—Original—

A New Look at Criteria for Damage Control Surgery

Hisashi Matsumoto[1,2], Kunihiro Mashiko[1,2], Yuichiro Sakamoto[1,2], Noriyoshi Kutsukata[1,2], Yoshiaki Hara[1,2] and Hiroyuki Yokota[1]

[1]Department of Emergency and Critical Care Medicine, Graduate School of Medicine, Nippon Medical School
[2]Shock and Trauma Center, Nippon Medical School Chiba Hokusoh Hospital

Abstract

Background: Several reports have validated the criteria for damage control surgery (DCS). However, although metabolic acidosis and body temperature can be measured quickly, tests for predicting the severity of coagulopathy require special laboratory equipment and take 15 to 30 minutes. Such delays could be life-threatening for patients requiring DCS. The aim of this study was to establish simplified and practical criteria to enable rapid decision-making regarding the need for DCS.

Methods: Thirty-four consecutive patients with unstable hemodynamics after initial fluid resuscitation who had undergone DCS for severe abdominal or pelvic injuries were retrospectively analyzed. The patients' characteristics, clinical courses, laboratory data, and outcomes were reviewed using the data contained in their medical records.

Results: The overall survival rate was 55.9% (survivors group: n=19; nonsurvivors group: n=15), which was similar to the calculated mean probability of survival (Ps=0.5671). At the start of surgery, the systolic blood pressure (SBP) was less than 90 mm Hg in all cases in which surgery failed, and the mean SBP in the nonsurvivors group (69.6 ± 14.8 mm Hg) was significantly lower than that in the survivors group (93.2 ± 22.9 mm Hg, p=0.006). Except in two cases, the value of the base excess in the nonsurvivors group was less than −7.5 mmol/L, and the mean base excess (−11.5 ± 5.3 mmol/L) in the nonsurvivors group was significantly less than that in the survivors group (−5.5 ± 4.9 mmol/L, p=0.008) at the start of surgery. The core temperature at the start of surgery was less than 35.5℃ in all cases in the nonsurvivors group. On the basis of these results, three indicators (SBP less than 90 mm Hg, base excess less than −7.5 mmol/L, and core temperature less than 35.5℃ at the start of surgery) were identified. The success rate of DCS in patients who possessed all three indicators (28.6%) was significantly lower than that in patients who did not possess all three indicators (75.0%; p= 0.014).

Conclusion: Our results indicate that surgeons should decide to perform DCS when only one or two criteria defined in this study are met and should not wait for all three criteria. Although our proposed criteria are not strict and may broaden the indications for DCS, leading to an increase in the number of DCS procedures, saving the lives of patients who have sustained severe torso trauma must be the priority; 'over-triage' may be acceptable in situations where an appropriate decision-making protocol has been followed.
(J Nippon Med Sch 2010; 77: 13–20)

Key words: trauma, damage control surgery, coagulopathy, hypothermia, acidosis

Correspondence to Hisashi Matsumoto, MD, PhD, Shock and Trauma Center, Nippon Medical School Chiba Hokusoh Hospital, 1715 Kamakari, Inba-mura, Inba-gun, Chiba 270–1964, Japan
E-mail: hmatsu@nms.ac.jp
Journal Website (http://www.nms.ac.jp/jnms/)

Introduction

The conventional indications for damage control surgery (DCS) in patients with exsanguinous torso trauma are the well-known "deadly triad" of metabolic acidosis, hypothermia, and coagulopathy, and several reports have shown the effectiveness of DCS[1–7]. In an emergency, the level of metabolic acidosis can be quickly determined by means of blood gas analysis, and the patient's body temperature can be determined with a simple measurement. However, coagulation tests, such as determinations of the prothrombin time (PT) and the activated partial thromboplastin time (APTT), which are needed to determine the severity of coagulopathy, require special laboratory equipment and 15 to 30 minutes. Furthermore, PT and APTT are determined at 37℃, not at the patient's actual body temperature, and might not accurately reflect the coagulation abnormality in patients with hypothermia.

Therefore, surgeons may hesitate to perform DCS when information about the severity of coagulopathy is unavailable, even if metabolic acidosis and hypothermia are present. Such delays could be fatal if the patient does indeed require DCS. Shapiro et al.[8] have reported that the results of thromboelastography can simplify the diagnosis of coagulopathy and might serve as an early predictor of the need for transfusion in patients who have sustained blunt-force injuries. However, simpler criteria are needed to avoid delays in performing DCS. The aim of the present study was to establish simplified and practical criteria for deciding whether to perform DCS.

Materials and Methods

Clinical Series

From April 2000 through March 2008, 34 consecutive patients underwent DCS for the treatment of severe abdominal organ or pelvic injuries at the Shock and Trauma Center of Chiba Hokusoh Hospital, Nippon Medical School, which corresponds to a Level 1 trauma center in the United States. Patients who had sustained severe head injuries or had died in the emergency department (ED) were excluded. Patients judged as "nonresponders" or "transient responders" according to the criteria described below were considered to be candidates for DCS, although the decision to perform DCS was ultimately made by the surgeon.

On the basis of the Advanced Trauma Life Support course of the American College of Surgeons[9], the Japan Advanced Trauma Evaluation and Care program has developed criteria that classify patients with hemorrhagic shock into three types according to the response to initial fluid resuscitation (2,000 mL of warm Ringer's solution in adults): "responders," who respond well to an initial infusion and do not require "additional" crystalloid infusion or blood transfusion to maintain a systolic blood pressure (SBP) greater than 90 mm Hg; "transient responders," who respond well initially but require additional fluids or blood to maintain a SBP greater than 90 mm Hg; and "nonresponders," who have an SBP that remains less than 90 mm Hg after initial fluid resuscitation.

Damage control techniques, including suturing, selective vascular repair and ligation, and gauze and towel packing at the injured sites for uncontrolled hemorrhage, were performed with a direct approach in cases of solid organ, mesenteric, or retroperitoneal hemorrhage. Also, hollow viscus injuries were closed using a single-layer, full-thickness closure. Towel clip closure was usually selected for temporary abdominal wall closure, but some patients underwent silo closure to reduce the risk of abdominal compartment syndrome.

A high-flow blood fluid warming device (Level 1 System1000; Smiths Medical, London, UK) was used for fluid resuscitation and transfusion to avoid hypothermia in the ED and the operating room (OR). All 34 patients were transported to the intensive care unit and were rewarmed with warm blankets. Blood components, including fresh frozen plasma and platelets, were administered appropriately to correct the coagulopathies and normalize the values of hemoglobin, platelets, and PT (international normalized ratio). Reoperation was planned once the hemodynamics, acid-base balance, body temperature,

and coagulation had normalized. The outcomes of DCS were survival or death of the patients.

The patients' clinical characteristics, including age, sex, injuries, injury severity score (ISS), probability of survival (Ps), operating procedures, laboratory data, and outcome, were reviewed by using medical records (**Table 1**). The patients were 25 men and 9 women, ranging in age from 11 to 90 years (mean ± SD, 51.9 ± 20.5 years). The mechanism of injury was blunt trauma in 33 cases and stabbing in 1 case. The mean ISS was 35.6 ± 13.5 (range, 9 to 66), and the mean Ps calculated using the Trauma Score-Injury Severity Score method[10] was 0.5671.

Sixteen patients underwent ED laparotomy to control intraperitoneal hemorrhage. The remaining 18 patients were transported to the OR. The primary DCS procedure for controlling hemorrhage was hepatorrhaphy in 15 cases, pancreatorrhaphy in 2 cases, splenorrhaphy in 2 cases, splenectomy in 2 cases, aortorrhaphy in 1 case, and suture repair of the inferior vena cava in 2 cases. An ED thoracotomy with aortic cross-clamping was performed in 9 of these cases, cardiorrhaphy for cardiac injury was performed in 2 cases, suturing of lung lacerations was performed in 2 cases, and pulmonary hilar clamping was performed in 1 case. Planned reoperations were performed 1.5 ± 0.7 days after the initial operation. The primary procedure utilized in the planned reoperations was depacking; other procedures included hepatorrhaphy in 1 case, non-anatomical hepatectomy in 1 case, pancreatoduodenectomy in 4 cases, distal pancreatectomy in one case, intestinal repair in 3 cases, nephrectomy in 2 cases, and colostomy in 1 case.

Physiologic Variables

Three variables (SBP, base excess, and core temperature) were examined at the time of ED arrival and at the start of thoracotomy or laparotomy; these variables were investigated as predictors of patient outcome.

Characteristics were compared between survivors and nonsurvivors by means of the chi-square test or the Wilcoxon signed-rank test, as appropriate. Statistical significance was inferred when the p value was less than 0.05. Values are expressed as the mean ± standard deviation.

Results

The overall survival rate was 55.9% (survivors group: n=19; nonsurvivors group: n=15), which was similar to the calculated mean Ps. Patient characteristics, including age, sex, ISS, Ps, and mechanism of injury, were not related to outcome (**Table 2**). The survival rate was 50.0% among the "nonresponders" (9 of 18 patients) and 62.5% among the "transient responders" (10 of 16 patients); no significant relationship between the response to initial fluid resuscitation and the results of DCS was observed.

In the survivors group, the mean SBPs at the time of ED arrival (89.9 ± 21.3 mm Hg) and at the start of the surgical procedure (93.2 ± 22.9 mm Hg) were similar. In contrast, in the nonsurvivors group the mean SBP at the start of the surgical procedure (69.6 ± 14.8 mm Hg) was slightly but not significantly lower than that at the time of ED arrival (84.6 ± 21.8 mm Hg). At the start of the surgical procedure, the SBPs in all cases in the nonsurvivors group was less than 90 mm Hg, and the mean SBP in the nonsurvivors group was significantly lower than that in the survivors group (p=0.006). In the survivors group, the levels of base excess were not significantly different at the time of ED arrival (−7.2 ± 5.1 mmol/L) and at the start of surgery (−5.5 ± 4.9 mmol/L). In the nonsurvivors group, however, the levels of base excess were greater at the start of surgery (−11.5 ± 5.3 mmol/L) than at the time of ED arrival (−7.6 ± 6.4 mmol/L, p=0.013). At the start of surgery, the base excess in the nonsurvivors group (with the exception of two cases) was less than −7.5 mmol/L; this value was significantly lower than that in the survivors group (p=0.008). The core temperature at the start of surgery was less than 35.5℃ in all cases in the nonsurvivors group (**Fig. 1**).

On the basis of these results, three indicators (SBP less than 90 mm Hg, base excess less than −7.5 mmol/L, and core temperature less than 35.5℃ at the start of surgery) were identified. The survival

Table 1 Demographics and Clinical Characteristics of 34 Patients Who Underwent DCS

Patient no.	Age	Gender	Injuries	ISS	Ps	DCS	Planned re-operation	Outcome
1	34	M	Pancreas, L-Kidney, L-rib fx	29	0.9116	packing	DP, Lt-nephrectomy	Alive
2	11	M	Liver, T-SAH, DAI	18	0.8692	EDT/EDL, hepatorrhaphy, packing	—	Dead
3	45	M	Pancreas, SMV	16	0.9963	pancreatorrhaphy, packing	depacking	Alive
4	40	M	Pancreas, Cerebral contusion	43	0.3121	packing	PD	Alive
5	31	F	Liver	25	0.9684	hepatorrhaphy, packing	depacking	Alive
6	57	M	Liver, Heart (RV, LA)	42	0.0864	EDT/EDL, cardiorrhaphy, hepatorrhaphy, packing	depacking	Alive
7	22	M	Rt-Kidney, Spleen	45	0.9416	splenectomy, packing	Rt-nephrectomy	Alive
8	80	F	Liver, Flail chest, Pulmonary contusion	50	0.2147	EDT/EDL, packing	—	Dead
9	50	F	Pancreas, Liver, Spleen, L-pneumothorax, R-open patella fx	38	0.9650	splenorrhaphy, packing	PD	Alive
10	67	M	Liver, Flail chest, Pulmonary contusion, Bil. femur fx, R-open tibia fx	41	0.1998	hepatorrhaphy, packing	depacking	Alive
11	63	M	Liver, Mesenterium, Small intestine	9	0.9665	hepatorrhaphy, packing	depacking	Alive
12	33	M	Pancreas, Spleen	25	0.9827	splenorrhaphy, packing	PD	Alive
13	64	M	Liver, Pancreas, PV, Skull base fx, R-forearm fx, R-acetabular fx, R-tibia fx	38	0.0538	EDL, hepatorrhaphy, packing	—	Dead
14	64	M	Liver, Spleen, Pulmonary laceration	41	0.2875	EDT/EDL, hepatorrhaphy, packing, suturing for pulmonary laceration,	—	Dead
15	27	M	Liver, Spleen, IVC, Pulmonary contusion	50	0.8912	EDL, hepatorrhaphy, packing	—	Dead
16	55	M	Pancreas	25	0.9177	pancreatorrhaphy, packing	PD	Alive
17	90	M	Liver, Flail chest, Pulmonary laceration, R-open tibia fx	43	0.2617	EDT/EDL, hepatorrhaphy, packing, suturing for pulmonary laceration	—	Dead
18	54	F	Mesenterium, Small intestine, Pulmonary contusion, C1/2 dislocation	50	0.1325	EDL, repair, packing	Anastomosis	Alive
19	20	M	Liver, Spleen, L-forearm fx, R-femur fx	27	0.6425	EDT/EDL, packing	—	Dead
20	37	M	Liver, HV, Heart (RV), R-pneumothorax	41	0.8997	EDT/EDL, cardiorrhaphy, hepatorrhaphy, packing	hepatorrhaphy	Alive
21	62	M	Mesenterium, Pulmonary contusion	25	0.3432	EDL, repair, packing	Anastomosis	Alive
22	84	M	Small intestine, Pelvic fx	25	0.6938	EDL, repair, packing	—	Dead
23	62	M	Rectum, Bladder, Pelvic fx, L-open femur fx, L-open tibia fx	50	0.1417	EDL, packing	colostomy	Dead
24	70	M	Liver, Flail chest, Pulmonary contusion, Skull fx, pneumoencephalus, L-open tibia fx	41	0.2133	hepatorrhaphy, packing	depacking	Alive

25	40	M	Liver, Thoracic aorta	50	0.8927	hepatorrhaphy, packing	depacking	Alive
26	59	M	Pancreas, SPV	25	0.4125	EDL, repair, packing	—	Dead
27	78	F	Abdominal aorta, Bil-hemothorax, L1 fx	36	0.8272	EDT, aortorrhaphy, packing	—	Dead
28	76	F	Liver	9	0.9177	hepatorrhaphy, packing	depacking	Alive
29	25	M	Liver, IVC, Facial bone fx, R-humeral fx	24	0.8910	hepatorrhaphy, IVC repair packing	depacking	Dead
30	57	F	Lt-Kidney, Spleen, Flail chest, L-hemothorax, Pelvic fx, L-open tibia fx	57	0.1377	EDL, splenectomy, packing	Anastomosis	Dead
31	22	F	Liver, IVC	25	0.9292	hepatorrhaphy, IVC repair, packing	depacking	Alive
32	55	M	R-massive hemothorax, Pelvic fx	41	0.3790	EDT, packing	—	Dead
33	73	F	L-ext-iliac vein, Flail chest, Pelvic fx, T-SAH	41	0.0332	repair, packing	depacking	Alive
34	58	M	Liver, Flail chest, Pelvic fx	66	0.1526	EDL, packing	hepatic resection, repacking	Dead

DCS, damage control surgery; ISS, injury severity score; Ps, probability of survival; fx, fracture; EDT, emergency department thoracotomy; EDL, emergency department laparotomy; DP, distal pancreatectomy; PD, pancreatoduodenectomy; T-SAH, traumatic subarachnoid hemorrhage; DAI, diffuse axonal injury; SDH, subdural hematoma; RV, right ventricle; LA, left atrium; IVC, inferior vena cava; PV, portal vein; SMV, superior mesenteric vein; SPV, splenic vein; HV, hepatic vein

Table 2 Clinical Characteristics of Patients between Two Groups

	Survivors group	Non-survivors group	p value
Age	47.7 ± 16.3	55.6 ± 24.3	0.41
Gender (M/F)	13/6	12/3	0.71
Mechanism of injury (Blunt/Penetrate)	18/1	15/0	1.00
ISS	32.2 ± 13.0	39.7 ± 13.8	0.23
Ps	0.70 ± 0.36	0.45 ± 0.30	0.10

ISS, injury severity score; Ps, probability of survival

rate after DCS among patients who possessed all three indicators (28.6%) was significantly lower than that among patients who did not possess all three indicators (75.0%; p=0.014; **Table 3**). Also, the mortality rate increased as the number of indicators increased (**Fig. 2**).

Discussion

Our results show that the presence of all three indicators—a systolic blood pressure of less than 90 mm Hg, a base excess of less than −7.5 mmol/L, and a core temperature less than 35.5℃ at the start of surgery—suggests a poor prognosis.

The indications for DCS are based on the physiologic status of the patient and technical issues related to the surgical procedure. Moore et al.[11] and Shapiro et al.[8] have suggested the following indications for DCS: 1) an inability to achieve hemostasis as a result of coagulopathy, 2) inaccessible major venous injury, 3) a time-consuming procedure in a patient with a suboptimal response to resuscitation, 4) the management of extra-abdominal life-threatening injury, 5) the need to reassess the intra-abdominal contents, and 6) the inability to reapproximate the abdominal fascia as a result of visceral edema. Coagulopathy is the most common reason for performing DCS, although many reports have shown that the survival rate is lower after DCS once coagulopathy or a metabolic disorder has developed.

The generally accepted indications for DCS[12–16] include a core temperature at the time of laparotomy <35℃, a pH <7.2, a base excess <−15

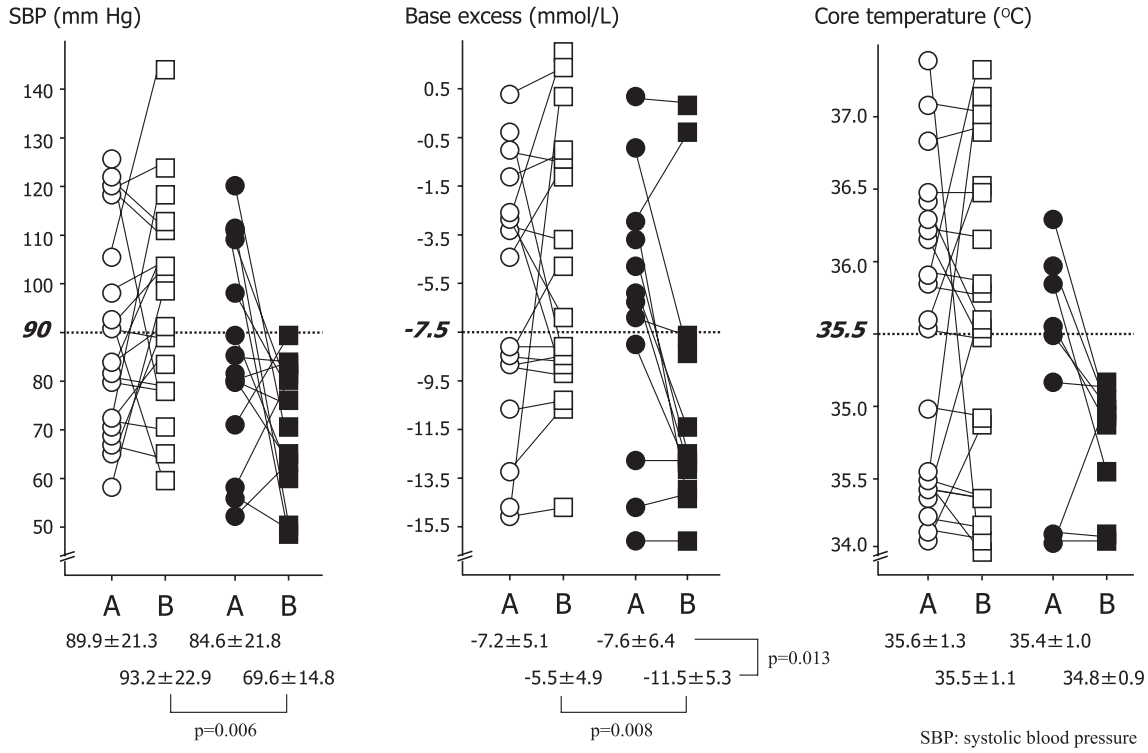

Fig. 1 Three indicators on ED arrival (A) and at the start of surgery (B), in the survivors group (○—□) and the nonsurvivors group (●—■). SBP, systolic blood pressure

Table 3 Survival Rate after DCS Based on Three Clinical Indicators at the Start of Surgery

	Alive	Dead	
Triad (+)	4 (28.6%) [a]	10	14
Triad (−)	15 (75.0%)	5	20
	19	15	34

[a] p = 0.014 compared to Triad (−)

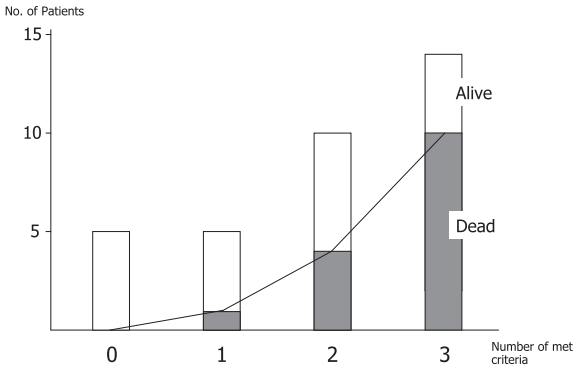

Fig. 2 DCS success rates as a function of the number of criteria fulfilled (systolic blood pressure less than 90 mmHg, base excess less than −7.5 mmol/L, and core temperature less than 35.5℃ at the start of surgery).

mmol/L (age <55 years) or <6.0 mmol/L (age >55 years), and PT and APTT <50% of normal. Moore et al.[6] have reported that severe coagulopathy (PT > twice normal and APTT > twice normal), massive rapid blood transfusion (10 units/4 hours), and persistent cellular shock (oxygen consumption index <110 mL/min/M^2 and lactate >5 mmol/L) were predictors of death. Rotondo and his colleagues[2] have reported that DCS may be triggered by the transfusion of 10 or more units of packed red-blood cells (estimated blood loss > 4 liters). Therefore, DCS should be performed before the patient's condition deteriorates to this degree, and readily accessible data are essential during resuscitation and surgery.

However, several problems exist with these criteria. For example, the oxygen consumption index is not always immediately available. Results of coagulation tests, including determinations of PT and APTT, may not always accurately reflect the coagulation abnormality in patients with hypothermia, because such tests might be performed at 37℃, not at the patient's actual body temperature. Massive blood transfusion and crystalloid infusion themselves lead to hemodilution and severe coagulopathy[5,6,11,17,18]; consequently,

knowing the amount of blood loss during primary resuscitation would be ideal. Accurately determining blood loss is difficult, however, because the amount of blood lost before the patient's arrival at the hospital is rarely available. Therefore, we felt that the criteria for deciding to perform DCS should be simplified; thus, the objective of this study was to establish a practical and reliable standard for emergency physicians and surgeons.

Among several recorded vital signs and laboratory data, we selected three variables: SBP, base excess, and core temperature at the time of ED arrival and at the start of surgery. In particular, SBP is a new factor that has not been described in previous reports. Although our data showed no relationship between the response to initial fluid resuscitation and the outcome of DCS, there is a significant statistical difference in SBP at the start of surgery between survivors and nonsurvivors.

Persistent hemodynamic instability typically leads to excessive fluid administration. As mentioned above, massive crystalloid infusion and blood transfusion can contribute to coagulopathy and increase blood loss. Hess and his coworkers[19] have described that Acute Coagulopathy of Trauma-Shock (ACoTS) is altered by subsequent events and treatments, in particular acidemia, hypothermia, and dilution. The coagulopathy of trauma is the result of multiple independent but interacting mechanisms, including tissue trauma, shock, hemodilution, hypothermia, acidemia, and inflammation. It is also proposed the new concept of "ACoTS", in which initiation, of coagulation occur with activation of anticoagulant and fibrinolytic pathways. These mechanisms may contribute to traumatic coagulopathy.

However, coagulopathy can be understood classically as the result of metabolic acidosis, hypothermia, and hemodilution induced by administration of large volumes of fluids and blood; therefore, hemodynamic instability requiring administration of such volumes should be recognized before coagulation tests are performed. Thus, we suspect that SBP at the start of the surgical procedure, which reflects hemodynamic instability and the need for additional fluid resuscitation, might predict coagulopathy and be an important variable in deciding whether to perform DCS.

The base excess and core temperature can be readily determined in an emergency. The diagnosis of acidemia is based on the arterial blood pH. However, pH might not accurately reflect the severity of metabolic acidosis in a patient because of the effects of respiratory compensation; therefore, we focused on the value of the base excess rather than the pH. Our study did not show a significant difference in the mean core temperature at the time of ED arrival and at the start of surgery in the two groups. However, core temperature is a classic, important, and simple variable for determining the need to perform DCS and can be measured easily and quickly in an ED or OR.

We feel that the decision to perform DCS should not be made according to strict criteria, because once coagulopathy has passed the "point of no return," any efforts to achieve hemostasis will be unsuccessful[20]. Because the DCS success rate among cases in which all three criteria were met (28.6%) was significantly lower than that among cases in which they were not (75.0%), the presence of all three indicators might mark the "point of no return." Consequently, we concluded that surgeons should decide to perform DCS before all three indicators are present. Mikhail[21] has similarly suggested that the injury-incision time should be shortened to ensure that the operation is started before the patient's physiologic limit is reached, defined as the onset of the triad of hypothermia, acidosis, and coagulopathy.

Recognizing when all three indicators are present is the key point. If the decision to perform DCS is made too early, too many unnecessary DCS procedures will be performed. On the other hand, our results suggest the decision to perform DCS should be made when only one or two criteria are fulfilled at the start of surgery. Although our proposed criteria are not strict and may broaden the indications for DCS, leading to an increase in the number of DCS procedures, saving lives must be the priority, and 'over-triage' may be acceptable when an appropriate decision-making protocol has been followed.

The limitations of this investigation include its retrospective design, the small number of subjects, the uncontrolled examinations, and the fact that the final decision to perform DCS depended largely on the surgeon. Further study involving a larger numbers of patients is needed, even if such studies are also retrospective, because attempting a randomized controlled study would be difficult given the small number of trauma cases treated at our center.

In conclusion, we suggest that surgeons should decide to perform DCS when only one or two following criteria are fulfilled: a systolic blood pressure less than 90 mm Hg, a base excess less than −7.5 mmol/L, and a core temperature less than 35.5℃ at the start of the surgical procedure. With this concept in mind, we believe that achieving a high survival rate in patients who have sustained severe torso trauma requires a high rate of DCS, even if the number of unnecessary DCS procedures may increase.

References

1. Burch JM, Ortiz VB, Richardson RJ, Martin RR, Mattox KL, Jordan GL: Abbreviated laparotomy and planned reoperation for critically injured patients. Ann Surg 1992; 215: 476–484.
2. Cosgriff N, Moore EE, Sauaia A, Moyniham MK, Burch JM, Galloway B: Predicting life-threatening coagulopathy in the massively transfused trauma patient: Hypothermia and acidosis revisited. J Trauma 1997; 42: 857–862.
3. Hirshberg A, Mattox KL: Planned reoperation for severe trauma. Ann Surg 1995; 222: 3–8.
4. Moore EE: Staged laparotomy for the hypothermia, acidosis, and coagulopathy syndrome. Am J Surg 1996; 172: 405–410.
5. Rotondo MF, Schwab CW, McGonigal MD, et al.: "Damage control": an approach for improved survival in exsanguination penetrating abdominal injury. J Trauma 1993; 35: 375–383.
6. Rotondo MF, Zonies DH: The damage control sequence and underlying logic. Surg Clin North Am 1997; 77: 761–778.
7. Sharp KW, Locicero RJ: Abdominal packing for surgically uncontrollable hemorrhage. Ann Surg 1992; 215: 467–475.
8. Shapiro MB, Jenkins DH, Schwab CW, Rotondo MF: Damage control: Collective review. J Trauma 2000; 49: 969–978.
9. Committee on Trauma: Chapter 3 Shock. In Advanced Trauma Life Support for Doctors, 1997; pp 87–124, America College of Surgeons, Chicago.
10. Boyd CR, Tolson MA, Copes WS: Evaluating trauma care: The TRISS method. J Trauma 1987; 27: 370–378.
11. Moore EE, Burch JM, Franciose RJ, Offner PJ, Biffl WL: Staged physiologic restoration and damage control surgery. World J Surg 1998; 22: 1184–1191.
12. Abramson D, Scalea TM, Hitchcock R, Trooskin SZ, Henry SM, Greenspan J: Lactate clearance and survival following injury. J Trauma 1993; 35: 584–589.
13. Cushman JG, Feliciano DV, Renz BM, et al.: Iliac vascular injury: Operative physiology related to outcome. J Trauma 1997; 42: 1033–1040.
14. Davis JW, Kaups KL: Base deficit in the elderly: A marker of severe injury and death. J Trauma 1998; 45: 873–877.
15. Feliciano DV, Moore EE, Mattox KL: Trauma damage control. In Trauma 5th ed (Moore EE, Feliciano DV, Mattox KL, eds), 2004; pp 877–900, McGraw-Hill, New York.
16. Tremblay LN, Feliciano DV, Rozyski GS: Assessment of initial base deficit as a predictor of outcome: Mechanism of injury does make a difference. Am Surg 2002; 68: 689–693.
17. Ferrara A, MacArthur JD, Wright HK, Modlin IM, McMillen MA: Hypothermia and acidosis worsen coagulopathy in the patient requiring massive transfusion. Am J Surg 1990; 160: 515–518.
18. Phillips TF, Soulier G, Wilson RF: Outcome of massive transfusion exceeding two blood volumes in trauma and emergency surgery. J Trauma 1987; 27: 903–910.
19. Hess JR, Brohi K, Dutton RP, et al.: The coagulopathy of trauma: a review of mechanisms. J Trauma 2008; 65: 748–754.
20. Cue JI, Cryer HG, Miller FB, Richardson JD, Polk HC Jr: Packing and planned reexploration for hepatic and retroperitoneal hemorrhage: Critical refinements of a useful technique. J Trauma 1990; 30: 1011–1013.
21. Mikhail J: The trauma triad of death: hypothermia, acidosis, and coagulopathy. AACN Clin Issues 1999; 10: 85–94.

(Received, June 2, 2009)
(Accepted, August 26, 2009)