

Damage control resuscitation: history, theory and technique

Chad G. Ball, MD, MSc

From the University of Calgary and Foothills Medical Centre, Calgary, Alta.

Accepted for publication
Feb. 4, 2013

Correspondence to:

C.G. Ball
University of Calgary
Foothills Medical Centre
1403–29 St. NW
Calgary AB T2N 2T9
ball.chad@gmail.com

DOI: 10.1503/cjs.020312

Damage control resuscitation (DCR) represents the natural evolution of the initial concept of damage control surgery. It currently includes early blood product transfusion, immediate arrest and/or temporization of ongoing hemorrhage (i.e., temporary intravascular shunts and/or balloon tamponade) as well as restoration of blood volume and physiologic/hematologic stability. As a result, DCR addresses the early coagulopathy of trauma, avoids massive crystalloid resuscitation and leaves the peritoneal cavity open when a patient approaches physiologic exhaustion without improvement. This concept also applies to severe injuries within anatomical transition zones as well as extremities. This review will discuss each of these concepts in detail.

La réanimation salvatrice et conservatrice découle naturellement du concept initial de chirurgie salvatrice et conservatrice. Ce concept repose actuellement sur l'administration rapide de transfusions sanguines, l'arrêt et(ou) la temporisation immédiats de l'hémorragie active (c.-à-d., dérivations intravasculaires temporaires et(ou) tamponade par ballonnet), de même que le rétablissement du volume sanguin et la stabilisation de l'état physiologique et hématologique. Ainsi, la réanimation salvatrice et conservatrice corrige rapidement la coagulopathie post-traumatique, permet d'éviter les mesures de réanimation par cristalloïdes et laisse la cavité péritonéale ouverte lorsqu'un patient atteint l'épuisement physiologique sans amélioration. Le concept s'applique aussi aux blessures graves affectant des zones anatomiques de transition ou les membres. Cette analyse abordera chacun des éléments du concept plus en détail.

Damage control is a Navy term defined as “the capacity of a ship to absorb damage and maintain mission integrity.”¹ When applied to surgery and critically ill patients, damage control surgery (DCS) incorporates fundamental tenets: arresting surgical hemorrhage, containing gastrointestinal spillage, inserting surgical sponges and applying a temporary abdominal closure. This sequence is followed by immediate transfer to the intensive care unit (ICU) with subsequent rewarming, correction of coagulopathy and hemodynamic stabilization. Return to the operating theatre is then pursued 6–48 hours later for a planned re-exploration that includes definitive repair and primary fascial closure if possible. In essence, a typical operative sequence is interrupted by completing only the most crucial aspects during the first phase.

Although the adaption of the term “damage control” to the field of traumatology can be credited to Schwab and colleagues² in 1993, its dominant principles are more accurately rooted in the 1976 address by Lucas and Ledgerwood³ to the American Association for the Surgery of Trauma. More specifically, they described a small series of patients who underwent sponge-based packing of major liver injuries.³ This concept was reiterated shortly thereafter by Calne and colleagues⁴ and by Feliciano and colleagues⁵ in 1979 and 1981, respectively. Despite these small series outlining the success of perihepatic packing, the visionary extrapolation of this principle to patients with multiple, concurrent life-threatening injuries and major coagulopathy was not published until 1983.⁶ Stone and colleagues⁶ retrospectively described 31 patients in whom major bleeding diatheses developed. Of these,

17 patients underwent the modern damage control principles of arresting surgical hemorrhage and abbreviating the operative intervention. This led to the survival of 11 patients who were predicted to have a lethal coagulopathy. Once DCS was established, it was quickly marketed into other disciplines, including but not limited to neck,⁷ vascular,⁸ orthopedic,⁹ thoracic¹⁰ and military injuries.¹¹ Although each discipline uses DCS in a slightly different manner, it is clear that the DCS approach leads to improved survival in patients with either blunt or penetrating injuries who are approaching physiologic exhaustion.¹²

The widespread propagation of DCS throughout the trauma community has led to an overuse of this technique. More specifically, patients with multiple injuries who are not approaching physiologic exhaustion are often exposed to the potential risks associated with an open abdomen and the DCS process. As a result, the pertinent question is that of who truly requires DCS. The succinct response is patients who are more likely to die from an uncorrected state of shock than from failure to complete organ repairs. Depending on the centre, these patients with severe metabolic compromise encompass 3%–8% of all severely injured patients (penetrating v. blunt; military v. civilian). In essence, they continue to experience the sequelae of tissue shock that manifests as persistent hypothermia, persistent metabolic acidosis and nonmechanical (i.e., nonsurgical) bleeding. Damage control surgery triggers, therefore, include core temperature less than 35°C, pH less than 7.2, base deficit greater than –15 and/or significant coagulopathy.^{13–16} It must be emphasized, however, that not all patients with initial physiologic deficits require DCS. With rapid arrest of hemorrhage, as well as ongoing resuscitation, some patients will show dramatic improvement in all parameters on repeated intraoperative blood gases. It should also be stated that patients with multiple intra-abdominal injuries do not always experience metabolic failure.

The natural extension and development of DCS has been damage control resuscitation (DCR). This concept includes DCS, but also the early initiation of blood product transfusions, reduced crystalloid fluid administration, permissive hypotension in selected populations and immediate hemorrhage control (operative or angiographic). In summary, DCR is a structured, mobile intervention that can be delivered to a critically ill patient in any location (emergency department, interventional radiology suite, operating theatre and/or ICU). Basic principles include arresting hemorrhage; restoring blood volume; and correcting coagulopathy, acidosis and hypothermia.

MASSIVE TRANSFUSION

Although the traditional definition of a massive transfusion is 10 units or more of red blood cells (RBC) within a 24-hour period, this term has been modified to better

reflect true coagulation biochemistry with regards to both the amount of blood product and the associated time interval.^{16,17} The 3% and 8% of civilian and military patients, respectively, who require a massive transfusion have injuries that are predictably associated with high mortality (27%–51%).¹⁷ Furthermore, the early coagulopathy of trauma is a well-recognized entity that is present at the time of admission in more than 25% of injured patients with a base deficit greater than 6.¹⁸ Equally interesting, although coagulopathy was historically viewed as a byproduct of resuscitation, hemodilution and hypothermia, the bloody vicious cycle is now understood to be substantially more complex.¹⁹ Tissue trauma, shock, hemodilution, hypothermia, acidemia and inflammation all play key trigger roles in the acute coagulopathy of trauma in patients with shock.¹⁹ The improved understanding of inter-relationships and recognition of these 6 key initiators of coagulopathy supports the modern use of massive transfusion protocols (MTPs). In brief, a modern MTP aims to approximate delivery of 1:1:1 ratio of RBC: fresh frozen plasma: platelets.¹⁹ By addressing the early coagulopathy of trauma, MTPs have been shown to improve mortality in patients with multiple injuries.^{20,21} While the specific structure of MTPs varies slightly from centre to centre,²¹ they are all approximations of the fresh whole blood resuscitation principles reported by Sheldon and colleagues²² in 1975. It should also be noted that reasonable scientific concern remains with regard to the apparent improvement in survival associated with MTPs.²³ The possibility of a strong survival bias (i.e., surviving long enough to receive the most RBC units) is significant.²³

Additional benefits of a formal MTP include earlier administration of blood products during resuscitations, improved blood banking efficiency, decreased total blood product use during a hospital stay and significant economic savings.²⁴ Another major benefit surrounds the avoidance of excess crystalloid fluid administration.²⁵ This reduction in crystalloid volume during resuscitation minimizes several associated side effects, including reperfusion injury, increased leukocyte adhesion and inflammation, associated acidosis and resulting acute respiratory distress syndrome, systemic inflammatory response syndrome and multiorgan failure.^{25–28} Excess crystalloid administration also remains an obstacle to obtaining early definitive fascial closure secondary to both visceral and abdominal wall edema.

In summary, if the initiation of an MTP is accurately based on rapid recognition of physiologic exhaustion secondary to persistent hemorrhage, then it also typically acts as a trigger for the entire damage control process. It must be re-emphasized, however, that some severely injured patients will improve dramatically with an MTP and reverse their physiologic derangements to an extent that allows the surgeon to complete their operative intervention (i.e., rather than leaving the abdomen open).

VASCULAR DAMAGE CONTROL TECHNIQUES

Although it is clear that arresting ongoing hemorrhage is the most crucial of damage control tenets, vascular damage control has been traditionally limited to vessel ligation. More recently, however, balloon catheter tamponade and temporary intravascular shunts (TIVS) have increased in popularity. The impressive utility of balloon catheters for tamponade of exsanguinating hemorrhage has a long history, dating back more than 50 years.²⁹ Although this technique was originally described for esophageal varices,³⁰ it was quickly extended to patients with traumatic vascular and solid organ injuries.³¹ Since the initial treatment of an iliac arteriovenous lesion in 1960,³ balloon catheters have also been used for cardiac,³² aortic,³³ pelvic,³⁴ neck (carotid, vertebral and jugular),^{35,36} abdominal vascular,³⁷ hepatic,³⁸ subclavian,³⁹ vertebral²⁹ and facial trauma.⁴⁰ While this technique was originally intended as an intraoperative endovascular tool,²⁹ it has since been used as an emergency department manoeuvre, with the balloon being placed outside of the lumen of the injured vessel.^{41,42}

Modern indications for this damage control technique are limited, primarily because routine methods for controlling hemorrhage, such as direct pressure, are typically successful. As a result, indications for catheter tamponade include inaccessible (or difficult to access) major vascular injuries, large cardiac injuries and deep solid organ parenchymal hemorrhage (liver and lung).^{29,42} Of interest, the type of balloon catheter (Foley, Fogarty, Blakemore or Penrose with red rubber Robinson) as well as the duration of indwelling can vary substantially.

In summary, balloon catheter tamponade is a valuable tool for damage control of exsanguinating hemorrhage when direct pressure fails or when tourniquets are not applicable. The technique can be used in several anatomic regions and for variable patterns of injury. Prolonged catheter placement for maintenance of hemostasis is particularly useful for central hepatic gunshot injuries.⁴²

Temporary intravascular shunts (TIVS) are intraluminal synthetic conduits that offer nonpermanent maintenance of arterial inflow and/or venous outflow.⁴³ As a result, they are frequently life- and limb-saving when patient physiology is hostile. By bridging a damaged vessel and maintaining blood flow, they address both acute hemorrhage and critical warm ischemia of distal organs and limbs.

Although Eger and colleagues⁴⁴ are commonly credited for pioneering the use of TIVS in modern vascular trauma, this technique was initially used by Carrel in animal experiments.⁴⁵ The first documented use in humans occurred in 1915 when Tuffier⁴⁶ used paraffin-coated silver tubes to bridge injured arteries. This technique evolved from glass to plastic conduits in World War II,⁴⁷ and continues to vary both in structure and material among today's surgeons.⁴⁸

Modern indications for TIVS include replantation, open extremity fractures with concurrent extensive soft tissue loss

and arterial injury (Gustilo IIIc), peripheral vascular damage control, truncal vascular damage control and temporary stabilization before transport.^{43,49} Although the understanding of TIVS use for military and civilian settings is increasing,⁴⁸ the optimal shunt material, dwell time and anticoagulation requirements remain poorly studied. It can be noted, however, that TIVS are remarkably durable and rarely clot unless they are too small (diameter), kink because of inappropriate length and/or are placed in an extremity without appropriate (or shunted) venous outflow (venous hypertension leads to arterial thrombosis).⁴⁹ Although the literature is peppered with case series and reports on practical experience using TIVS, the most dramatic example of their utility surrounds the near-complete disappearance of limb loss following ligation of the common and/or external iliac arteries.⁴⁹ More specifically, despite similar injury and patient characteristics, the improvement in amputation rate from 47% to 0% in 22 patients with penetrating trauma after the introduction of TIVS at a high-volume trauma centre is remarkable.⁴⁹ Further advantages included an observed reduction in fasciotomies (from 93% to 43%) and extra-anatomic bypasses (from 6 to 0).⁴⁹

Despite the penetrating mechanism dogma associated with TIVS over the past 40 years, the majority (64%) of TIVS in a large national database (National Trauma Data Bank; NTDB) were used in patients injured via a blunt mechanism.⁵⁰ Although the kinetic force of a motor vehicle collision (MVC) or MVC–pedestrian collision can be tremendous, TIVS is often discussed in the context of extremity damage control for gunshot wounds in patients with hostile physiology.⁵⁰ This NTDB analysis, however, indicated that most extremity TIVS were actually placed for blunt vascular trauma associated with extensive orthopedic and/or soft tissue injuries (74%). They were also most often used as a temporizing manoeuvre to provide distal flow to a limb while orthopedic injuries are assessed and fixated. The use of TIVS for this scenario is well recognized and documented to significantly reduce the rate of amputation. In the patients who did not receive TIVS for fractures and soft tissue defects, it appears that shunting was used as an extremity damage control technique in those who presented with hemodynamic instability and severe base deficits (26%).⁵⁰ These patients displayed a much lower level of subsequent amputation than patients with blunt trauma with concurrent fractures and soft tissue trauma.

In addition to using TIVS in patients with blunt injuries, the NTDB also indicates that this technique is being performed relatively uncommonly across a wide range of hospitals.⁵⁰ Of 111 trauma centres using TIVS, only 6 used 5 or more shunts throughout the study period. In addition, only 3 centres used more than 10 shunts.

In summary, TIVS appear to be useful in any scenario with a major vascular injury and concurrent hostile patient physiology. This includes cases of blunt MVC trauma with concurrent severe extremity fractures and/or

soft tissue injuries. Despite their simplicity, however, TIVS are underused.

ABDOMINAL COMPARTMENT SYNDROME

Many of the dominant risk factors for abdominal compartment syndrome (ACS) mirror variables that have been proposed as physiologic triggers for DCS/DCR.^{51,52} When taken as a whole, these indicators (pH, base deficit, core temperature) clearly reflect the extreme biochemical and physiologic stress that characterizes many of our most critically ill patients. Closing abdomens in patients manifesting physiologic extremis often leads to ACS, as first demonstrated by Morris and colleagues⁵³ in 1993. With fascial closure, the authors described severe abdominal distension in concert with raised peak airway pressures, CO₂ retention and oliguria.⁵³ The 63% mortality associated with subsequent reperfusion injury (i.e., after removing sponges from the intraperitoneal cavity during the second operation) that they observed was dramatic and also emphasized the importance of preventing “recurrent” or “tertiary” ACS.⁵⁴ In summary, these publications indicate that maintaining an open abdomen via a temporary abdominal closure, and therefore planning a delayed fascial closure, is not only a vital component of DCR, but is also a clear method for preventing ACS. Despite the increased understanding surrounding this anatomic and physiologic complication, however, it is clear that clinicians require more education with regard to preventing, monitoring and treating all forms of ACS.⁵⁵ More specifically, while the incidence of primary ACS (i.e., injury to the torso) has decreased dramatically over the past decade,⁵¹ continued vigilance is crucial to guard against secondary (i.e., resuscitation-induced ACS) and recurrent ACS (i.e., creating an inadequate fascial opening and/or closing the fascia too early in repeat operations).

OPEN ABDOMINAL MANAGEMENT

The concept of delaying abdominal wall closure is credited to Stone and colleagues⁵⁶ at Grady Memorial Hospital in 1981. Among 167 patients, mortality approximated 85% in those whose abdomens were closed under tension, compared with only 22% who underwent delayed fascial closure. This truly remarkable report altered the DCS landscape dramatically. Unfortunately, the open abdomen is also responsible for significant short-term (fluid and protein loss, sepsis, intestinal fistulae, nursing care challenges, economic costs) and long-term (chronic physical discomfort, physique embarrassment, delayed return to work, poor quality of life) morbidity.⁵⁷⁻⁶⁰

Several techniques for covering the open abdomen at the index operation are available (Box 1). Because no single technique has been shown to be superior to others, a given trauma centre should use a single consistent option that

ensures familiarity among all surgeons, house staff and nurses. This allows accurate assessment of fluid losses as well as intermittent troubleshooting. A simple and cost effective choice is a large sterile radiograph cassette drape with 2 closed suction drains and a covering plastic adhesive drape. Avoiding commercial negative suction devices during the initial critical phase also prevents any concern for generating recurrent ACS.⁶¹

It is clear that as the patient is exposed to multiple subsequent operative interventions, intestinal coverage (via endogenous abdominal wall or skin or split-thickness skin graft) must be achieved as soon as possible to limit the development of fistulae. It is also evident that regardless of technique, severely injured patients more commonly achieve fascial closure during their initial hospital stay than their nontrauma, acute care surgery counterparts. If closed too early, ACS, fascial dehiscence, necrotizing fasciitis and ventilation challenges are notable complications. Despite the poor methodology inherent in the open abdomen literature (i.e., mixed patient cohorts, lack of complete inclusion, ignorance of nonsurvivors, variable individual surgeon effort and interest), it is evident that the application of negative suction dressings at subsequent operations has improved closure rates and reduced complications, such as intestinal fistulae. Whether “homegrown”⁶² or commercially derived,⁶³ these technologies have become commonplace. The 2 dominant principles when using negative suction therapy remain: maintenance of the peritoneal/abdominal domain and continuous and progressive tension on the midline abdominal wall. These goals are achieved by insertion of a plastic barrier deep into the paracolic gutters (maximally lateral to prevent adhesions between the colon and abdominal wall) and by generation of midline abdominal wall tension using nonfascial retention sutures or commercial systems. It should also be noted that intra-abdominal pressures often exceed “normal” (> 20 mm Hg)

Box 1. Open abdomen coverage techniques

- Skin only
- Towel clip
- Silastic sheet
- Bogota bag
- 3 L genitourinary bag
- Steri-drape/x-ray cassette
- Zippers
- Slide fasteners
- Velcro analogue/Wittmann
- Polypropylene mesh
- Polyglycolic/polyglactac acid mesh
- Polytetrafluoro-thylene mesh
- Parachute silk
- Hydrogel/aquacel
- Loban
- Vacuum pack
- Abdominal wound vacuum-assisted closure
- Bioprosthetics

immediately after progressive increases in tension at the midline during repeat laparotomy and attempted closure. This typically abates over the subsequent few hours and is considered acceptable in the absence of end organ ischemia (decreased urine output, increased airway pressures). As a result, it is considered fundamentally different from the acute phase of ACS. If the intra-abdominal pressure does not normalize, however, the abdomen must be reopened to prevent recurrent ACS.

In summary, an individual patient with an open abdomen will either continue to improve, mobilize fluid and allow gradual abdominal closure via repeat laparotomies, or continue to be challenged with sepsis and multiorgan failure, will not mobilize fluid, and will eventually require skin graft coverage. Enteroatmospheric fistulae must also be prevented at all costs. This morbidity not only complicates short-term management, but also eventual abdominal wall reconstruction (component separation, modified component separation). If present, these fistulae are best intubated by soft rubber catheters placed within the sponge material of negative pressure suction dressing.⁶⁴ Over time, this will allow the clinician to develop a granulation plate around the fistulae appropriate for a skin graft (i.e., conversion into a stoma).

A detailed discussion of the tiered algorithm for abdominal wall reconstruction, as well as the indications for occasional use of biologic materials, is beyond the goals of this review. The appropriate timing of reconstruction (8–12 mo) is crucial to the success of the repair (adhesions v. rectus muscle lateral retraction). Extensive experience in reconstructive techniques is crucial to ensure acceptable outcomes. These principles include but are not limited to timing, sequencing (stoma reversal, fistula closure), ensuring adequate skin coverage, sparing of periumbilical perforators, minimally invasive lateral releases and management of wound complications.

HYBRID CARE SUITES

Given that DCR is dedicated to our most critically injured patients, it is not surprising that the development of a single location (i.e., resuscitation with angiography, percutaneous techniques, and operative repair [RAPTOR] suites) where percutaneous therapies, operative interventions, cross-sectional imaging and critical care can all be delivered holds substantial promise.⁶⁵ This concept modifies care from a location-based approach to a truly disease and urgency-based algorithm. The best example is a patient with ongoing hemorrhage from an unstable pelvic fracture in the context of additional associated injuries that may or may not require emergent operative intervention. The concerns associated with this technology are the tremendous cost of building hybrid suites and the clinician training paradigm required for safely performing hybrid procedures.⁶⁵

CONCLUSION

Damage control resuscitation currently includes early blood product transfusion, immediate arrest and/or temporization (i.e., TIVS and balloon tamponade) of ongoing hemorrhage, and restoration of blood volume and physiologic/hematologic stability. As a result, DCR addresses the early coagulopathy of trauma, avoids massive crystalloid resuscitation and leaves the peritoneal cavity open when a patient approaches physiologic exhaustion without improvement. Future evolution of the DCR concept will include further elucidation of personalized resuscitations (individual blood product ratios based on point of care testing) as well as introduction of hybrid angiography operating suites in centres with resource availability.

Competing interests: None declared.

References

1. *Warfare Manual*. Department of the Navy. The Department: Washington (DC);1996.
2. Rotondo MF, Schwab CW, McGonigal MD, et al. Damage control: an approach for improved survival in exsanguinating penetrating abdominal injury. *J Trauma* 1993;35:375-82.
3. Lucas CE, Ledgerwood AM. Prospective evaluation of hemostatic techniques for liver injuries. *J Trauma* 1976;16:442-51.
4. Calne RY, McMaster P, Pentlow BD. The treatment of major liver trauma by primary packing with transfer of the patient for definitive treatment. *Br J Surg* 1979;66:338-9.
5. Feliciano DV, Mattox KL, Jordan GL Jr. Intra-abdominal packing for control of hepatic hemorrhage: a reappraisal. *J Trauma* 1981;21:285-90.
6. Stone HH, Strom PR, Mullins RJ. Management of the major coagulopathy with onset during laparotomy. *Ann Surg* 1983;197:532-5.
7. Firoozmand E, Velmahos GC. Extending damage control principles to the neck. *J Trauma* 2000;48:541-3.
8. Granchi T, Schmittling Z, Vascuez J, et al. Prolonged use of intraluminal arterial shunts without systemic anticoagulation. *Am J Surg* 2000;180:493-6.
9. Scalea TM, Boswell SA, Scott JD, et al. External fixation as a bridge to nailing for patients with multiple injuries and with femur fractures: damage control orthopedics. *J Trauma* 2000;48:613-21.
10. Vargo DJ, Battistella FD. Abbreviated thoracotomy and temporary chest closure: an application of damage control after thoracic trauma. *Arch Surg* 2001;136:21-4.
11. Holcomb JB, Helling TS, Hirshber A. Military, civilian and rural application of the damage control philosophy. *Mil Med* 2001;166:490-3.
12. Nicholas JM, Rix EP, Easley KA, et al. Changing patterns in the management of penetrating abdominal trauma: the more things change, the more they stay the same. *J Trauma* 2003;55:1095-108.
13. Lier H, Krep H, Schroeder S, et al. The influence of acidosis, hypocalcemia, anemia, and hypothermia on functional hemostasis in trauma. *J Trauma* 2008;65:951-60.
14. Wyrzykowski AD, Feliciano DV. Trauma damage control. In: Feliciano DV, Mattox KL, Moore EE, editors. *Trauma*. 6th ed. New York: McGraw-Hill Medical; 2008. p. 851-70.
15. Cushman JG, Feliciano DV, Renz BM, et al. Iliac vessel injury: operative physiology related to outcome. *J Trauma* 1997;42:1033-40.
16. Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma* 2007;62:307-10.

17. Como JJ, Dutton RP, Scalea TM, et al. Blood transfusion rates in the care of acute trauma. *Transfusion* 2004;44:809-13.
18. Brohi K, Singh J, Hern M, et al. Acute traumatic coagulopathy. *J Trauma* 2003;54:1127-30.
19. Hess JR, Brohi K, Dutton RP, et al. The coagulopathy of trauma: a review of mechanisms. *J Trauma* 2008;65:748-54.
20. Borgman MA, Spinella PC, Perkins JG, et al. The ratio of blood products transfuse affects mortality in patients receiving massive transfusions at a combat support hospital. *J Trauma* 2007;63:805-13.
21. Dente CJ, Shaz BH, Nicholas JM, et al. Improvements in early mortality and coagulopathy are sustained better in patients with blunt trauma after institution of a massive transfusion protocol in a civilian level I trauma center. *J Trauma* 2009;66:1616-24.
22. Sheldon GF, Lim RC, Blaisdell FW. The use of fresh blood in the treatment of critically injured patients. *J Trauma* 1975;15:670-7.
23. Snyder CW, Weinberg JA, McGwin G, et al. The relationship of blood product ratio to mortality: Survival benefit or survival bias? *J Trauma* 2009;66:358-62.
24. O'Keeffe T, Refaai M, Tchorz K, et al. A massive transfusion protocol to decrease blood component use and cost. *Arch Surg* 2008;143:686-90.
25. Cotton BA, Guy JS, Morris JA, et al. Cellular, metabolic, and systemic consequences of aggressive fluid resuscitation strategies. *Shock* 2006;26:115-21.
26. Rhee P, Koustova E, Alam HB. Searching for the optimal resuscitation method: recommendations for the initial fluid resuscitation of combat casualties. *J Trauma* 2003;54:S52-62.
27. Pruitt BA Jr. Protection for excessive resuscitation: "pushing the pendulum back." *J Trauma* 2000;49:567-8.
28. Ball CG, Kirkpatrick AW. Intra-abdominal hypertension and the abdominal compartment syndrome. *Scand J Surg* 2007;96:197-204.
29. Feliciano DV, Burch JM, Mattox KL, et al. Balloon catheter tamponade in cardiovascular wounds. *Am J Surg* 1990;160:583-7.
30. Myhre JR. Balloon tamponade of hemorrhagic esophageal varices. *Tidsskr Nor Lægeforen* 1958;78:511-3.
31. Taylor H, Williams E. Arteriovenous fistula following disk surgery. *Br J Surg* 1962;50:47-50.
32. Pearce CW, McCool E, Schmidt FE. Control of bleeding from cardiovascular wounds: balloon catheter tamponade. *Ann Surg* 1966;163:257-9.
33. Foster JH, Morgan CV, Threlkel JB. Proximal control of aorta with a balloon catheter. *Surg Gynecol Obstet* 1971;132:693-4.
34. Sheldon GF, Winestock DP. Hemorrhage from open pelvic fracture controlled intraoperatively with balloon catheter. *J Trauma* 1978;18:68-70.
35. Belkin M, Dunton R, Crombie HD, et al. Preoperative percutaneous intraluminal balloon catheter control of major arterial hemorrhage. *J Trauma* 1988;28:548-50.
36. Bendahan J, Swanepoel E, Muller R. Tamponade of vertebral artery bleeding by Foley's catheter balloon. *Injury* 1994;25:473-4.
37. Smiley K, Perry MO. Balloon catheter tamponade of major vascular wounds. *Am J Surg* 1971;121:326-7.
38. Morimoto RY, Birolini D, Junqueira AR, et al. Balloon tamponade for transfusing lesions of the liver. *Surg Gynecol Obstet* 1987;164:87-8.
39. DiGiacomo JC, Rotondo MF, Schwab CW. Transcutaneous balloon catheter tamponade for definitive control of subclavian venous injuries: case reports. *J Trauma* 1994;37:111-3.
40. Sing RF, Sue SR, Reilly PM. Balloon catheter tamponade of exsanguinating facial hemorrhage: a case report. *J Emerg Med* 1998;16:601-2.
41. Navsaria P, Thoma M, Nicol A. Foley catheter balloon tamponade for life-threatening hemorrhage in penetrating neck trauma. *World J Surg* 2006;30:1265-8.
42. Ball CG, Wyrzykowski AD, Nicholas JM, et al. A decade's experience with balloon catheter tamponade for the emergency control of hemorrhage. *J Trauma* 2011;70:330-3.
43. Frykberg ER, Schinco MA. Peripheral vascular injury. In: Feliciano DV, Mattox KL, Moore EE, editors. *Trauma*. 6th ed. New York: McGraw-Hill Medical; 2008. p. 956-7.
44. Eger M, Goldman L, Goldstein A, et al. The use of a temporary shunt in the management of arterial vascular injuries. *Surg Gynecol Obstet* 1971;132:67-70.
45. Makins GH. *On gunshot injuries to the blood-vessels, founded on experience gained in France during the great war, 1914-1918 (1919)*. London: Simpkin, Marshall, Hamilton, Kent & Co; 1919.
46. Tuffier. French surgery in 1915. *Br J Surg* 1916;4:420-32.
47. Matheson NM, Murray G. Recent advances and experimental work in conservative vascular surgery. In: Hamilton Bailey, editor. *Surgery of modern warfare*. Vol. 1. Baltimore (MA): Williams and Wilkins; 1941. p. 324-7.
48. Ding W, Wu X, Li J. Temporary intravascular shunts used as a damage control surgery adjunct in complex vascular injury: collective review. *Injury* 2008;39:970-7.
49. Ball CG, Feliciano DV. Damage control techniques for common and external iliac artery injuries: Have temporary intravascular shunts replaced the need for ligation? *J Trauma* 2010;68:1117-20.
50. Ball CG, Kirkpatrick AW, Rajani RR, et al. Temporary intravascular shunts: When are we really using them according to the NTDB? *Am Surg* 2009;75:605-7.
51. Balogh ZJ, van Wessem K, Yoshino O, et al. Postinjury abdominal compartment syndrome: Are we winning the battle? *World J Surg* 2009;33:1134-41.
52. Balogh Z, McKinley BA, Holcomb JB, et al. Both primary and secondary abdominal compartment syndrome (ACS) can be predicted early and are harbingers of multiple organ failure. *J Trauma* 2003;54:848-59.
53. Morris JA Jr, Eddy VA, Binman TA, et al. The staged celiotomy for trauma: issues in unpacking and reconstruction. *Ann Surg* 1993;217:576-84.
54. Ball CG, Kirkpatrick AW, Karmali S, et al. Tertiary abdominal compartment syndrome in the burn injured patient. *J Trauma* 2006;61:1271-3.
55. Kirkpatrick AW, Laupland KB, Karmali S, et al. Spill your guts! Perceptions of Trauma Association of Canada member surgeons regarding the open abdomen and the ACS. *J Trauma* 2006;60:279-86.
56. Stone HH, Fabian TC, Turkleson ML, et al. Management of acute full-thickness losses of the abdominal wall. *Ann Surg* 1981;193:612-8.
57. Balogh ZA, Moore FA, Goettler CE. Surgical management of the abdominal compartment syndrome. In: Ivatury RR, Cheatham ML, Malbrain MLNG, et al., editors. *Abdominal compartment syndrome*. Georgetown: Landes Biomedical; 2006. p. 266-9.
58. Ball CG, Kirkpatrick AW, McBeth PB, et al. The secondary abdominal compartment syndrome: not just another post-traumatic complication. *Can J Surg* 2008;51:399-405.
59. Cheatham ML, Safcsak K, Llerena LE, et al. Long-term physical, mental and functional consequences of abdominal decompression. *J Trauma* 2004;56:237-41.
60. Cheatham ML, Safcsak K. Long-term impact of abdominal decompression: a prospective comparative analysis. *J Am Coll Surg* 2008;207:573-9.
61. Ouellet JF, Ball CG. Recurrent abdominal compartment syndrome induced by high negative pressure abdominal closure dressing. *J Trauma* 2011;71:785-6.
62. Brock WB, Barker DE, Burns RP. Temporary closure of open abdominal wounds: the vacuum pack. *Am Surg* 1995;61:30-5.
63. Garner GB, Ware DN, Cocanour CS, et al. Vacuum-assisted wound closure provides early fascial reapproximation in trauma patients with open abdomens. *Am J Surg* 2001;182:630-8.
64. Al-Khoury G, Kaufman D, Hirshberg A. Improved control of exposed fistula in the open abdomen. *J Am Coll Surg* 2008;206:397-8.
65. Ball CG, Kirkpatrick AW, D'Amours SK. The RAPTOR: Resuscitation with angiography, percutaneous techniques and operative repair. Transforming the discipline of trauma surgery. *Can J Surg* 2011;54:E3.