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DAMAGE CONTROL RESUSCITATION IS ASSOCIATED WITH A REDUCTION IN RESUSCITATION VOLUMES AND IMPROVEMENT IN SURVIVAL IN 390 DAMAGE CONTROL LAPAROTOMY PATIENTS

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Abstract

OBJECTIVE—To determine if implementation of damage control resuscitation (DCR) in patients undergoing damage control laparotomy (DCL) translates into improved survival.

SUMMARY BACKGROUND DATA—DCR aims at preventing coagulopathy through permissive hypotension, limiting crystalloids and delivering higher ratios of plasma and platelets. Previous work has focused only on the impact of delivering higher ratios (1:1:1).

METHODS—A retrospective cohort study was performed on all DCL patients admitted between 01/2004–08/2010. Patients were divided into pre-DCR implementation and DCR groups, and were excluded if they died prior to completion of the initial laparotomy. The lethal triad was defined as immediate post-operative temperature <95° F, INR>1.5, or a pH<7.30.

RESULTS—390 patients underwent DCL. Of these, 282 were pre-DCR and 108 were DCR. Groups were similar in demographics, injury severity, admission vitals and laboratory values. DCR patients received less crystalloids (median 14 L vs. 5 L), RBC (13 U vs. 7 U), plasma (11 U vs. 8 U) and platelets (6 U vs. 0 U) in 24-hr; all $p<0.05$. DCR patients had less evidence of the lethal triad upon ICU arrival (80% vs. 46%, $p<0.001$). 24-hour and 30-day survival were higher with DCR (88% vs. 97%, $p=0.006$ and 76% vs. 86%, $p=0.03$). Multivariate analysis controlling for age, injury severity, and ED variables, demonstrated DCR was associated with a significant increase in 30-day survival (O.R. 2.5, 95% C.I. 1.10–5.58, $p=0.028$).

CONCLUSION—In patients undergoing DCL, implementation of DCR reduces crystalloid and blood product administration. More importantly, DCR is associated with an improvement in 30-day survival.

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Description: This is a retrospective cohort study evaluating the impact of implementing the Damage Control Resuscitation components of permissive hypotensive and limited crystalloid resuscitation into a mature trauma center already practicing the 1:1:1 red blood cell: plasma: platelet transfusion strategy.

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Keywords

trauma; laparotomy; damage control; resuscitation; transfusion; crystalloids

INTRODUCTION

Beginning with its early descriptions by Stone, Feliciano and others and codified by Rotondo et al, damage control laparotomy (DCL) allows the trauma surgeon a viable option in dealing with the exsanguinating trauma patient. (1–5) When first described, DCL was performed in approximately 5% of trauma laparotomies. DCL is an abbreviated, resuscitative surgical approach with the primary goal being the rapid control of hemorrhage and contamination, focused on restoring normal physiology at the expense of normal anatomy. Patients often undergo temporary closure of the abdominal wall and are rapidly transported to the intensive care unit (ICU) for correction of coagulopathy, hypothermia, and acidosis. (6) At the same time the DCL process was being popularized, blood bankers replaced whole blood with component therapy. (7) This change occurred without adequate clinical efficacy data. Simultaneously, Carrico and Shires proposed that crystalloid therapy was an effective initial resuscitative fluid. Unfortunately clinicians used this cheap and ubiquitous product indiscriminately. At the same time, Shoemaker and colleagues established the concept of supra-normal resuscitation and argued for the use of large volumes of crystalloid in resuscitating trauma patients. (8, 9) While this practice led to higher oxygen delivery, it also increased extravasation of fluid, tissue edema, and higher overall blood pressures. (10) Taken together, these separate but convergent processes coalesced to generate an iatrogenic resuscitation injury, resulting in edematous patients, an epidemic of open abdomens and significant mortality and morbidity. (6, 11)

In 2005, damage control resuscitation (DCR) was developed by military clinicians who had seen first-hand the benefits of whole blood-based resuscitation in severely injured patients. (12, 13) DCR soon gained popularity in the civilian centers as combat surgeons demonstrated how patients resuscitated with this approach were less edematous and coagulopathic post-operatively. DCR aims at preventing or reversing coagulopathy through permissive hypotension, limiting crystalloids and delivering higher ratios of plasma and platelets. Subsequently, multiple authors have demonstrated favorable outcomes associated with plasma and platelet ratios approximating whole blood including decreases in costs of care, blood product utilization, organ failure rates, open abdomens, and six and 24-hour and 30-day mortality. (14–18) While significant attention has been paid to the ratios of plasma and platelets, insufficient focus has been paid to the other two components of DCR: permissive hypotension and minimizing crystalloid-based resuscitation. Given the severity of injury and gross disturbances in physiology associated with the DCL patient, we chose this population to assess the full impact of the application of DCR on survival. Therefore, the purpose of the current study was to determine if implementation of all three tenets of DCR would translate to improved survival in DCL patients.

METHODS

Study setting

Memorial Hermann Hospital is an American College of Surgeons verified level I trauma center that is the primary teaching hospital for the University of Texas Health Science Center. Memorial Hermann is one of only two level-1 trauma centers in Houston, Texas, the fourth largest city in the United States. The hospital is an 800-bed facility within Texas Medical Center. The trauma center currently admits over 5,000 trauma patients annually with the most severely injured cared for in our 23-bed Shock-Trauma ICU.

Selection of Subjects

Approval was obtained from the University of Texas at Houston Institutional Review Board. This was a single-center, retrospective cohort study of trauma patients admitted to Memorial Hermann Hospital using the institution's Trauma Registry of the American College of Surgeons (TRACS) database. We evaluated all adult trauma patients admitted between January 2004 and August 2010 who underwent immediate exploratory laparotomy. These were defined by proceeding directly from Emergency Department (ED) to Operating Room (OR). We then evaluated only the subgroup of these patients who were managed by damage control laparotomy techniques (DCL). In accordance with the expedited approval process, protected populations such as minors (age <18), prisoners, and pregnant women were excluded from this study. Patients were also excluded from analysis if they received ≥5 minutes cardiopulmonary resuscitation (CPR) prior to the OR or died in the ED or OR during their procedure.

While our institution has long been using higher plasma and platelet ratios in our resuscitations, an emphasis on the other two critical components of DCR (permissive hypotension, minimizing crystalloid administration) was not practiced prior to 2009. (19, 20) Beginning in January 2009, our faculty began to champion the adoption and consistent implementation of these other two components. From this time point, DCL resuscitations were discussed at our Morning Report (sign-out) among the Trauma and Surgical Critical Care faculty, fellows, and residents. Non-compliance and "outliers" were identified in a real-time fashion and feedback provided to all involved parties (Pre-hospital, ED, OR, Trauma, and Surgical Critical Care personnel). This was followed with structured and directed educational conferences, Grand Rounds presentations, and individual provider educational interventions consistent with previously published data. (21) As such, two groups were generated for comparison. The pre-DCR group consisted of those patients who were managed prior to DCR implementation (01/01/2004 to 12/31/2008). The DCR group was made up of those patients who were managed after implementation of DCR (01/01/2009 to 08/31/2010).

Data collection

Data collected included demographics, mechanism of injury, abbreviated injury scores, injury severity scores, specific injury profiles, procedures performed, fluids and blood products administered, vital signs, blood gases, coagulation profiles, intra-abdominal pressures, time to closure, complication rates, and 24-hour and 30-day survival.

Definitions

DCL was defined as any emergent trauma laparotomy that resulted in temporary abdominal closure, regardless of procedures performed or placement of abdominal packing. Emergent trauma laparotomies were identified if the primary injury was traumatic in nature and if the patient went to the operating room directly from the Emergency Department. As no objective DCL protocol was in place, the decision to implement damage control was dependent on the surgeon's gestalt in the emergency department or operating room.(11) In all cases the patients were resuscitated in the intensive care unit before attempting a second-look operation. The take-back operation afforded the opportunity to perform definitive repairs and, if possible, primary fascial closure. Primary fascial closure in this study was defined as approximation of the fascial edges and does not include synthetic or biological mesh closure without fascial approximation.

The identification of *prehospital vital signs* was obtained through a review of ground or air transport documentation. *ED vital signs* were defined as the initial set of vital signs captured and documented in the trauma bay. All patients had a single comprehensive *ED laboratory*

panel obtained in the ED. The results of these labs were used for populating the ED laboratory value data fields through an electronic medical records data query. *OR vital signs* were the initial set of vital signs documented in the electronic medical records. Similarly, the *OR laboratory results* were defined as the initial values obtained after arrival in the operating theater. The *ICU vital signs and laboratory values* were defined as the initial values obtained immediately after patient arrival in the ICU. *Peak intra-abdominal pressures (IAP)* were defined as the highest transduced bladder pressure recorded in the electronic medical record prior to the first take-back.

Prehospital, ED, and OR crystalloid administration were defined as the sum of all normal saline, lactated Ringer's solution, and plasmalyte received while in these locations. Intra-operative colloids (albumin, THAM, and hetastarch) were also recorded. Prehospital, ED, and OR blood products (red blood cells, plasma, and platelets) were defined as those products received while in these hospital locations. 24-hour blood product calculations were defined as the total number of products received 24 hours from time of arrival to the hospital. This included blood in the trauma bay, operating room, and post-operatively up to the 24-hour post-admission time point.

The lethal triad was defined by the initial laboratory values and vital signs upon arrival to the ICU. *Acidosis* was defined by an initial pH <7.30. *Hypothermia* was defined as temperature <95.0 degrees Fahrenheit. *Coagulopathy* was defined as an INR value >1.5.

Organ dysfunction and multiple organ failure were defined using the Denver Multiple Organ Failure (MOF) scoring system. (22) This system evaluates four organ systems: pulmonary, hepatic, renal, and cardiac. Organ dysfunction is graded on a scale from 0 to 3. The pulmonary score is determined by the PaO2/F1O2 (P/F) ratio. P/F ratios greater than 208 received a zero (0) points, while ratios of 208-165, 165-83, and 83 received 1, 2, and 3 points, respectively. The renal system was graded by serum creatinine level in mg/dL: 0 points for <1.8, 1 point for 1.8–2.5, 2 points for 2.5–5.0, and 3 points for >5.0 mg/dL. The hepatic score was calculated by total serum bilirubin level in mg/dL: 0 points for bilirubin <2.0, 1 point for 2.0–4.0, 2 points for 4.0–8.0, and 3 points for bilirubin >8.0 mg/dL. Cardiac dysfunction was graded based on inotropic support and cardiac index (C.I.). No inotropes and cardiac index >3.0 L/min per meter squared yielded a score of zero (0), whereas minimal inotropic support or C.I. <3.0 yielded a score of 1. Moderate and high dose inotropic received scores of 2 and 3, respectively. Scores that were not recorded were assumed to be normal and were calculated as zero (0). For *multiple organ failure*, the MOF score was calculated as the sum of the simultaneously obtained individual organ scores on each hospital day. *Single system organ failure* was defined as an organ failure grade greater than 0, and MOF was defined as a total score of 4 or higher. (23, 24)

Hospital length of stay (in days), ICU length of stay (in days), and ventilator-free days, are expressed in calendar days. Ventilator free-days were calculated by the days alive (up to 28 days) and free of the ventilator. (25, 26) The incidences of 24-hour and 30-day mortality were recorded and evaluated from the in-patient and clinic charts.

Statistical analysis

Continuous data are presented as medians with 25^{th} and 75^{th} inter-quartile range (IQR) with comparisons between groups performed using the Wilcoxon rank sum (Mann-Whitney U test). Categorical data are reported as proportions and, where appropriate, tested for significance using χ^2 or Fisher exact tests. The primary data analysis evaluated 24-hour and 30-day survival. Secondary outcomes evaluated the fluid administration volumes and the number of red blood cells (RBC), plasma, platelets, and cryoprecipitate. As well, we evaluated the incidence of lethal triad components.

Purposeful regression modeling was then used to construct a multivariate logistic regression model evaluating 30-day survival. This was done using the technique of purposeful selection of covariates described by Hosmer and Lemeshow. (27) In an effort to minimize the risk of falsely identifying significant results with multiple comparisons, all variables were pre-specified and judged *a priori* to be clinically sound. These independent variables included age, gender, injury severity (ISS), ED vitals and labs, pre-hospital and hospital fluid administration and transfusions. Following this the variables were entered into step-wise regression that generated four variables of significance (age, ED systolic blood pressure, ED base deficit, ED INR value). These were then applied to a multivariate logistic regression analysis evaluating these four variables and exposure to DCR. STATA Statistical software (version 10.1; College Station, TX) was used for analysis.

RESULTS

During the six-year study period, 25,069 adult trauma patients were admitted. 1514 underwent emergent laparotomy for trauma, while 197 met our predetermined exclusion criteria, leaving 1317 patients for analysis during the study period. (FIGURE 1) 927 patients underwent a single-staged procedure and 390 (30%) underwent DCL. The rates of DCL were consistent between the time frames, with a range of 29–37% for 2004–2008 and 29–36% for 2009–2010. These 390 DCL patients were the focus of this resuscitation study. Among these DCL patients, 282 (72%) were admitted prior to DCR implementation (pre-DCR group) and 108 (28%) were admitted during the DCR period (DCR group). This equated to approximately 4.6 DCL patients per month during the pre-DCR period and 5.4 DCL patients per month during the DCR period.

Demographics and injury severity were similar between the two DCL groups. (TABLE 1) While the pre-hospital systolic blood pressures were the same, the blood pressure in the DCR group was lower on arrival to the ED and to the OR. (TABLE 2) As well, the field and ED arrival GCS were lower in the DCR patients compared to the pre-DCR group. Interestingly, the HR was higher in the pre-DCR group. Consistent with the tenet of minimizing crystalloids, the DCR group received less fluid in the field, in the ED, and in the OR. Despite this, the DCR group had less base deficit in the ED and OR. The DCR patients also received more ED plasma but less intra-operative blood products. Intra-operative times were also similar between the groups (median time of 98 minutes in the DCR group vs. 109 minutes pre-DCR; $p=0.130$). However, in-hospital use of recombinant factor VIIa was significantly lower in the DCR group (6% vs. 17% in the pre-DCR patients; $p<0.001$).

Consistent with our practice across the time frames, there were no differences in early (ED and OR) ratios of PRBC: plasma (median ratio of 1.08 in DCR vs. 1.19 pre-DCR group; $p=0.785$). Evaluating by years, 2004–2008 had a PRBC: plasma range of 1.01–1.35, while 2009–2010 had a range of 1.03–1.19. As for other tenets of DCR, there was less change from ED arrival to ICU arrival blood pressures in the DCR group compared to pre-DCR (median delta SBP 1 mmHg with IQR -16, 24 vs. 18 mmHg with IQR -7, 47; $p<0.001$). Despite receiving less crystalloid fluid volumes and less blood products in the operating room, DCR patients arrived to the ICU warmer, less acidotic, and less coagulopathic. (TABLE 3) As such, DCR patients had less evidence of the lethal triad upon ICU arrival (80% vs. 46%, $p<0.001$). The range for the pre-DCR years was 78–87% and that of the DCR years was 44–51%. The DCR group was also less likely to have all three components of the triad present upon ICU arrival (3% vs. 12%, $p<0.001$). Importantly, 24-hour crystalloid volumes, as well as RBC, plasma, and platelet transfusions were reduced after implementation of DCR. Though numeric reductions were observed in acute renal failure, ARDS, and multi-organ failure among the DCR population, these values trended towards but did not reach statistical significance.

Univariate analysis demonstrated improvement in 24-hour and 30-day survival among those on the DCR group. (TABLE 3) Prior to 2009, the 30-day mortality rates were consistent and were in the range of 23% –27%. A multivariate logistic model was then developed predicting 30-day survival (dependent variable). After controlling for age and arrival base deficit, INR and blood pressure, DCR was independently associated with a 2.5 fold increased odds of 30-day survival. (TABLE 4)

After demonstrating favorable outcomes in the DCL group, we felt obliged to examine the safety of the DCR approach in the non-DCL group. Evaluation of these 927 single-stage laparotomies (719 pre-DCR patients and 208 DCR patients) demonstrated similar findings as the DCL patients, although not as robustly. Demographics and injury severities were also similar as were pre-hospital and ED arrival vital signs. The non-DCL and DCR population, however, received less fluids in the *pre-hospital* (median 0.5 L, IQR 0.1 and 1.2 vs. 0.8 L, IQR 0.3 and 1.2; p=0.029), *Emergency Department* (median 1.0 L, IQR 0.1 and 1.3 vs. 1.0 L, IQR 0.6 and 2.0L; p<0.001), and *Operating Room* settings (median 2.5 L, IQR 1.8 and 3.5 vs. 3.0 L, IQR 2.0 and 4.5; p<0.001). The DCR group arrived to the ICU less acidotic *by pH* (median 7.39, IQR 7.33 and 7.44 vs. 7.32, IQR 7.26 and 7.37; p<0.001), *base deficit* (median -4, IQR -6 and -2 vs. 0, IQR -4 and 4; p<0.001) *and lactate measurements* (median 1.8, IQT 1.0 and 3.7 vs. 5.2, IQR 4.0 and 7.5; p<0.001). With respect to the lethal triad, the DCR group also arrived less hypothermic (median 98.2, IQR 97.6 and 98.7 vs. 97.1, IQR 95.0 and 98.6; p<0.001), as well as less coagulopathic by *INR* (median 1.32, IQR 1.18 and 1.48 vs. 1.36, IQR 1.21 and 1.58; p=0.009) *and fibrinogen values* (median 290, IQR 194 and 472 vs. 167, IQR 144 and 222; p<0.001). There were no differences in *ED* (median 0 U, IQR 0 and 2 vs. median 0 U, IQR 0 and 2; p=0.181), *OR* (median 1 U, IQR 0 and 6 vs. median 1 U, IQR 0 and 4; p=0.299) and *24-hour* (median 2 U, IQR 0 and 8 vs. median 2 U, IQR 0 and 8; p=0.988) RBC transfusions. In addition, there were no differences in plasma or platelet transfusions at these points and the 30-day survival was similar between groups (97.3% vs. 97.1%, p=0.878).

DISCUSSION

Numerous authors have demonstrated favorable outcomes with the DCR approach to resuscitation.(14–18, 20, 28–30) However, these publications have focused primarily on the plasma and platelet ratio component of DCR while ignoring the other tenets. In addition, several busy centers have questioned the survival benefit of these ratios. These authors have suggested that patients achieving 1:1:1 ratios are simply living long enough to receive them and those who die early are not alive long enough to receive 1:1:1. (31, 32) As early as 2002, we had already began delivering higher ratios of plasma and platelets, but we have now demonstrated that the prospective application of limited crystalloid and permissive hypotension is possible and is associated with improved outcomes.(19) When all three tenets of DCR are applied, despite the use of early and high ratio plasma and platelets, a reduction in overall RBC, plasma, and platelet use is observed. More importantly, we noted an associated increase in 24- hour and 30-day survival. This survival difference remained even after controlling for age and arrival vital signs and laboratory values.

Large volume crystalloid-based resuscitations are associated with increased frequency of and longer time to recovery from acute respiratory distress syndrome and higher mortality. (6, 33, 34) Systemically, the incidence of pulmonary complications, gastrointestinal dysmotility, and coagulation disturbances are higher as the volume of early crystalloid infusion is increased. Even when controlling for shock and severity of injury, the volume of crystalloid infused is associated with increased risk of developing intra-abdominal hypertension and abdominal compartment syndrome. (11, 35–42) In addition, investigators from L.A. County have recently demonstrated that infusing as little as ten liters of

crystalloids in the first three days after injury is associated with a 5-fold increased risk for colonic anastomotic leaks. (43) Moreover, when Ley and colleagues evaluated their trauma outcomes, they found that as little as 1.5 liters of fluid in the ED was associated with a 2-fold increased risk in mortality (even after controlling for ISS and arrival vital signs). (44) Through a dedicated and recurring educational process, we were able to reduce the amount of crystalloids infused in the pre-hospital, ED, OR and ICU settings. By using plasma as the primary resuscitation fluid and broad application of DCR principles, we noted an associated decrease in 24-hour crystalloids administration by almost nine liters in the first 24 hours after admission. Intuitively, one would assume that as crystalloid use went down that blood product use would increase. However, we found just the opposite. All blood product use decreased over the same time frame. Coincident with this change in resuscitative fluids, all measured physiologic variables improved.

In 1994, Bickell and colleagues published their results from a randomized trial of pre-hospital hypotensive resuscitation in patients sustaining penetrating torso injuries. (45) When compared to the standard group, those randomized to receive no fluids until arriving in the OR had less intra-operative blood loss, shorter length of stay and increased survival. As with our findings, these investigators noted similar field SBP but lower blood pressure on ED arrival. Eight years later, Dutton and colleagues conducted a randomized trial of hypotensive resuscitation beginning in the ED. (46) While there was no survival difference detected in this small study, the authors demonstrated that aiming for a SBP of 70 mmHg (versus >100 mmHg) was safe in patients arriving with evidence of hemorrhage. Morrison and colleagues have recently lent support to intra-operative hypotensive resuscitation. (47) These investigators randomized patients in hemorrhagic shock to target mean arterial pressures (MAP) of 50 or 65 mm Hg. In this pilot study, investigators from Ben Taub General hospital found that targeting resuscitation to a MAP of 50 mmHg results in reduced intra-operative RBC and plasma transfusions, less post-operative coagulopathy and higher 24-hour survival. The current study noted that SBP measurements on arrival to the ED, OR and ICU were lower in the DCR group. Similar to what are colleagues at Ben Taub noted, the DCR group had less blood transfusions, hypothermia, coagulopathy and acidosis and had improved survival.

While we feel that the current data supports the adoption of DCR, this study has several limitations. First, and most notable, this is not a randomized study. While we implemented the DCR concept in a prospective fashion through an educational initiative, such a pre-/post-intervention study design is not a substitute for a well-designed randomized trial. In addition, though the groups appear similar in all baseline comparisons (Table 1), differences among the groups not measured or captured in the current study may very well exist. As well, this was not a multi-center investigation but rather the results from a single institution. In addition, the indication or use of DCL was based solely on Trauma Surgeon judgment and nothing else. No protocols or guidelines were (or are) in place at our institution. However, all of our faculty are full-time trauma surgeons at a busy, level-1 ACS trauma center and all are board certified in Surgery and Surgical Critical Care. Finally, we focused our study on a select group of patents (those undergoing DCL). Therefore, DCR may not be suitable or helpful in those not requiring DCL. However, when we did look at those undergoing emergent laparotomy (but not DCL) we noted similar favorable findings in resuscitation end-points and avoidance of the lethal triad. Though these outcomes did not result in improved survival, no evidence of harm was noted in those treated with the DCR strategy.

CONCLUSION

Beginning in 2003, military clinicians started developing and implementing the concepts of DCR in the Iraq theater of operations. Subsequently, leaders in trauma, critical care, and emergency medicine, anesthesia, and transfusion medicine called on those caring for the injured patient to reconsider their approach to resuscitation. (12, 13) These editorials outlined the concept of Damage Control Resuscitation. (48, 49) While many civilian centers began to embrace the DCR concept, most only applied the principle of higher plasma and platelet ratios. Applying all three tenets, the current study identified a reduction in crystalloid and blood administration in patients managed with DCR. More importantly, the DCR strategy was associated with a 2.5 fold increased odds in 30-day survival.

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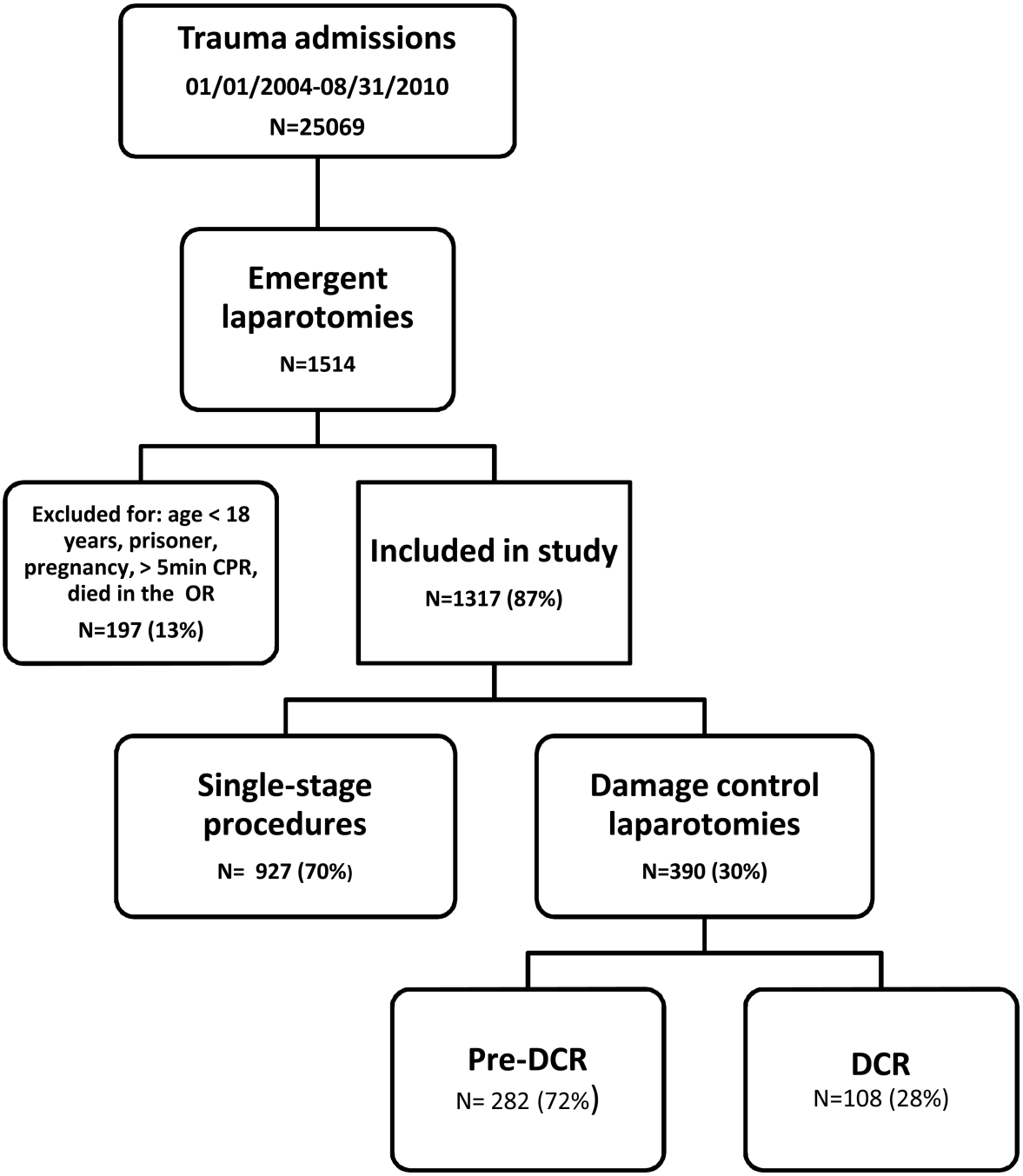

Figure 1.
Study design and patient selection

Table 1

Demographics and baseline comparison of the pre-DCR and DCR groups

	Pre-DCR (n=282)	DCR (n=108)	p-value
Median age, in years (IQR)	35 (25, 47)	34 (23, 47)	0.534
Male gender, %	78%	77%	0.746
Blunt mechanism of injury, %	65%	69%	0.395
Median head AIS (IQR)	0 (0, 3)	0 (0,3)	0.614
Median chest AIS (IQR)	3 (0, 4)	3 (0, 4)	0.672
Median abdomen AIS (IQR)	3 (2, 4)	3 (3, 4)	0.244
Median ISS (IQR)	27 (17, 38)	26 (16, 41)	0.979

DCR: damage control resuscitation; IQR: inter-quartile range, 25^{th} and 75^{th} percentiles; AIS: abbreviated injury scale; ISS: injury severity score

Table 2

Comparison of pre-hospital, emergency department and operating room vitals, labs, and fluid and transfusion practices

Prehospital variables			
	Pre-DCR (n=282)	**DCR (n=108)**	**p-value**
Median SBP, mmHg (IQR)	100 (81, 124)	99 (90, 118)	0.669
Median heart rate, bpm (IQR)	112 (97, 130)	103 (86, 120)	0.005
Median GCS (IQR)	14 (7, 15)	12 (3, 15)	0.017
Median fluid administration, mL (IQR)	1.0 (0.5, 1.6)	0.5 (0.3, 0.9)	<0.001
Emergency Department variables			
Median arrival temperature, F° (IQR)	97.2 (96.4, 98.0)	97.1 (96.2, 98.0)	0.812
Median arrival SBP, mmHg (IQR)	109 (83, 123)	95 (74, 123)	0.018
Median arrival heart rate, bpm (IQR)	110 (91, 130)	108 (90, 128)	0.306
Median arrival GCS (IQR)	14 (3, 15)	7 (3, 15)	0.029
Median arrival hemoglobin, g/dL (IQR)	12.2 (10.3, 13.7)	12.4 (11.1, 13.8)	0.514
Median arrival platelet count (IQR)	242 (199, 311)	222 (178, 289)	0.086
Median arrival INR value (IQR)	1.3 (1.1, 1.6)	1.4 (1.2, 1.6)	0.028
Median arrival pH value	7.22 (7.08, 7.31)	7.23 (7.12, 7.28)	0.717
Median arrival base value (IQR)	−11 (−14, −6)	−8 (−12, −4)	0.012
Median fluid administration, L (IQR)	2.0 (1.0, 3.0)	1.0 (0.2, 2.0)	<0.001
Median RBC transfusion, U (IQR)	2 (0, 4)	2 (0, 3)	0.771
Median plasma transfusion, U (IQR)	0 (0, 0)	0 (0, 1)	0.044
Median platelet transfusion, U (IQR)	0 (0, 0)	0 (0, 0)	0.106
Operating room variables			
Median arrival temperature, F° (IQR)	95.0 (93.0, 96.1)	98.6 (98.1, 99.0)	<0.001
Median arrival SBP, mmHg (IQR)	120 (100, 135)	108 (84, 130)	0.015
Median arrival heart rate, bpm (IQR)	107 (93, 122)	98 (87, 110)	0.002
Median arrival pH value (IQR)	7.20 (7.12, 7.30)	7.27 (7.18, 7.33)	<0.001
Median arrival base value (IQR)	−9 (−13, −6)	−6 (−9, −4)	<0.001
Median fluid administration, L (IQR)	4.0 (3.0, 6.4)	2.8 (1.8, 4.1)	<0.001
Median RBC transfusion, U (IQR)	9 (3, 16)	5 (2, 11)	0.001
Median plasma transfusion, U (IQR)	5 (2, 11)	4 (2, 10)	0.079
Median platelet transfusion, U (IQR)	0 (0, 12)	0 (0, 6)	0.024

DCR: damage control resuscitation; IQR: inter-quartile range, 25^{th} and 75^{th} percentiles; F°: Fahrenheit; SBP: systolic blood pressure; bpm: beats per minute; GCS: Glasgow coma scale; g/dL: grams per deciliter; INR: international normalized ratio; RBC: red blood cells; U: units

Table 3

Comparison of ICU, in-hospital, and outcome variables

ICU variables			
	Pre-DCR (n=282)	**DCR (n=108)**	**p-value**
Median arrival temperature, F° (IQR)	95.0 (93.0, 96.1)	98.3 (97.0, 98.8)	<0.001
Median arrival SBP, mmHg (IQR)	131 (110, 162)	120 (108, 132)	0.005
Median arrival heart rate, bpm (IQR)	103 (87, 119)	92 (78, 106)	<0.001
Median arrival hemoglobin, g/dL (IQR)	11.7 (10.1, 13.4)	10.3 (9.0, 11.5)	<0.001
Median arrival platelet count (IQR)	116 (75, 150)	279 (123, 515)	<0.001
Median arrival INR value (IQR)	1.3 (1.5, 1.7)	1.3 (1.2, 1.5)	0.004
Median arrival fibrinogen value (IQR)	166 (144, 212)	292 (196, 460)	<0.001
Median arrival pH value	7.31 (7.26, 7.37)	7.38 (7.33, 7.44)	<0.001
Median arrival base value (IQR)	−4 (−7, −2)	0 (−4, 4)	<0.001
Median arrival lactate value (IQR)	5.6 (4.4, 8.0)	1.8 (1.2, 3.9)	<0.001
In-hospital variables			
Median 24-hr fluid administration, L (IQR)	13.9 (9.4, 20.0)	5.0 (3.8, 8.9)	<0.001
Median 24-hr RBC transfusion, U (IQR)	13 (5, 22)	8 (3, 16)	0.001
Median 24-hr plasma transfusion, U (IQR)	11 (5, 20)	8 (2, 18)	0.029
Median 24-hr platelet transfusion, U (IQR)	6 (0, 12)	0 (0, 12)	0.005
Outcome variables			
Median length of stay, days (IQR)	22 (9, 42)	19 (11, 33)	0.601
Median ICU length of stay, days (IQR)	9 (3, 20)	7 (3, 16)	0.421
Median ventilator-free days (IQR)	17 (1, 25)	20 (9, 26)	0.134
Discharged with open abdomen %	13.8%	9.6%	0.298
Acute renal failure, %	15.6%	8.3%	0.060
Acute respiratory distress syndrome, %	4.6%	0.9%	0.080
Multi-organ failure	6.0%	2.0%	0.107
24-hr survival, %	88%	97%	0.007
30-day survival, %	76%	86%	0.030

ICU: intensive care unit; DCR: damage control resuscitation; IQR: inter-quartile range, 25^{th} and 75^{th} percentiles; F°: Fahrenheit; SBP: systolic blood pressure; bpm: beats per minute; g/dL: grams per deciliter; INR: international normalized ratio; hr: hour; L: liter; RBC: red blood cells; U: units; platelet unit= one apheresis platelet or six-pack random donor platelet

Table 4

Multivariate logistic regression model predicting 30-day survival

	Odds ratio	95% C.I.	p-value
Damage control resuscitation	2.48	1.10–5.58	0.028
ED arrival base value	1.10	1.02–1.20	0.011
ED arrival INR value	0.93	0.79–1.10	0.414
ED arrival SBP, mmHg	1.02	0.99–1.02	0.075
Age, years	0.98	0.96–1.01	0.192

95% C.I.: 95 percent confidence interval; ED: emergency department; INR: international normalized ratio; SBP: systolic blood pressure