Damage Control Resuscitation in Combination With Damage Control Laparotomy: A Survival Advantage

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Background: Damage control laparotomy (DCL) improves outcomes when used in patients with severe hemorrhage. Correction of coagulopathy with close ratio resuscitation while limiting crystalloid forms a new methodology known as damage control resuscitation (DCR). We hypothesize a survival advantage in DCL patients managed with DCR when compared with DCL patients managed with conventional resuscitation efforts (CRE).

Methods: This study is a 4-year retrospective study of all DCL patients who required ≥10 units of packed red blood cells (PRBC) during surgery. A 2-year period after institution of DCR (DCL and DCR) was compared with the preceding 2 years (DCL and CRE). Univariate analysis of continuous data was done with Student's *t* test followed by multiple logistic regression.

Results: One Hundred twenty-four and 72 patients were managed during the DCL and CRE and DCL and DCR time periods, respectively. Baseline patient characteristics of age, Injury Severity Score, % penetrating, blood pressure, hemoglobin, base deficit, and INR were similar between groups. There was no difference in quantity of intraoperative PRBC utilization between DCL and CRE and DCL and DCR study periods: 21.7 units versus 25.5 units ($p = 0.53$); however, when compared with DCL and CRE group, patients in the DCL and DCR group received less intraoperative crystalloids, 4.7 L versus 14.2 L ($p = 0.009$); more fresh frozen plasma (FFP), 18.2 versus 6.4 ($p = 0.002$); a closer FFP to PRBC ratio, 1 to 1.2 versus 1 to 4.2 ($p = 0.002$); platelets to PRBC ratio, 1:2.3 versus 1:5.9 (0.002); shorter mean trauma intensive care unit length of stay, 11 days versus 20 days ($p = 0.01$); and greater 30-day survival, 73.6% versus 54.8% ($p < 0.009$). The addition of DCR to DCL conveyed a survival benefit (odds ratio; 95% confidence interval: 0.19 (0.05–0.33), $p = 0.005$).

Conclusion: This is the first civilian study that analyses the impact of DCR in patients managed with DCL. During the DCL and DCR study period more PRBC, FFP, and platelets with less crystalloid solution was used intraoperatively. DCL and DCR were associated with a survival advantage and shorter trauma intensive care unit length of stay in patients with severe hemorrhage when compared with DCL and CRE.

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Damage control laparotomy (DCL) is a widely accepted practice used in the management of the injured trauma patient with evidence of severe tissue injury and tissue hypoperfusion.[1] DCL provides a survival advantage of 60% to 90% when compared with 58% survival in patients not managed with DCL.[2,3] Conventional resuscitation efforts (CRE) with aggressive crystalloid resuscitation and no predefined close ratio transfusion protocol fails to address the physiologic coagulopathic derangements present in the severely injured patient managed with DCL,[4] Figure 1.

Correction of coagulopathy, with close ratio resuscitation while limiting crystalloid, forms a new methodology known as damage control resuscitation (DCR). Previous studies have demonstrated an increased survival in patients managed with DCR.[5–9] Survival is improved by early correction of trauma-induced coagulopathy with close ratio intraoperative component blood products resuscitation.[10] Close ratio resuscitation is an independent predictor of death at 6 hours, at 24 hours, and at 30 days and conveys an improved 30-day survival among massively transfused civilian trauma patients. This aggressive approach to resuscitation has shown promising outcomes in both military and civilian groups.[5]

For DCL to be optimized, effective early hemostatic resuscitation of the exsanguinating patient should be intimately coupled with surgical control of life-threatening injury. We hypothesize that the addition of DCR to patients undergoing DCL will provide a survival advantage over the DCL patients managed with CRE.

METHODS

This study is a 4-year retrospective cohort study of all trauma patients requiring ≥10 units of packed red blood cells (PRBC) during DCL. Only patients who underwent DCL with intraoperative use ≥10 units of PRBC were included in the analysis. A 2-year period after institution of DCR (DCL and DCR) was compared with the preceding 2 years (DCL and CRE). Consistent with the three-staged approach defining the technique of DCL, a damage control patient was defined

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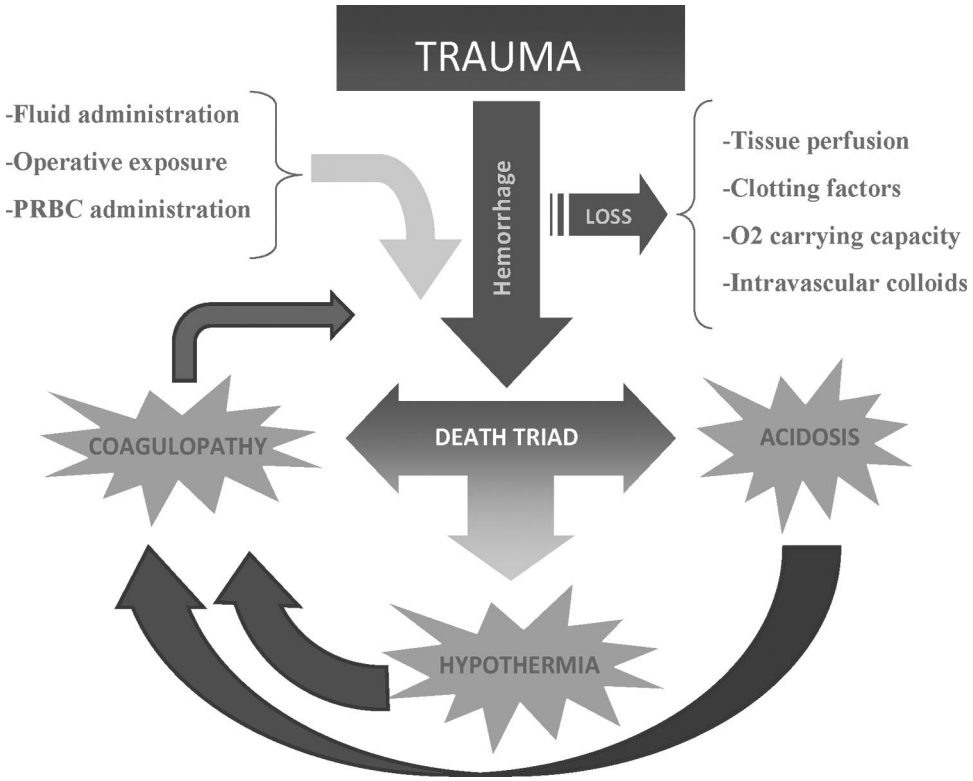

Figure 1. CRE of trauma patients with severe hemorrhage.

as a patient who was in extremis undergoing a truncated laparotomy (part I), followed by physiologic optimization in the intensive care unit (part II), and further definitive organ management at a later second operation (part III).[11] DCR involved the use of a close ratio massive transfusion protocol (MTP) with predetermined blood component transfusion ratios of 1:1 for fresh frozen plasma (FFP) to PRBC and 1:2 for platelets (PLT) to PRBC while minimizing the utilization of crystalloids, Figure 2. CRE involved the use of aggressive crystalloid resuscitation and no predefined close ratio transfusion protocol. CRE resuscitation efforts were used before institution of DCR.

Demographic and clinical data were retrieved from the trauma registry and charts. PRBC, FFP, and PLT transfusion data were retrieved from the blood bank.

Anatomic and physiologic variables analyzed included patient age, gender, mechanism of injury, initial systolic blood pressure (mm Hg) on presentation to operating room, emergency department international normalized ratio (INR), initial hemoglobin (g/dL), initial base deficit (mmol/L), Injury Severity Score (ISS), mean transfer time from emergency department to operating room measured in minutes, and mean operative time for DCL measured in minutes. After arrival to the trauma intensive care unit (TICU), initial systolic pressure, arterial blood pH, base deficit, temperature (°C), and INR were analyzed. Coagulopathy was defined as INR >1.2 seconds. Intraoperative amount of PRBC, FFP, PLT, and isotonic crystalloid solution administered was defined as the amount given from initial presentation to the operating room until the end of initial surgical intervention. Mean intraoperative FFP: PRBC and PLT: PRBC ratios were calculated and compared between the study periods. Outcomes measured between the groups included 30-day survival and TICU length of stay (LOS).

Survival was defined as alive at 30 days of hospital stay or discharge from the hospital before 30 days. Patients who were younger than 18 years, those who died in the emergency department, and those with nonsurvivable head injuries were excluded from the analysis. This institutional review board-approved protocol was conducted in an urban Level I Trauma Center to which all major traumas were brought by emergency medical services from January 2005 to December 2008.

Statistical Methods

Demographic characteristics, clinical parameters in the emergency department and the operating room, and prevalence of 30-day survival were calculated between the study periods. The statistical significance of differences in the means and prevalence estimates for participants were determined using least square means and maximum likelihood ratio for continuous and categorical variables, respectively. Multivariable log binomial regression models, adjusted for age, gender, ISS, and type of injury, were used to analyze the association of overall mortality between the study periods. In secondary analysis, using a linear regression model after adjusting for same variables, additional TICU LOS was determined. All statistical analyses were performed using SAS version 9.1.3 (SAS Institute, Cary, NC).

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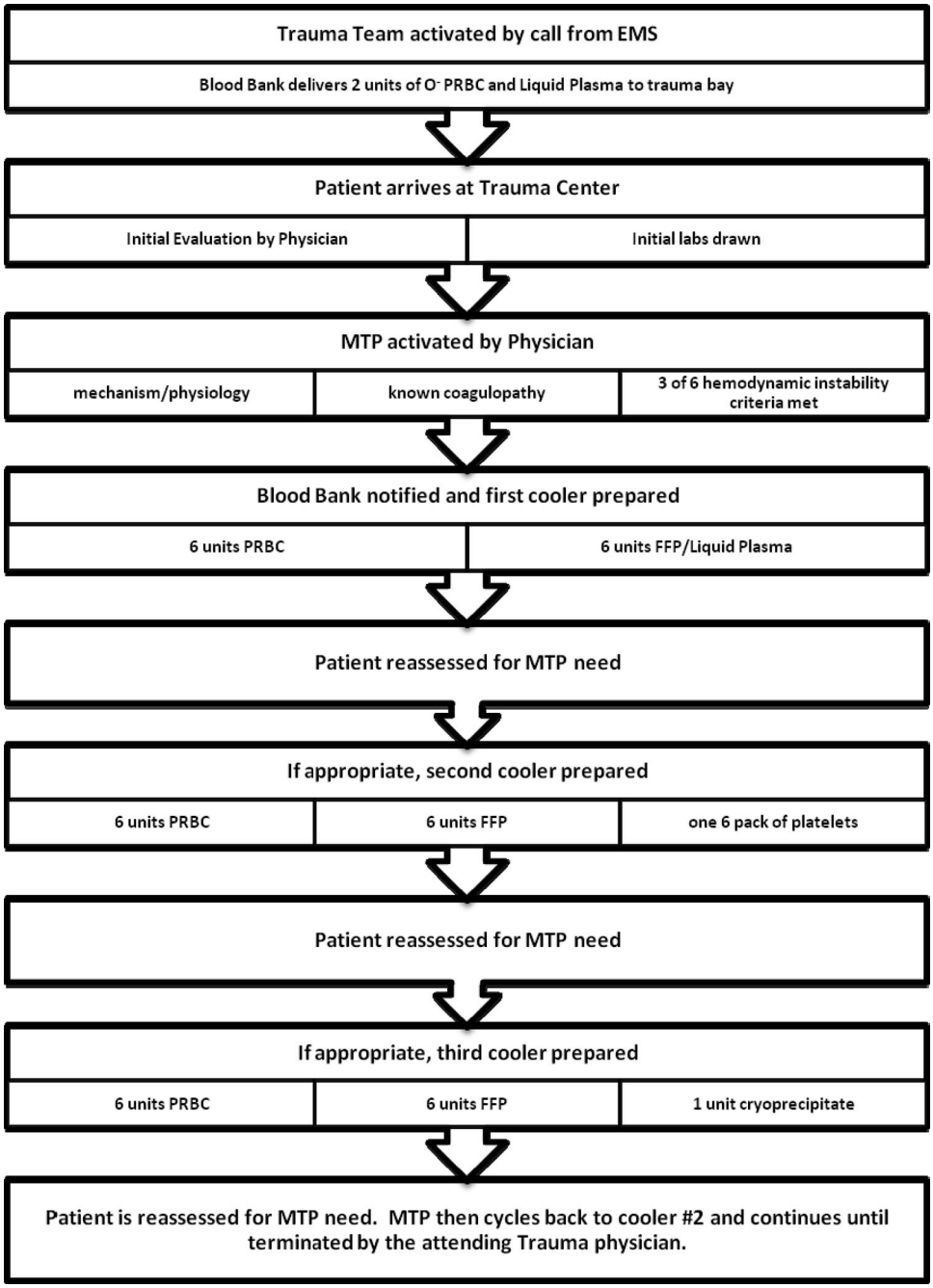

Figure 2. MTP used. Hemodynamic instability criteria include heart rate >110, systolic blood pressure <100 mm Hg, hemoglobin <9 g/dL, pH <7.25, INR >1.5, and temperature <35°C.

RESULTS

During the four-year study period, a total of 196 patients with blunt and penetrating injuries underwent DCL and received ≥10 units of PRBC in the operating room. A total of 124 (63.2%) patients were managed during the DCL and CRE period and 72 (36.8%) during DCL and DCR. The overall mortality of the study group was 75 (38.2%). When comparing patient demographics between DCL and CRE to DCL and DCR, there was no significance difference with regard to patients mean age in years, 28 versus 30 ($p = 0.19$); male gender, 83% versus 88% ($p = 0.59$); penetrating mechanism of injury, 67% versus 77% ($p = 0.86$); mean ISS, 23 versus 25 ($p = 0.79$); mean initial base deficit, −8.7 versus −7.3 mmol/L ($p = 0.52$); mean initial INR, 1.4 seconds versus 1.5 seconds ($p = 0.42$); and mean initial hemoglobin, 9.2 g/dL versus 8.8 g/dL ($p = 0.96$), Table 1.

There was a trend toward decreased transfer time to the operating room between study groups but of no statistical

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TABLE 1. Patient Demographics for DCL Patients Between Study Periods

	DCL and CRE (n = 124)	DCL and DCR (n = 72)	*p*
Age (yr), mean (SD)	28 (12)	30 (14)	0.19
Male (%)	83	88	0.59
Penetrating injury (%)	67	77	0.86
ISS, mean (SD)	23 (11)	25 (12)	0.79
Initial base deficit (mmol/L), mean (SD)	−8.7 (1.9)	−7.3 (1.7)	0.52
INR, mean (SD)	1.4 (0.5)	1.5 (0.7)	0.42
Initial hemoglobin (d/dL), mean (SD)	9.2 (2.1)	8.8 (1.8)	0.96

TABLE 2. Outcomes Between Resuscitation Strategies in DCL Patients

Outcomes	DCL and CRE (n = 124)	DCL and DCR (n = 72)	*p*
Mean transit time to OR (min), mean (SD)	9.3 (4.2)	7.8 (5.2)	0.26
Emergency department crystalloids (L), mean (SD)	4.7 (2.1)	1.1 (1.2)	0.0001
Intraoperative crystalloids (L), mean (SD)	14.2 (5.3)	4.7 (2.5)	0.009
Intraoperative PRBC (units), mean (SD)	21.7 (7.2)	25.5 (6.9)	0.53
Intraoperative FFP (units), mean (SD)	6.4 (3.9)	18.2 (7.1)	0.002
Intraoperative PLT (units), mean (SD)	6.1 (3.2)	13.8 (6.2)	0.01
Intraoperative FFP:PRBC ratios	1:4.2	1:1.2	0.002
Intraoperative PLT:PRBC ratios	1:5.9	1:2.3	0.002

significance: DCL and CRE 9.3 minutes ± 4.2 minutes versus DCL and DCR 7.8 minutes ± 5.2 minutes, $p = 0.26$. After arrival to the operating room, there was no difference in initial systolic pressure between groups: 88 mm Hg versus 92 mm Hg ($p = 0.06$) for DCL and CRE and DCL and DCR, respectively. There was no difference in quantity of intraoperative PRBC utilization between DCL and CRE and DCL and DCR study periods: 21.7 units versus 25.5 units ($p = 0.53$); however, when compared with DCL and CRE group in the DCL and DCR group, patients received less intraoperative crystalloids, 4.7 L versus 14.2 L ($p = 0.009$); more FFP, 18.2 versus 6.4 ($p = 0.002$); a closer FFP to PRBC ratio, 1 to 1.2 versus 1 to 4.2 ($p = 0.002$); and PLT to PRBC ratio, 1:2.3 versus 1:5.9 (0.002), Table 2.

Once transferred to the operating room, there was no difference in mean operative time between groups: 142 minutes ± 32 minutes versus 131 minutes ± 41 minutes ($p = 0.23$) and initial TICU systolic pressure: 127 mm Hg versus 130 mm Hg ($p = 0.31$) for DCL and CRE and DCL and DCR, respectively. On arrival to TICU, DCL patients managed with DCR had lower base deficit, −3.4 versus −7.9 ($p < 0.002$); higher pH, 7.31 versus 7.22 ($p < 0.03$); less hypothermia, 35.8°C versus 33.3°C ($p < 0.002$); and lower INR, 1.12 versus 1.37 ($p < 0.03$), when compared with DCL patients managed with CRE, Table 3.

TABLE 3. Mean OR Time and on Arrival TICU Physiologic and Laboratory Comparison Between Study Periods

	DCL and CRE	DCL and DCR	*p*
Mean or time (min), mean (SD)	142 (32)	131 (41)	0.23
TICU systolic (mm Hg), mean (SD)	127 (27)	130 (31)	0.31
TICU base deficit (mmol/L), mean (SD)	−7.9 (3.9)	−3.4 (1.7)	0.002
TICU pH, mean (SD)	7.22 (0.05)	7.31 (0.02)	0.03
TICU temperature (°C), mean (SD)	33.3 (0.5)	35.8 (0.6)	0.002
TICU INR, mean (SD)	1.37 (0.4)	1.12 (0.2)	0.03

TABLE 4. Mean TICU LOS and Overall 30-Day Survival for DCL Patients During Study Periods

Outcomes	DCL and CRE	DCL and DCR	*p*
Mean TICU LOS (d), mean (SD)	20 (24)	11 (9)	0.01
30-d Survival	54.8%	73.6%	0.009

TABLE 5. Multiple Logistic Regression With Adjusted Odds Ratio for Survival in DCL Patients

Variables	*p*	OR (95% CI)
DCR	0.005	0.19 (0.05–0.33)
Age (yr)	0.74	1.00 (0.97–1.02)
Gender	0.30	2.00 (0.54–7.49)
ISS	0.60	0.99 (0.96–1.02)
Penetrating vs. blunt trauma	0.68	0.87 (0.43–1.73)

Of significance, from a linear regression model, there was increased mean TICU LOS: 20 versus 11 ($p < 0.01$) and lower 30-day survival: 54.8% versus 73.6% ($p < 0.009$) in the DCL and CRE group, when compared with DCL and DCR group, Table 4. After adjustment for age, gender, ISS, and type of injury, DCR conveyed a survival benefit to DCL patients (odds ratio; 95% confidence interval: 0.19 (0.05–0.33), $p = 0.005$), Table 5.

DISCUSSION

The premise of DCL is that the metabolic derangement of ongoing bleeding supersedes the need for definitive operation. As such, the main thrust of DCL is the rapid surgical control of bleeding. DCL has led to better outcomes than expected in these grievously injured patients. One study examining the evolution of damage control techniques and outcomes >7 years noted that patients who received DCL for penetrating abdominal trauma at the end of that time period boasted higher survival rates, decreased incidence of intraop-

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TABLE 6. Comparison of DCL Patient Outcomes From Initial Damage Control Study to our Current Study With the Addition of DCR

	Rotondo et al.,[13] (DCL)	Charity Hospital, 2009 (DCL and DCR)
Number (n)	24	72
Age (yr)	30.6	30
Male (%)	95	88
ISS	24.2	25
Penetrating (%)	100	77
Temperature (°C)	32.9	35.8
pH	7.20	7.31
PRBC (units)	22.7	25.5
Survival (%)	58	73.6

erative hypothermia, and more frequent definitive colon repair.[2] However, outcomes of DCL patients with regard to the choice of resuscitation strategy still needs further analysis.

These surgeries tend to have a high complication rate, because survival is given a higher priority than morbidity, in these patients who are in poor physiologic condition.[12] Our research sought to further expand on this concept by examining the outcomes of DCL patients managed with DCR versus CRE. Although the number of PRBC used intraoperatively was similar between groups, the addition of DCR to DCL patients conveyed a significant survival benefit. DCR patients were aggressively resuscitated with blood component therapy with a close ratio of FFP:PRBC and minimal use of intraoperative crystalloid solution in contrast to the CRE time period, which included a FFP:PRBC ratio of 1:4 and significantly more crystalloid solution intraoperatively. These CRE patients arrived to the TICU volume overloaded, with overaggressive, unnecessary crystalloid resuscitation, and with signs of tissue hypoperfusion, hypothermia, and coagulopathy, markers of increased mortality after arrival to TICU.[6]

A direct comparison of our patient population with previous report on damage control from Rotondo et al. was done, Table 6. Studies are very similar with regard to patient demographics. In our study, the addition of DCR to DCL improved the overall physiologic status of these patients after arrival to TICU, with improved pH, less hypothermia, and ultimately an overall improved survival. The overall survival rate improved from 58% in Rotondo et al.[13] initial study to 73.6% in our study. The most likely explanation for this survival benefit is the combination of (1) new techniques of damage control and (2) early correction of physiologic and coagulopathy derangements with institution of DCR with less crystalloid utilization.

In 1985, a retrospective review of 68 massively transfused patients by Hewson et al. found that coagulopathy was common after crystalloid administration and that PTT correlated with the volume of crystalloids given. He recommended that FFP and PRBC be given at a ratio of 1:1.[14] For ~2 decades, this recommendation was largely ignored. However, in 2002, while describing the effect of fluids on coagulation, Hirshberg et al.[15] concluded that to avoid coagulopathy PRBC and FFP must be given in a 3:2 ratio.

The use of a 1:1 FFP to PRBC ratio is based largely on the evidence acquired during the military's recent experience with the management of combat casualties. Borgman et al. compared mortality rates associated with varying ratios of FFP to PRBC in the management of trauma seen in Iraq. They found that patients receiving a "high" ratio of FFP to PRBC (1:1.4) had the lowest overall mortality rates and hemorrhage mortality rates, concluding that high FFP to PRBC ratio is independently associated with improved survival to hospital discharge, primarily by decreasing death from hemorrhage.[8] Similar results were found at our institution in a civilian trauma center when DCR transfusion ratio outcomes were analyzed.[7,10]

DCR has been shown to help manage the coagulopathy of trauma through the early and aggressive administration of blood products to the severely injured trauma victim, while minimizing crystalloid utilization. This does run contrary to dictum, because intravenous fluids have remained a mainstay of resuscitative therapy since the development of advanced life support care in the 1960s.[16] Its place as the mainstay of initial therapy for the patient in hemorrhagic shock is predicated on the early work of Carrico et al.[17] and Shires and coworkers,[18] which revolves on observations of fluid and salt shifts in the intracellular and extracellular spaces after hemorrhagic shock. Although isotonic and hypotonic saline solutions still have their place in the armamentarium of the trauma patient, the view of these as a panacea for hemodynamic instability as part of DCR should be reexamined.

Minimal use of crystalloids is essential to the concept of DCR.[9] One of the prime mechanisms by which over utilization of crystalloids is counterproductive to DCR efforts is the exacerbation of the components of the "death triad" of acidosis, hypothermia, and coagulopathy.[19–21] Unwarmed crystalloids can simultaneously cause a dilutional coagulopathy and be a major cause for hypothermia, whereas an inadequately corrected shock state can result in persistent, profound acidosis. In addition, CRE can also cause imbalances at the cellular level, causing cellular swelling with resultant dilution of intracellular proteins and dysfunction of protein kinases, ultimately leading to decreased function of many cell types, including hepatocytes, pancreatic islet cells, and cardiac myocytes. As a result of the pathologic changes associated with trauma, capillary permeability increases, causing a loss of osmotic pressure and a loss of fluid to the interstitial and intracellular space.[22] This fluid shift is magnified by conventional fluid resuscitation protocols and can have profound systemic complications that prolong recovery and increase TICU LOS.[12] Among DCL and CRE patients, extended lengths of TICU stay were likely as a result of resuscitation-associated complications including cardiac dysfunction, decreased cardiac output, pulmonary complications, and abdominal compartment syndrome.[16]

Although it has also been suggested that the survival benefit provided by DCR in the massively transfused patient could perhaps be a result of survival bias,[23] the selection criteria of patients for whom an MTP would be beneficial must be specific and should not include subjects for whom resuscitation is inconsequential. Certainly, resuscitation of any kind will not demonstrate a survival

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advantage in the case of catastrophic aortic or brain injury. The study of DCR as it relates to clearly defined DCL patients, those with a baseline mortality rate, dismisses the possibility of survival bias as a reasonable explanation for observed survival outcomes.

CONCLUSION

This is the first civilian study that analyses the survival advantage of DCR in patients managed with DCL. In the civilian trauma setting, DCL is best applied to the group of severely injured, exsanguinating patients who may have multiple physiologic derangements. DCL patients are often managed with DCR as a means to address coagulopathy intraoperatively. Previous literature has failed to elucidate the extent to which DCR independently influences survival outcomes among DCL patients. This study finds evidence that DCR in 1:1 FFP to PRBC ratio in combination with less crystalloid utilization is an independent factor for DCL patient survival.

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DISCUSSION

Dr. Frederick A. Moore (Houston, Texas): In this retrospective historic control study, Doctor Duschesne and colleagues from New Orleans show a dramatic reduction in mortality in damage control laparotomy patients after they instituted a massive transfusion (MT) protocol that emphasize early one-to-one ratio FFP to PRBC resuscitation as standard of care. Now, the million dollar question is what caused this reduced mortality. In the introduction the authors state, and I quote, "Correction of coagulopathy with close ratio resuscitation while limiting crystalloids forms a new form of methodology known as damage control resuscitation." They then cite several references, two of which I coauthored. Now if you go back and review these references you will find that decreasing crystalloids really was not emphasized. The decreased crystalloids is a byproduct of having an effective MT protocol that can be started early in the emergency department. Now, you might think I'm quibbling but it is quite possible that the decreased mortality is due to decreased crystalloids. A mean of 14 liters crystalloids in the OR in the Pre-MT cohort is impressively large and probably not standard of care in most Trauma Centers. If you go back to the seminal work of Carl Moyer in the mid-1060s studying optimal resuscitation using a modified Wigger's prep, the best survival was obtained when the ratio of crystalloids to blood was 2.8 to 1. I believe this is the origin of the so-called three-to-one rule. However, Gerald Moss and others subsequently showed that with increasingly severe shock insults this optimal survival ratio of crystalloids to blood increases to eight-to-one. This is consistent with what we learned in the late 1990s studying ICU shock resuscitation in blunt trauma patients undergoing damage control surgery at the University of Texas (UT) Houston. Aggressive early crystalloid resuscitation allows them to survive previously lethal insults. However, the survivors develop problematic tissue edema that is manifested by acute lung injury (ALI) and the abdominal compartment syndrome (ACS) which leads to multiple organ failure (MOF) and death. We referred to this as the "vicious salt water cycle" and showed that less aggressive crystalloid resuscitation deceased ACS, MOF, and late

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deaths. Now, the alternative explanation for the reduced mortality (which has gained widespread popularity despite limited prospective data) is early one-to-one ratio FFP to PRBC resuscitation. Ernest Gonzalez at UT Houston and Bryan Cotton at Vanderbilt have both reported similar retrospective historic control studies showing reduced mortality after MT protocols were instituted that delivered early one-to-one resuscitation in the ED. However, two database analyses from Denver, one by Jeff Kashuk and the other by Jeff Johnson, indicate that aggressive FFP may reduce mortality but it comes at a cost of more ALI and MOF. They suggest a less aggressive ratio of one-to-two might optimize the risk versus benefits of damage control resuscitation. So, Juan, I have three questions for you.

Number 1. So what's the answer, no waffle, is it less crystalloids or more FFP? Could comment on platelets, since early 1:1:1 units of FFP to Platelets to PRBCs is now being advocated despite very limited clinical data? Also when and how did your patients die?

Number 2. What was the incidence of ALI, ACS and MOF? These are important pieces of the puzzles and should be reported in all outcome studies related to traumatic shock resuscitation.

Number 3. John Holcomb is planning a multi-institutional prospective randomized controlled trial testing optimal ratios. Are you going to be able to ethically participate? Will you randomize half of your patients to receive low ratio FFP to PRBC resuscitation?

I'd like to congratulate the New Orleans group on presenting yet another provocative study related to damage control resuscitation and to thank the association for the privilege of the floor.

Dr. Juan C. Duchesne (New Orleans, Louisiana): Thank you, Doctor Moore, for the provocative questions. I will go ahead and start with the first one.

Mortality, what really impacts mortality is the early use of FFP PRBC starting in the emergency department or is the combination of a good massive transfusion protocol that will lessen the use of crystalloids.

And I think I would go in favor of Number 2. I think if you have a very effective massive transfusion protocol running in your institution you don't have the time to actually hook up a bag of crystalloids.

Most of these patients are basically being aggressively resuscitated with FFP, PRBC, plasma, platelets and very rarely we see that they just have crystalloids running solely. So I believe it's a combination of an effective massive transfusion protocol that will eventually decrease the amount of crystalloid solutions.

Regarding platelets, we have our own bias on platelets. In our institution we don't have a one-to-one for platelets. We do a one-to-two.

And within the first 135 minutes we have data that actually doesn't impact survival. But within six hours, like Doctor Schreiber, Martin Schreiber described in a paper this year, there is definitely an impact in survival.

When and how they died. During the DCR era most of our patients survived the OR and arrived to the ICU where they, the ones that died, died there. During the CRE, the opposite.

Regarding our acute lung injury, abdominal compartment syndrome, multiple organ failure, although I don't have that specific data, I can guarantee you that this group of patients during the DCR, they fared better. They get out of the unit faster and they were sent home faster.

And the provocative question, multi-center trial by Doctor Holcomb, I think this is a very important question, where do we go from here. I think there is a lot of data but most of it retrospective.

We need to move forward and do a prospective trial but how? How we are going to convince people we are going to give you one-to-one and you are going to get a one-to-four? I'm sorry, you die. So it's really hard. And it's going to be very interesting to figure this out. I don't have the right answer right now.

Dr. Zsolt Balogh (Newcastle, Australia): The first question is, changing from crystalloid-based resuscitation to damage control resuscitation is a major change.

It doesn't happen overnight and you showed that two years before or two years after. How did you achieve that or would you think that it would be good to leave a transitional period between the two timeframes?

My second question is: Did you open abdomen strategy more liberally during the damage control resuscitation period than the previous one? Could that affect the observed better outcomes? Congratulations, again.

Dr. Reuven Rabinovici (Boston, Massachusetts): Another major factor that affects outcome from laparotomy or damage control laparotomy is time to operation. I wonder if you have any information that you can share with us regarding this parameter.

Dr. Lewis Kaplan (New Haven, Connecticut): I know I'm a little short. I'm below your horizon. Juan, you identified an improvement in Ph in those that had damage control resuscitation.

Was that from abrogation of hyperchloremic metabolic acidosis from a lack of crystalloid resuscitation? Was it from an improvement in clearance of lactate as a marker of abrogation of hypoperfusion or both?

Dr. Juan C. Duchesne (New Orleans, Louisiana): Regarding the first two questions from Doctor Zsolt, we, education, it takes a lot of downtime in the OR explaining to the anesthesiologist how we do this.

And while you have basically a patient that is exsanguinated, if you don't have good communication with them they can do whatever they want. And we know that.

So you need to establish that excellent communication. Get them involved in your trauma peer review committee so they will understand what you have running.

Regarding – because of time issues, regarding the time in the OR for the second question, most of our patients underwent damage control laparotomy within 120 minutes-130 minutes, so there was no big difference during study periods.

And regarding Kaplan question, Ph, how we fixed that, and I believe it's a combination of less crystalloids and more effective oxygen carrying capacity with blood products.

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