who had a pH of 6.76, another with a temperature of 32°C , and another who required a total of 55 L of fluids and blood during his emergency department and operating room course (1).

On the basis of their extensive experience, the authors proposed a model consisting of easily followed, objective intraoperative parameters as predictors of outcome and guidelines for when to institute damage control (1). Parameters in this model include operating room temperature of $\leq 34^{\circ}\text{C}$, pH ≤ 7.2 , serum bicarbonate level ≤ 15 mEq/L, transfusion volume ≤ 4000 mL of PRBCs, total blood replacement ≤ 5000 mL if both PRBCs and whole blood were used, and total operating room fluid replacement including crystalloids, blood, and blood products $\leq 12,000$ mL. All of these predictors of outcome were statistically validated and considered as the absolute upper limits that would be acceptable prior to institution of damage control.

We strongly recommend close monitoring of intraoperative predictors of outcome as validated within our guidelines and recommend following our model for institution of damage control as early as possible and definitely prior to reaching the upper limits of these parameters. We cannot overemphasize the need for the earliest possible interruption of the initial surgical procedure, especially in patients who have an estimated blood loss that approximates 5000 mL and those sustaining injuries that are known to cause exsanguination (1–7, 14–16).

The most important goal of early institution of damage control is patient survival (1–19). These patients are then returned to the operating room when physiological derangements such as acidosis, hypothermia, and coagulopathy have been corrected (1–19). Frequently, these patients experience posttraumatic open abdomen as a logical extension of the damage control procedure. The management of posttraumatic open abdomen is quite challenging, as these patients continue to lose significant amounts of fluid and heat through their open abdomens, which are often covered by laparotomy packs and a plastic intravenous bag. This exposes them to the development of gastrointestinal tract fistulas, further complicating their fluid management (1).

Institution of damage control implies immediate control of life-threatening hemorrhage, placement of chest tubes, thoracic packing if needed, closure of the skin if the chest has been opened, hepatic packing, temporary duodenal and hollow viscus closures or rapid stapled resections, drainage of pancreatic injuries, rapid stapled resection of pancreatic injuries if present to the left of the superior mesenteric artery, rapid splenectomy and nephrectomy or occlusion of their vascular pedicles with a vascular clamp left in situ, use of intraluminal shunts, and judicious abdominal packing with temporary abdominal wall closures (1, 14–16).

We recognize that significant research remains to be done to better understand the cellular and subcellular mechanisms triggered by profound shock, exsanguination, acidosis, hypothermia, and coagulopathy. With awareness of these guidelines (7), we have been able to objectively and statistically validate our model, detect improvements in some predictors of outcome, and improve the time for closure of the posttraumatic open abdomen. However, we have not been able to decrease mortality rates in these patients. Thereby, the ongoing challenge is to continue to identify better predictors of outcome, improved means of resuscitation, greater understanding of the physiological derangements incurred by these patients, and most importantly better timing to institute damage control. Only then can we begin to reduce the high mortality rates experienced by these patients.

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