

Damage Control Resuscitation for the Special Forces Medic: Simplifying and Improving Prolonged Trauma Care

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ABSTRACT

Current operational theaters have developed to where medical evacuation and surgical assets are accessible in times comparable to the United States. While this has been an essential tool in achieving the best survivability on a battlefield in our history, the by-product of this experience is a recognized shortcoming in current protocols and capabilities of Special Forces medics for prolonged care. The purpose of this article is to provide a theory of care, identify training and support requirements, and to capitalize on current successful resuscitation theories in developing a more efficient and realistic capability under the worst conditions.

Our forces enjoy high confidence in rapid casualty evacuation and surgical intervention thanks to the proximity and availability of those assets in today's developed theaters. While prehospital care has evolved to an exceptionally high standard due to these capabilities, the ability of Special Forces (SF) medics to conduct independent and prolonged care has suffered because of them. Past experiences and future possibilities will require a return to prolonged, sustained care, as well as the development of new standards for all Special Operations Forces (SOF) medics based on the latest and proven theories. This capability can have significant application in today's missions ranging from Foreign Internal Defense in austere environments, a distant outstation isolated by weather, a Special Reconnaissance patrol inaccessible due to conditions in a mountain range, or even in short-term direct action missions when cut off from friendly forces and support. We can also take lessons from the recent Israeli invasion of southern Lebanon in 2006 when both conventional and Special Forces medics were forced into long term care. In this example of a modern low intensity conflict, even casualty evacuation over short distances was limited due to battlefield dynamics, troop disposition, and terrain.¹

Critical care and resuscitation between point of injury and surgical intervention may be the missing element here, and should include the capacity for extended care, delayed hand-off, and late surgical suc-

cess. Prehospital care is relative to the situation. For most, it is only the treatment given at the point of injury immediately prior to hand-off to an evacuation platform. In Special Operations those lines are neither so clear nor so rapid; the scope and duration of care provided by SF medics may equate to that of a physicians at more than one conventional level. Prehospital care for operations in austere environments includes: point of injury, evacuation concerns, resuscitation, prolonged care, as well as all the skills required of those (Figure 1). Sustaining a patient in an operational environment for days post-event is no easy task. And while maintenance of a casualty for 72 hours post-op is required training at the Special Warfare Center, the focus here is the implementation of current and proven strategies to maximize the patient's chances of survival.

Compared Stages of Care	
<u>Conventional Forces</u>	<u>Special Forces</u>
Point of Injury	Point of Injury
Evacuation	Resuscitation & Prolonged Care
Field Surgical Team	Evacuation
Evacuation	Surgical Intervention
Definitive Care	Definitive Care

Figure 1 – The stages of care differ remarkably not only due to the assets involved but by the scope of care required from SF medics and Independent Duty Corpsman when compared to conventional systems.

This article is the first of two meant to provide an approach to critical care using damage control resuscitation (DCR) as a guideline adapted for our use. DCR is a current and proven practice and provides aggressive and effective trauma management with minimal support while preparing the patient for the next level of care. DCR consists of two parts: first, keeping blood pressure at approximately 90mmHg, and second, to rapidly reverse coagulopathy and restore oxygen carrying capacity with fresh frozen plasma (FFP) and packed red blood cells (PRBC).² Since FFP and PRBC's are unavailable far forward, we are advocating the earlier and more aggressive use of type-specific fresh whole blood (FWB) as the only workable solution for salvaging patients with life-threatening injuries. Fresh whole blood delivers normal physiological ratios of essential elements, with more active clotting factors than banked component blood, and at normothermic temperatures.³ Indications for FWB use is based on patient presentation and lab results such as lactate, base deficit, pH, and hematocrit which will also later serve as endpoints in resuscitation, ensuring efficient therapeutic objectives. While "balanced" or "hypotensive" resuscitation works well in the short term, a patient left hypotensive and under-resuscitated for a prolonged period cannot be sustained. Trauma patients who do not normalize their pH or base deficit have significantly higher mortality at 24 hours and near universal mortality at 48 hours.⁴

Patient care in the austere environment is incomparable to that in a U.S. hospital. With that in mind, the scope of practice, therapeutic guidelines, procedures used, benefit vs. risk analysis, and clinical tenets, significantly differ from a civilian emergency room or even that of a combat surgical hospital (CSH). Many may question the standards of care recommended, but they probably do not appreciate the challenges SF medics face. Prolonged care in the primitive setting cannot support current hospital-based parameters, and a return to unconventional warfare practices is warranted and necessary. Strict clinical practices are respected and exercised, but not always attainable in our environment. Careful review of many long forgotten practices from previous conflicts may yield surprising results. The use of tourniquets, damage control surgery, plasma and whole blood transfusions, are all being resurrected with improved patient outcomes in the 21st century. Many believe the most difficult challenges are found in the austere environment, and this may be where DCR is of most benefit.

The following recommendations to SF medics were gathered from a number of physicians and institutions that have compiled an impressive bank of credible and groundbreaking theories over the last seven years. Our effort here is to capitalize on those lifesaving protocols, merge the conventional levels of care with our overlapping SOF capabilities, and apply them in our rigorous environment.

OVERVIEW

The Tactical Combat Casualty Care Committee (TCCC) has continually updated guidelines since 1996 for prehospital care on the battlefield as defined in three levels: Care Under Fire, Tactical Field Care, and Tactical Evacuation Care. These guidelines are based on medic, corpsman, and physician input and experiences through quarterly conferences. However, TCCC guidelines only provide the basis for care at the point of injury through evacuation. This article maintains those guidelines, but will leave initial management behind as SF medics move on to 12 to 96 hours post event.

Damage control surgery (DCS) is a well established and proven modality of medical intervention in both civilian and military practice.⁵ The U.S. Army Institute of Surgical Research (ISR) has provided the most up-to-date collection, evaluation, and development of critical care for war wounded, and additionally has driven implementation of this theory within all the services. Damage control focuses on principles that allow for highly efficient care while compensating for inexperience and limited resources as the "great equalizer" of trauma surgery.⁶ Using the damage control model for a

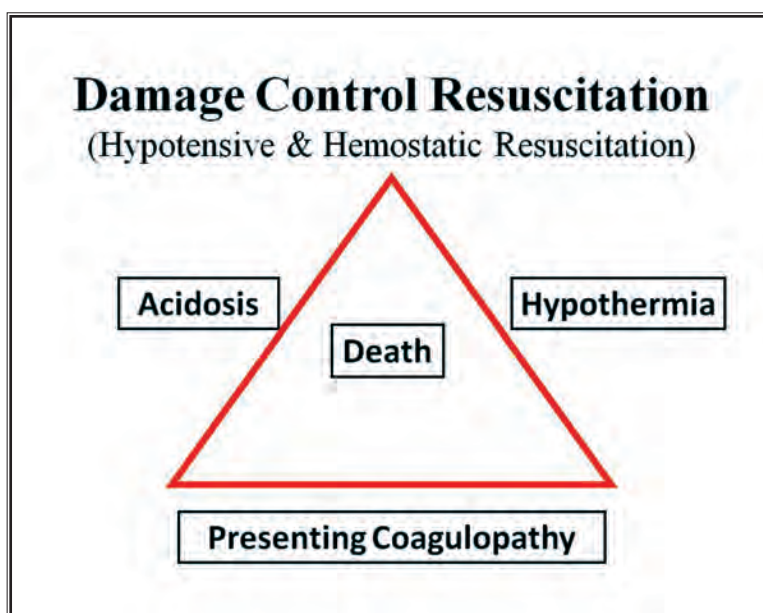


Figure 2 – The Lethal Triad easily visualized, attributed to Colonel John Holcomb.

ship, the goal is to rapidly implement measures that prevent further deterioration before irreversible injury occurs, or “the ship sinks.” Most initial treatments are temporary to minimize patient exposure to stressful surgical conditions and to reduce a physiological loss which maximizes the patient’s preparation for more extensive care. Definitive surgical repair of injuries prior to adequate resuscitation may lead to a fully repaired but unsalvageable patient. The primary and most immediate goal is surgical control of hemorrhage with judicious fluid resuscitation, which is accomplished with a number of advanced surgical procedures such as rapid closure, shunting, or packing. Stopping further contamination through exploration and additional therapeutic serves as a concurrent effort and significantly decreases septic effects that can impact mortality over time. The patient then moves to the intensive care unit to receive resuscitative care preparing him for return to the operating room within 24 to 48 hours for definitive surgical repair. Understanding this entire process is paramount to “act tactically, but think strategically”⁷ in preparing patients for a successful outcome. This treatment strategy must be understood to prepare the patient at this level; the SOF medics primary goal is to ensure that the patients arrive at surgical assets properly resuscitated.

Damage control resuscitation guidelines are specifically focused on the prevention of the “lethal triad” consisting of hypothermia, coagulopathy, and acidosis; all of which can be either mutually supporting or mutually destructive (see Figure 2). The factors of the lethal triad are all proven independent and codependent indicators of mortality which also apply to DCS. Damage control resuscitation guidelines also include aggressive hypotensive and hemostatic resuscitation while



Figure 3—An OSS doctor conducts minor surgery in China circa 1944. (Courtesy USASOC Historian’s Office)

providing parameters for addressing all three areas of the lethal triad. Ensuring that these efforts are proactive and continuous from the point of injury provides the most efficient care possible and uses a more scientific and therapeutic approach to combat trauma for SOF medics. Again, the medics care can and should potentiate success in supporting both TCCC and DCS in the hospital.

IMPORTANCE OF HEMOSTASIS

The single most essential weapon for DCR is immediate and effective hemostasis, and it is at the point of injury where resuscitation *begins* for the SOF medic. Hemorrhage control is the conservation of every single drop of blood and with it every key ingredient that provides success against the lethal triad. The loss of blood leads to hypoperfusion of tissues, relative hypoxia, and promotes anaerobic metabolism. This subsequently promotes acidosis, hypothermia, and loses key coagulation factors that are not easily reclaimed. Minimizing blood loss by immediate and effective treatments is a fundamental trauma skill. Perfecting the basics will gain hemorrhage control in the least amount of time and with minimal supplies while increasing survivability with DCR.

The physiologic picture resulting from hemorrhage easily demonstrates the interacting and accumulating factors that will be important later. Blood loss not only includes red blood cells essential for tissue oxygenation but also critical coagulation components such as platelets, clotting factors, and enzymes. Currently these factors can only be replenished in the most difficult procedures for the SF medic, especially when time, enemy situation, and supplies may all be at odds. A loss of blood volume reduces total oxygen carrying capability, which is compensated by increases in both inotropic (contractility) and chronotropic (heart rate) effort until the mismatch in oxygen delivery and demand result in tissue hypoxia, or true shock. At this point, the affected tissues convert from aerobic to anaerobic metabolism, which exacerbates all three components of the lethal triad. Cellular hypoxia results in a 90% reduction in energy production and an increased rate of lactate production promoting metabolic acidosis. This action leads to cellular swelling and edema, which further diminishes capillary flow and microcirculation irrespective of mean arterial pressure (MAP). Additional hypoperfusion due to vasoconstriction occurs naturally and simultaneously by lowered blood pressure, pain, and cortical recognition of injury. A lack of blood supply to the liver results in decreased



Figure 4 – A Special Forces Medic conducts an I & D procedure in Bolivia in 1967.
(Courtesy USASOC Historian's Office)

glucose and clotting factors further complicating coagulopathies. Other physiological damage occurs when pro-inflammatory mediators are released due to hemorrhage and tissue damage, and shock affects neuroendocrine responses producing severe metabolic changes.⁸

Direct pressure is always the first step for hemostasis. As soon as hemorrhage is noted, digital or manual pressure is paramount and almost always assures immediate effectiveness. Remember, the goal is not just to limit the amount of blood loss, but to save every single drop possible. Paramount towards this end is the expectation that each Soldier, if able, performs self-care. This requires mental preparation, muscle memory, and psychological hardening to perform under physical pain, stress, and challenging conditions. Pressure points are next, or act as an adjunct to minimize blood loss and always attempt to use other Soldiers to deal with pressure points even under the best circumstances. The benefit is reduced time to hemostasis and preserved blood volume, while maintaining combat power during the fight. Other essential multipliers include the medic placing pressure with his own knee while he works, or effective support from his teammates from prior cross-training or on-scene instruction. Tourniquets are extremely effective in the treatment of

extremity wounds and their success since 9/11 is inarguable. There have been no reports of amputations during the conflict directly attributable to tourniquet usage. Remember that bleeding control is a graded response, so if a limb is mangled enough a tourniquet will likely be the first step in hemorrhage control.⁹ Tourniquet effectiveness is based on the principles of ensuring they are placed proximal to the wound, active bleeding stops, the distal pulse is absent, and that reassessment is frequent and continuous. Keep in mind that the duration a tourniquet is applied will bring new concerns in prolonged care. Present standards call for removal within two hours and, if conscious, the patient will remind the medic of this with the pain that normally accompanies prolonged tourniquet use. Application over two hours can also predispose the patient to increased morbidity such as fasciotomies and amputations, all of which may later be the medic's responsibility in this scenario.¹⁰ Converting a tourniquet to an effective pressure dressing as soon as possible while leaving the tourniquet loose and in place, for use if reapplication is necessary, will likely prevent issues later in pro-

longed care.

Packing wounds is a science in itself, requiring effective technique, proper supplies, and completed with a pressure dressing to optimize the medic's work. Making the decision to pack early is important too; packing is dependent on the patient's ability to form good clots and if too many factors are lost, then packing will not be effective. Bowl-type wounds must be addressed immediately by packing with a maximum of two fingers using unrolled Kerlix® and working from the bottom of the wound up, left to right or circumferentially, as if filling a bucket. Finding and addressing all potential space in the wound to ensure that there is no opportunity for any leakage of blood is a difficult task, especially while packing blindly, in the dark, and under stress at speed. An effective packing job can provide hemostasis with a minimum amount of supplies. Packing should not only be reserved for bowl-type wounds but also used in anatomical girdle areas such as the groin or shoulder. Hemostatic agents provide additional tools for more difficult wounds but they require thorough training, ideally during predeployment trauma training, to utilize effectively. The same rule applies for hemostatics as with packing: hemostatics + packing + pressure = success.

Future technologies that are presently being developed for advanced hemostatics such as vessel closure and pressurized viscotic hemostatics may offer additional adjuncts in time.

PREVENTION OF HYPOTHERMIA

Within the lethal triad itself, the prevention of hypothermia is probably the simplest and most practiced effort for SOF medics. Hypothermia has significant effects and yields 100% mortality to severely traumatized patients with core temperatures less than 90°F (32°C).¹¹ The goal is to maintain the casualty's core temperature to greater than 95°F (35°C). Preventing hypothermia takes far less effort and time than attempting to treat it under combat conditions.

Temperature monitoring should be as continuous as possible. Use every tool in sequence from skin color and extremity warmth, patient feedback, and mentation. Objective findings such as inexpensive temperature dots placed on the forehead or intermittent temperatures taken with an oto-thermometer, or use a digital rectal thermometer for continuous and high confidence readings. The fact that most wounded patients very often feel cold post-insult or the observance of spontaneous shivering, should always key the medic in to the above steps. In short, simply treat every single patient relentlessly for hypothermia.

Hypothermia prevention should start immediately post-injury. Consideration of heat loss goes hand-in-hand with the initial assessment and optimally should occur during the primary survey or just immediately thereafter. Most of this work can be accomplished by cross-trained teammates automatically and simultaneously as the medic treats. Plan, prepare, and practice hypothermia prevention during all aspects of training; immediately insulating patient contact with the ground, minimizing exposure during the primary exam, removing only wet clothing, and even keeping the patient clean are all essential principles decreasing heat loss. Use every opportunity to get the patient off the ground, dried if possible, covered, and out of the elements and begin all proactive efforts for economy of time.

Both passive and active measures should be planned for. Standard commercial hypothermia kits should include a durable and effective solar blanket and a chemical warming blanket, and

these should be kept with litter kits. Open the warming blankets first as they normally take some time to reach its full exothermic reaction. The solar blankets are normally vacuum sealed so it should be stretched to full size to open any incorporated air cells. Position it diagonally on the litter so that the head and feet lie on the longest ends of the blanket and move the casualty to the blanket as quickly as possible to get him off the ground and negate conduction. The patient should then be 'burrito' wrapped with the blanket as tightly as possible; it is the air closest to the patient, or within the air cells of the blanket, that provides the insulation for heat retention. Trapped air between the patient and the blanket is warmed by the body and then retained and protected from loss or change by the blanket. If standard hypothermia kits are not available, wool or space blankets wrapped in the same manner and with some kind of head insulation (up to 60% heat loss here) such as a wool skull cap will provide much of the same effects.

Active warming measures require prior planning and usually cannot be achieved through improvisation. The chemical warming blanket opened first should be laid between the patient and the solar blanket to provide some degree of active heat on all patients. Be aware that there are differences in products and manufacturers so always rehearse this procedure. There are many different types of commercial kits and each has varying temperatures, durations, and effectiveness. Warming all fluids before giving should be



Figure 5 – A Detachment 101 surgeon works on wounded in an austere environment during World War II. (Courtesy USASOC Historian's Office)

the goal no matter what type, route, or environment. IV warmers such as the enFlow® warming system or the Thermal Angel® have proven effective, but both have specifics that need to be appreciated in the tactical environment. A significant amount of heat can be lost through the administration line. Using a closed-system kept next to the patient with minor infusion pressure, or protecting the administration line with insulation and minimizing its exposure to the elements, is important. Although the lack of a fluid warmer does not preclude establishing IV/IO access or administering appropriate fluids, withholding warm fluids is of detriment to the patient.

Primitive warming in an austere environment can be simple and is limited only by the medic's imagination. Using vegetation to insulate patients from the ground, finding civilian blankets from a house, simple shielding or overhead cover from the elements, heat bottles and active heat sources, and proper nutrition would all be beneficial. Primitive means to warming fluids such as using MRE heaters packaged in plastic bags are effective as well, but must be rehearsed and learned. Body bags, although not without possible psychological effect, are extremely effective in heat retention and protection and have been used in great success in Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF).

Expedited evacuation has become the norm in OIF, but exists as a double edged sword. Timely evacuation mitigates the environmental effects of prolonged exposure. Conversely, the customary rapid rotary wing evacuation may contribute significantly to converting a patient from normothermic to profoundly hypothermic — remember not to negate the benefits of a speedy transportation with an ill-protected patient. Timely air evacuation by itself is not the solution as a 2004 study from OIF found that 91% of all casualties that arrived to the CSH by air were hypothermic as exposure and convection from wind flow took its toll.¹² Optimization of these critically ill patients during transport is an important strategy to increase survival on the battlefield, replacing hope with capability.¹³

Hypothermia exacerbates coagulopathies primarily by negating the full potential of the clotting process itself due to a loss of homeostasis. If the body functions optimally at 98.7°F then anything less decreases its physiological response to a severe insult, thereby compounding the issues overall. Coagulation function in total is decreased by 10% with every degree point between 34°C and 32°C itself, and also independently affects specific clotting factors at different levels. Mortality is also significantly increased; a

trauma patient with a core temperature below 90°F (32°C) is associated with near 100% mortality.¹⁴⁻¹⁶ Hypothermia additionally delays the onset of thrombin generation, thrombin function, and additional cofactors required in the clotting cascade. Concurrently, platelet adhesion, interaction, and aggregation are debilitated outside of normal temperatures and all of the enzymes required in the clotting cascade are also suppressed.

MANAGING ACIDOSIS

The prevention of acidosis is the final component of the lethal triad which must be considered in managing patients for prolonged periods. Acidosis develops primarily due to cells being forced into anaerobic pathways for energy, and the degree of acidosis in the blood markedly underestimates the degree of intracellular acidosis. As cells die, they release an increasing amount of lactic acid into the system. As such, marked decreases in systemic pH may be a premonitory event, and mark irreversible uncompensated shock. Even more challenging is the fact that if perfusion is restored to previously hypoxic cells, accumulated acids will be released into the systemic circulation leading to a reperfusion syndrome. Therefore, combat casualties with any degree of hemorrhage should be considered at risk for developing acidosis, especially when the short-term care and rapid evacuation become a long-term resuscitation challenge due to a changing battlefield. The ability to maintain adequate breathing and ventilation, achieving the appropriate goals for endpoints, and again rapid evacuation, mutually supports all other efforts.

Small changes in pH have more profound effects on coagulation than even small changes in core temperature. The coagulation proteases necessary for the activation of both the intrinsic and extrinsic pathways are optimized to function in the alkaline range. A decrease in pH from 7.4 to 7.0 reduces the activity of the enzyme complex that activates thrombin by 70%.¹⁷ The previous point leads to the possibility that active correction of pH and administration of thrombin may be areas of future research in hemorrhagic trauma.

Hypothermia and acidosis provides a clear example of how the three factors in the lethal triad are interrelated: Acute hemorrhage leads to hypotension with decreased systolic blood pressures, which in turn lead to reduced oxygen perfusion to tissue. Decreased O₂ delivery results in a change in energy consumption from aerobic to anaerobic metabolism, which then produces lactic acid as a byproduct, which contributes to overall acidosis. Anaerobic metabolism itself is inefficient and decreases normal energy production and

heat generation which then negates the body's ability to recover from temperature loss. The resulting acidosis interferes with the clotting cascade, and leads to further hemorrhage during resuscitation, leading to death if not prevented or aggressively interrupted.

CORRECTING COAGULAPATHIES

Addressing presenting coagulopathies is the primary focus of DCR seconding acidosis and hypothermia in priority. As mentioned before, minimizing blood loss from the very onset with hemorrhage control keeps RBCs and clotting factors onboard, and assists in maintaining a natural acid base balance for as long as possible. Hemostatic dressings and vascular access provide additional lifesaving capabilities with increasingly effective products on the market, and advanced techniques developed to address non-compressible hemorrhages. The early use of recombinant Factor rVIIa in severe blood loss and coagulopathy can be another choice as a treatment adjunct. Rapid evacuation to the next level of care again provides access to surgical interventions and blood banking to interrupt the progress of the shock. If rapid transport is unavailable, we must consider other approaches to maintaining the casualty, and interventions of a more creative nature.

Additionally, it appears that a subset of trauma patients present coagulopathic not secondarily as a result of blood loss, acidosis, and hypothermia, but as a *primary* response to the traumatic insult.¹⁸ This group of patients become coagulopathic much earlier for reasons that are at present not well delineated. That fact, coupled with the hemorrhage due to penetrating trauma may lead to a disproportionate loss of clotting factors, inactivation of normal coagulation due to acidosis and hypothermia, and inactivity of factors due to storage in banked blood and blood components. Consequently, in non-compressible hemorrhage on the battlefield, in an otherwise young and healthy population, there may be a role for early augmentation of the coagulation cascade. As previously noted, the proteolytic enzymes of the coagulation cascade function poorly at pH below 7.2. Acidosis slows the rate of thrombin generation, while hypothermia delays the onset of thrombin generation. Biologically plausible arguments can be made for optimizing and augmenting the coagulation pathways and processes *before* significant hemorrhage occurs and shock develops as will be discussed.

While we have for the most part solved the challenge of extremity injury with the use of tourniquets, pressure, and wound packing, the developments of new types of hemostatic agents in the form of a pad,

packing sponge, or gel have provided adjuncts for compressible and accessible wounds. The current challenge in the field for the medic is the non-compressible and non-accessible wounds, primarily in the abdomen and pelvis in the absence of immediate surgical assets. Because of this, the use of non-surgical solutions needs to be aggressively considered. This would include the use of Factor VIIa, along with adjuncts such as calcium and sodium bicarbonate to optimize the biological activity of this potentially lifesaving modality. Factor VIIa works by activating through thrombin and various feedback loops both the intrinsic and extrinsic coagulation pathways. The desired endpoint is assistance in the formation of a thrombus at the site of injury in an otherwise inaccessible location such as solid organs (liver, spleen, and kidney), large vessels, and pelvic structures. This might be thought of as mobilizing a damage control party to the site of injury in our ship analogy. Because of the time involved, this must be considered for administration relatively early if severe hemorrhage is suspected. Delays awaiting conclusive evidence of Class III/IV shock may prove insurmountable. Interestingly, there is some evidence that thrombus created with rVIIa administration may be more resistant to lysis and breakdown with reperfusion.

Correcting coagulopathies will also be assisted by administering the proper blood products, and although FWB and FFP are specifically advocated in this article due to their natural contributions, they will be addressed in Part Two complete with all other fluid choices. In addition to resuscitation, prolonged care requires an exceptional skill set. Tracheotomies, fasciotomies, blood transfusions, anesthesia, and primary and delayed closure, all play an important role here. These are advanced skills and as such require proper sustainment. Nursing skills also have a huge importance in critical care and without them none of the above would be successful. Competence in aseptic technique, antibiotic therapy, labs for the monitoring of endpoints, input and output, and basic nursing care including patient hygiene need to be appreciated. Other concerns requiring attention are pre and post surgical skills, wound care, nutrition, rehabilitation, and even logistical needs. This skill set is now rarely exercised, except in the schoolhouse during records and reports, but is essential in the austere scenario.

The authors hope that Part One provides a foundation in general knowledge of DCR for the SF medic; Part Two will identify options for measuring shock and recommend relative endpoints to serve as goals in resuscitation. Additionally we will propose

not only the special equipment required, but the minimal equipment needed in care, and identify the critical care and nursing skills required to support DCR in our environment.

REFERENCES

1. BG Yeheskel Levy. (2007). Lecture. *Advanced Technology Applications for Combat Casualty Care Conference*.
2. Holcomb J. (2007). Damage Control Surgery. *Journal of Trauma*; 62:S36-S37.
3. Blackbourne L, McMullin N, Eastridge B, Baskin T, Holcomb J. (2007). Aggressive Proactive Combat Damage Control Surgery. *AMEDD Journal*; January-March 2007.
4. Kaplan L, Bailey H. (2007). Ongoing Resuscitation Endpoints and Strategies; *Trauma;Emergency Resuscitation Vol 1 2007*. Inpharma Healthcare.
5. Blackbourne L. (2008). Combat Damage Control Surgery. *Critical Care Medicine*; 36,7:S304-S310.
6. Hirshberg A, Mattox K (2006). Top Knife, *The 3-D Trauma Surgeon*, tfm Publishing Ltd, p. 5-17.
7. Ted Westmoreland. (2006). Attributed quote.
8. Anjaria D, Mohr A, Deitch D. (2008). Haemorrhagic shock therapy. Informa Healthcare.
9. Hirshberg A, Mattox K (2006). Top Knife. *Stop That Bleeding*, tfm Publishing Ltd, p. 19-34.
10. Kragh J, Walters T, Baer D, et al. (2008). Practical Use of Emergency Tourniquets to Stop Bleeding in Major Limb Trauma. *Journal of Trauma*; 2008:64:S38-S50.
11. Beekley A. (2008). Damage control resuscitation: A sensible approach to the exsanguinating surgical patient. *Critical Care Medicine*; 2008:36:S267-S274.
12. Arthurs Z, Cuadrado D, Beekley A, et al. (2006). The impact of hypothermia on trauma care at the 31st combat support hospital. *The American Journal of Surgery*; 191:610-614.
13. Blackbourne L, Gratwohl K, et al. (2007). Optimizing Transport of Postoperative Damage Control Patients in the Combat Zone. *AMEDD Journal*; January-March 2007.
14. Krishna G, Sleigh J, Rahman H. (1998). Physiological predictors of death in exsanguinating trauma patients undergoing conventional trauma surgery. *Aust NZ Surg*; 68:826-829.
15. Jurkovich G, Greiser W, et al. (1987). Hypothermia in trauma victims; an ominous predictor of survival. *Journal of Trauma*; 1019-24.
16. Pend R, Bongard F. (1999). Hypothermia in trauma patients. *J Am Coll Surg*; 188:685-96.
17. Hoffman M. (2004). The Cellular Basis of Traumatic Bleeding. *Military Medicine*; 169,12:000.
18. MacCleod J. (2008). Trauma and Coagulopathy; A New Paradigm to Consider. *Arch of Surgery*; 143(8):797-801.

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