Review of Damage Control Laparotomy (DCL) Outcomes in a Major Urban Trauma Centre

by

Dr Andries Michiel Kruger

KRGAND010

SUBMITTED TO THE UNIVERSITY OF CAPE TOWN

In fulfilment of the requirements for the degree

Masters of Medicine in Surgery

Faculty of Health Sciences

University of Cape Town

July 2019

Supervisor: Professor Pradeep Navsaria

University of Cape Town

Department of Surgery

Table of Contents

DECLARATION

I, Andries Kruger, hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

I empower the university to reproduce for the purpose of research either the whole or any portion of the contents in any manner whatsoever.

Signature: Signed by candidate

Date: 18 June 2019

ACKNOWLEDGEMENTS

I would like to express my sincerest gratitude to Professor Pradeep Navsaria who supported me through the process. His guidance and motivation helped in completing this manuscript.

My gratitude extends also to the individuals who tirelessly went through countless patient folders to capture the data. Thank you to Dr Juan Klopper who helped interpret the data and provided the statistics.

List of figures and tables

Literature review

Manuscript

ABBREVIATIONS

DCL – Damage control laparotomy

DCS – Damage control surgery

DCR – Damage control resuscitation

DC – Damage control

DL – Definitive laparotomy

ACS – Abdominal compartment syndrome

IAH – Intra-abdominal hypertension

TXA – Tranexamic acid

RT – Resuscitative thoracotomy

TIVS – Temporary intravascular shunt

TAC – Temporary abdominal closure

ARDS – Acute respiratory distress syndrome

TEG – Thromboelastogram

ER – Emergency room

ABSTRACT

REVIEW OF DAMAGE CONTROL LAPAROTOMY OUTCOMES IN MAJOR URBAN TRAUMA CENTRE

Kruger AM

Introduction

Damage control laparotomy (DCL) in an urban trauma centre is associated with high mortality.

Aim

The purpose of this prospective study was to review the outcomes of DCL in a level one urban trauma centre, looking particularly at primary closure rate and other factors influencing outcomes.

Methods

All patients undergoing DCL for penetrating trauma from May 2015 to July 2017 were retrieved from the prospectively recorded eTHR data base. Data retrieved were basic demographics, mechanism of injury, perioperative vitals and biochemical parameters. Injury severity was described by the Revised Trauma Score (RTS), Penetrating Abdominal Trauma Index (PATI), Injury Severity Score (ISS) and Trauma and Injury Severity Score (TRISS). Indications for DCL were determined as well as length of ICU stay, days of ventilation, number of procedures and primary abdominal closure rates. Complications and mortality were recorded.

Results

During the study period, 51 patients underwent DCL. Three patients sustained stab wounds and 47 patients suffered from gunshots. Only 1 female was included in the study with the other 50 being male. The mean age was 28 years and 4 months (range 15 to 48 years). Indications for laparotomy were haemodynamic instability (n = 27) and peritonism in stable patients (n = 22). The means for the different severity scores were RTS 7.36, ISS 17.5, TRISS 93.76 and PATI 28. Means were calculated for different physiological markers of trauma (lowest pH 7.12, highest lactate 7.11, lowest core temp 34.9˚C and lowest systolic BP 63.8 mmHg). The organs most commonly injured, in decreasing frequency, were small bowel (n = 33), large bowel (n = 25), abdominal vasculature (n = 22), liver (n = 18), stomach (n = 14), kidney (n = 10), diaphragm (n = 10), spleen (n = 9) and pancreas (n = 8). DCL procedures performed were abdominal packing (n = 36), bowel ligation (n = 30), vascular shunting (n = 5) and shunting of the ureter (n = 1). The median number of laparotomies done per patient was 3, with a primary fascial closure rate of 69%. The mortality rate was 29%.

Conclusion

DCL in our setting is associated with a 29% mortality rate. Severe acidosis, massive blood transfusion in first 24hours and median PATI score more than 47 are independent factors associated with increased mortality.

LITERATURE REVIEW

Review of Damage Control Laparotomy (DCL) Outcomes in a Major Urban Trauma Centre

Background

Stone et al[1] while attempting to address the problem of major coagulopathy during laparotomy, showed a significant survival benefit in performing an "abbreviated laparotomy", with deliberate delayed repair, versus definitive repair of complex injuries at the initial, index operation. He ascribed this benefit to giving the patient adequate chance to reverse the coagulopathy in a controlled ICU setting, before returning to the operating room with a patient that is more physiologically stable from both a metabolic and haemostatic perspective. A decade later, Rotondo et al[2] coined the term "Damage Control" surgery and found that in patients with major intra-abdominal vascular injury, and two or more visceral injuries, survival was markedly improved with damage control (DC) maneuvers, when compared to a group that underwent definitive laparotomy (DL): survival of 77% DC vs 11% DL.

The origins of damage control surgery date back to the early 1900's. The preferred management of a bleeding liver injury was simple hepatorrhaphy or more complex resections with ligation of individual vessels. Pringle[3] in 1902 initially applied this early concept in the form of liver packing for uncontrollable bleeding. Initially, gauze packs were inserted into deep lacerations within the liver parenchyma and then sutured over. These packs were then removed at the bedside days later[4]. Problems that arose with this technique were, adherence of the gauze to the liver parenchyma,

causing discomfort and potential rebleeding on removal. Furthermore, in addition to these complications, abscess formation, hepatic necrosis and further sepsis meant that by the end of World War II, the use of ***intra-hepatic*** packing was largely abandoned[5].

Perihepatic packing was introduced in the 1970's when Lucas and Ledgerwood[6] suggested its use for temporary haemorrhage control which would otherwise require hepatectomy, to quote, "when the surgeon is not mentally or technically qualified to perform such a procedure". Once the principle of coagulopathic bleeding was introduced, the indications for perihepatic packing grew. The concept of the "lethal triad" or "bloody vicious cycle" was introduced by the Denver General Hospital group[7]. They observed that exsanguinating haemorrhage was often associated with hypothermia, acidosis and coagulopathy (*Figure 1*). Thus, the true bearing of damage control surgery largely arose following the discovery of the lethal triad. The goal of DCS was to avoid the initiation, terminate or reverse the "bloody vicious cycle". Furthermore, Stone[1] showed an increased survival benefit when the operative time was significantly reduced. He introduced bowel and ureter ligation, and en bloc removal of the injured kidney and spleen as additional components of damage control surgery. He showed an increased survival rate of 65% (11 of 17) when compared to a historical survival rate of 7% (1of 14) among similarly injured patients managed with definitive surgery at the index operation. The staged or abbreviated laparotomy was now accepted as a potentially lifesaving procedure where other methods of haemostasis had failed.

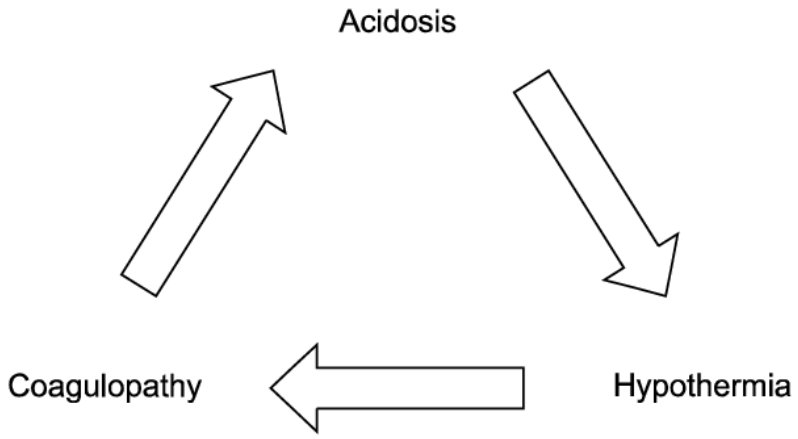

Figure 1

When Rotondo[2,8] coined the phrase “Damage Control”, these principles were fully accepted into mainstream trauma care. He went further to define the 3 stages of damage control, namely DC 1 (index laparotomy employing damage control principles), DC 2 (ICU admission with further hemostatic resuscitation) and DC 3 (definitive surgical repair). Later DC 0 (prehospital and front room resuscitation before surgery) and DC 4 (definitive abdominal closure) were added[9] (*Table 1*).

Stages of Damage Control	
DC 0	prehospital and front room resuscitation before surgery
DC 1	initial laparotomy using damage control principles with temporary abdominal closure and tranfser to ICU
DC 2	further resuscitation with the goal of correcting metabolic and hemodynamic abnormalities
DC 3	definitive surgical repair of injuries with closure of abdomen
DC 4	definitive abdominal closure / repair of ventral hernia

Table 1

By the turn of the millenium, there were reports of some 1000 patients managed by DCL, with a survival rate of 50%[10]. With this growing body of evidence and well-defined stages, the implementation of DCS was now a well-accepted management strategy for treating exsanguinating patients with abdominal injuries. This gave way for development of multiple novel, unorthodox surgical manoeuvres for rapid,

abbreviated management of injuries to ***arrest haemorrhage, limit hollow viscus contamination*** and ***reduce operative time*** (less than 60 minutes) to arrest the bloody vicious cycle of acidosis, hypothermia and coagulopathy.

Abdominal compartment syndrome (ACS) after major abdominal surgery was first reported in 1983 by Richards[11]. The understanding was that raised intra-abdominal pressure was caused by supranormal trauma resuscitation with crystalloids (which was the teaching at that time), ongoing intra-abdominal bleeding, intra-abdominal packing and the non-compliant abdominal wall[12]. Closure of the abdominal wall and skin, which was common practice at the time, would only increase the prevalence of intra-abdominal hypertension (IAH) and its complications. To prevent ACS, studies showed the use of early prophylactic decompression as a treatment modality to reverse the complications of IAH[13]. He noted the improvement in renal function in 4 patients after evacuation of large postoperative intra-abdominal hematomas. This was further described in a large number of patients following DCL.

The management of an open abdomen still provides massive problems for the patient and the surgeon today. Correct management of the open cavity from DCS to relook is crucial if adequate resuscitation and reversal of the “lethal triad” is to be achieved. Heat and fluid loss, as well as worsening sepsis are all possible early complications of an open abdomen. Early methods for temporary abdominal closure included the use of towel-clip closure, a method complicated somewhat by bowel evisceration due to high intra-abdominal pressure and ACS. Bridging silos were developed, most notably the “Bogota bag” which gained popularity due to its cheap and easy to use nature. Development of other techniques was commercially driven. These include synthetic meshes, zippers and slide fasteners. The abdominal vacuum dressing revolutionised the management of the open abdomen. Commercial

packs have made it much easier to provide a safe and easily replaceable cover with complicated patients requiring repeated surgery. These techniques provide adequate cover and protection until definitive fascial closure is achieved. The longer the delay to definitive closure, the less the likelihood of primary fascial closure. This is mainly due to retraction of the abdominal wall muscles and fascia, as well as visceral oedema. With failed closure, special care needs to be taken to protect the exposed abdominal viscera. A host of commercially available devices can be used to provide coverage of bowel and abdominal content in the ICU and hospital setting while the patient recovers from his injuries. After about 10-15 days, dense adhesions between bowel loops and the abdominal wall creates a layer of granulation tissue onto which a split skin graft can be applied once the patient has stabilised. Repair and reconstruction of the consequent ventral hernia is attempted after a year [14].

Despite the confirmed effectiveness of DCS in managing critically ill trauma patients, it remains associated with potentially life-threatening complications. Intra-abdominal abscesses and sepsis, massive ventral hernias, and entero-atmospheric fistulas are well documented complications. Occurrence of any of these complications results in multiple and extended admissions with subsequent surgical procedures [15]. It is with this in mind that the widespread use of DCS has now been scrutinised. The indications for its use have become the focus of many studies and review articles [16]. The trend is now to attempt to reduce the need for DCS by implementing revised resuscitation methods. These damage control resuscitation (DCR) methods look to reduce crystalloid fluid use by replacing blood loss with blood and blood products. The aim is to reverse acute traumatic coagulopathy and acidosis, and potentially reduce the need for DCS in the first place. DCR and the role of DCS surgery remain keenly investigated.

Much of the data supporting DCS comes from the military fraternity. The earliest documented use of military damage control surgery is in 2000 when Mabry[17] looked at the management of US soldier casualties, sustained in Mogadishu, Somalia. There was initial scepticism about applying DCS in battle situations, as logistically applying a concept requiring multiple surgeries and ICU care could prove impractical. Blackbourne further described this in 2008[18] where civilian DCS entails the 3 stage approach (at that time), “combat damage control surgery” could entail up to 10 stages from initial battleground evacuation, surgical operations and multiple resuscitations, to multinational clinician support and transcontinental transport. Even in 2013 the International Committee of the Red Cross cautioned the use of DCS in a resource scarce setting. In 2004 a paper by Neuhaus *et al* contrasted this scepticism by deducing that “philosophy of damage control is uniquely suited to the Australian military environment.” [19]. He did however place specific emphasis on evacuation capabilities incorporating critical care transport teams, as these patients require definitive treatment within 48hrs. Also, in 2004 DCS was incorporated into US and British military surgery doctrine. A 2015 prospective observational study by Smith *et al* focused on a military trauma system in Afghanistan. It concluded damage control as the optimal approach to severe abdominal war injuries [20].

Indications for Damage Control

Patient selection for DCS is critical. Early recognition assures prompt aggressive resuscitation and speedy transfer to theatre. Delayed recognition will inevitably result in irreversible metabolic and physiological derangements with poor outcomes.[21] A high index of suspicion is required to fine tune identifiable factors. The danger of a too low threshold is overuse of DCS, thereby denying patients with adequate physiological reserve the opportunity to recover from a definitive repair of their

injuries. These patients are then exposed to repeated surgeries and the other possible complications associated with DCS.

There are a wide range of suggested indications found in the literature. An analysis by Roberts *et al*[22] narrowed down the more important indications. Many of these are clearly defined factors, but among these are indications that require insight and experience from the surgeon. Indications were most often based on physiological markers that serve to describe the extent of patient injury. The signs most commonly quoted are hypothermia (<34°C), acidosis (pH<7.2) and coagulopathy (PT/PTT >1.5 normal and/or failure to form clots with diffuse oozing).[22] A lactate of >4mmol/l is also a sign of significant acidosis, possibly warranting DCS. Although these are clearly defined cut offs (except maybe visible coagulopathy) they fall on a spectrum of physiologic homeostasis during trauma. This degree of physiologic derangement can occur at any time from injury scene to theatre, making it crucial to continuously monitor for these changes as resuscitation and surgery take place. Outcomes are improved if the decision to perform DCS is made before the lethal triad occurs.[22]

Another indication independent of physiological status is injury pattern. Major injuries to two different cavities of the body can indicate a patient that requires DCS. Specific patterns of injury in the abdomen itself also requires application of DCS principles, including difficult to access major venous injury, major pancreaticoduodenal injury and major liver injury with intraoperative instability or 2 or more associated solid and/or hollow viscous injuries.[23] In summary, DCS should be applied when two injuries have competing management priorities (where exsanguination is risked during attempted definitive repair), where injuries would better be treated with therapeutic packing or where pancreaticoduodenectomy is indicated.

An additional indication for DCS independent of physiological status is a patient that requires massive transfusion due to ongoing or massive haemorrhage. Massive transfusion protocols are implemented in many trauma centres, which can be used for rapid dispensing of blood and blood products when DCS is indicated. In centres where massive transfusion protocols aren't in place, a cut off of >10 units of packed red cells can be used to define a massive transfusion. The basis of this is the potential risk of dilutional coagulopathy and abdominal visceral oedema related to large volume fluid resuscitation. Also, it provides an indirect measure of the severity of the injuries as well as an obvious significant vascular injury.

An inability to close abdominal fascia under tension or development of ACS during closure, is also an indication for DCS.[24] Finally an anticipated lengthy (>60-90min) surgery in a severely injured patient with suboptimal response to resuscitation, is an indication for DCS.[22] This suggests that patients not presenting in extremis, who respond adequately to resuscitation may be candidates for definitive repair of their injuries, as long as they remain hemodynamically and metabolically stable without signs of coagulopathy.[22]

Overall an estimated 10% of major trauma patients will benefit from DCS, with no single factor predicting who these patients are. It is very important to note that an early decision to apply DCS when indicated is crucial to avoid poor outcome.[21]

Stages of Damage Control

Damage Control part 0 (DC 0)

This is the earliest phase of damage control, starting in the pre-hospital setting and continuing in the emergency department. This stage relies heavily on injury pattern recognition by the first response team. The emergency services are then required to cut on-scene time to a minimum in an effort to get the patient to the nearest trauma centre as soon as possible. The increasing presence of on-scene doctors in rapid response teams and air ambulances makes it possible for more advanced procedures to be done on scene, e.g. rapid sequence induction and intubation. Prehospital availability of blood products and tranexamic acid (TXA) is becoming more common practice.[21] The literature has shown support for the early administration of TXA. The CRASH-2 trial showed significant survival benefit in patients receiving TXA within the first hour after trauma ($p<0.0001$), also shown with administration between 1-3 hours after trauma ($p<0.03$). It did however show increased risk of death due to bleeding when administered after 3 hours.[25] A review article by Ausset *et al*[26] (including the CRASH-2 trial) showed overwhelming support for the use of early TXA administration in prehospital damage control resuscitation (DCR).[26]

A system needs to be in place whereby ambulances can proceed directly to major trauma centres, rather than the nearest hospital, based on certain injury patterns and physiological abnormalities. The referral of these patients needs to be communicated to the trauma centre, giving them time to prepare for all the practical and logistic implications of DC.[21]

Once in the trauma department, an emphasis is placed on confirming or identifying patients for DC. It then becomes imperative to prevent deterioration of the patient, doing only what is needed to get the patient to theatre as soon as possible and in the best condition. DCR has streamlined this approach based on patient physiology in trauma and correcting it in as few steps as possible.

Damage Control Resuscitation

Coagulopathy of trauma is a well-documented entity, described amongst others by Brohi *et al*[27] in 2003. He found that the incidence of coagulopathy increases as the injury severity increases (ISS >15 is associated with and incidence of coagulopathy of 24%). This “acute traumatic coagulopathy” is independent of crystalloid fluid resuscitation and is associated with poor outcomes. It has been identified as an endogenous coagulopathy in severely injured trauma patients. Using crystalloid fluid for aggressive fluid resuscitation as described in advanced trauma life support (ATLS) and prehospital trauma life support (PHTLS) guidelines, may worsen presenting coagulopathy and acidosis in trauma patients. This could increase the risk of acute respiratory distress syndrome (ARDS), systemic inflammatory response syndrome (SIRS) and multiple-organ dysfunction syndrome (MODS).[28] Other complications include massive abdominal viscera and wall oedema, increasing the likelihood of failed abdominal closure or the development of IAH/ACS. Holcomb *et al* also described using hypotensive resuscitation (systolic blood pressure about 90 mmHg) and blood and blood products as the main resuscitation fluid in an attempt to correct acidosis and coagulopathy in these severely injured patients.[28]

Hodgetts *et al*[29] went further to define DCR into distinct elements. The ultimate goal of DCR is to minimise blood loss and maximise tissue oxygenation in order to optimise outcome. These elements are:

- <C> ABC resuscitation (<C> being for control of catastrophic haemorrhage)
- Permissive hypotension
- Limiting crystalloid use with early use of blood and blood products
- Early use of TXA
- DC 1 (surgery)

A retrospective cohort study by Cotton *et al*[30] has shown benefit in DCR when compared to historic controls. It showed reduction in the use of crystalloid and blood products, as well as improved 30 day survival. Not only is the early use of blood important, DCR also focuses on the ratio of components used to directly address coagulopathy. A ratio of 1:1:1 (fresh frozen plasma : platelets : packed red cells) was suggested in the PROPPR trial in 2015[31]. It showed no significant survival difference when compared to 1:1:2 protocols. More patients in the 1:1:1 group achieved haemostasis but did show increased use of plasma and platelet transfusion.

More centres are using protocolised administration of blood products in an attempt to prevent delays in accessing appropriate blood products for the exsanguinating patient. These protocols tend to differ between trauma centres based on availability and individual need, but should all initially provide uncross-matched units for immediate use, followed by fully cross-matched units once specimens are received by the blood bank for analysis. Again, these massive transfusion protocols are aimed at addressing acute traumatic coagulopathy and coagulopathy associated with large fluid transfusion.[21]

The use of imaging in the DC setting depends entirely on how stable the patient is. By definition, a patient who is a candidate for DC will be severely injured and in all likelihood unstable, requiring aggressive resuscitation just to get the patient to theatre in a reasonable state. If immediately available in the resus bay, plain chest and/or abdominal x-rays or full body x-rays (LODOX) may prove valuable to identify haemo- or pheumothoraces which (when immediately addressed) can prove vital in the stabilisation of a polytrauma patient. This imaging can also show the presence and location of foreign bodies which could provide clues to the expected injury pattern. The use of CT imaging is rarely used in the true DC patient as delaying of immediate surgery could prove fatal. It can't be emphasised enough that the use of any imaging is dictated by the condition of the patient and should never take priority over the endpoint of DCR which is getting the patient to surgery in the best condition possible.

Resuscitative thoracotomy (RT) has limited use. It is a last-resort intervention in a patient who arrests due to suspected haemorrhagic shock from a chest or abdominal injury. Its main indication is for penetrating trauma to the torso, with little evidence showing its benefit in blunt trauma. RT provides rapid access to the chest, allowing for haemorrhage control of injured viscera (heart, lungs, major vessels). The injuries are then definitively addressed in the ensuing formal thoracotomy in theatre. An additional intervention possible with RT is aortic cross-clamping. This can be done for isolated abdominal trauma with exsanguination due to suspected abdominal aortic or large arterial injuries. This manoeuvre buys time for emergency laparotomy to address the abdominal injuries. Resuscitative endovascular balloon occlusion of

the aorta (REBOA) has recently become an alternative to RT with aortic cross clamping for non-compressible haemorrhage below the diaphragm. Many decision-making algorithms have been described to outline the indications and placement positions of the REBOA. The aorta is divided into three zones and depending on the area of suspected injury, the balloon is placed and positioned under screening (figure 2). Contraindications to its use are suspected thoracic aortic of large vessel injuries (hemothoraces, widened mediastinum on CXR). The algorithm below can be used to guide decision making (figure 3).[32]

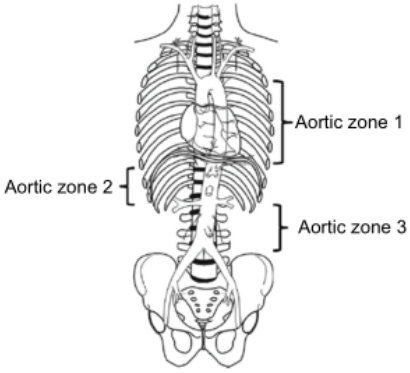

Figure 2

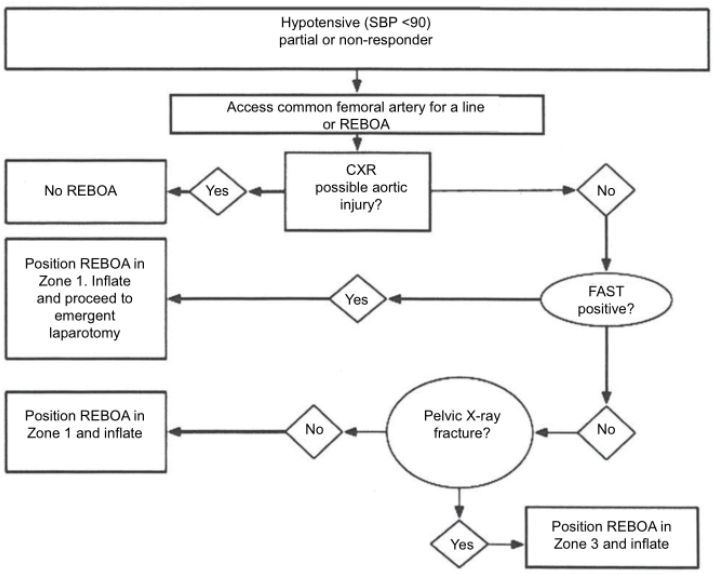

Figure 3

Damage Control part 1 (DC 1)

The basic principles for DCS are to control bleeding and contamination, abdominal packing, and temporary abdominal closure.[21] These need to be done as speedily as possible with the endpoint of restoring physiology, not anatomical reconstruction. It is important to realise that DCR doesn't stop when the patient leaves the resuscitation unit. It remains an ongoing part of DC 1 and DC 2.

DC 1 starts before the patient reaches the theatre. Early and accurate communication to alert the theatre staff and anaesthetic team is very important to avoid any delays with patient transfer and starting time in theatre. Additional equipment like fluid warmers, cell salvage suction, vascular and chest sets (sternotomy saw) need to be immediately available. Anaesthetic staff also needs to ready additional monitoring lines (arterial and central) that can be placed without delaying surgery.

The patient is positioned supine with arms abducted at right angles. Make sure the chest and abdomen are kept clear of ECG leads and monitoring. Urinary catheters and nasogastric tubes are placed if not done already. The patient is cleaned from chin to the mid-thighs in anticipation of any chest, abdomen or groin exposure if needed. Placement of lines for further resuscitation and monitoring should not delay surgery. In some circumstances, induction of the patient can be delayed until the surgeon is ready to cut. This is in anticipation of the potential hypotensive effect of the induction agents.

Surgery

A midline incision remains the best incision for exposure of abdominal injuries. The incision should start at the xiphisternum and end at the pubic symphysis. The exception is where there is suspicion of pelvic fracture with haematoma. Here the incision can be stopped just below the umbilicus so not to disturb the retroperitoneal haematoma.[21,33] Once through the peritoneum, the first aim should be to gain temporary haemostasis. The mechanism and area of impact (or trajectory of projectile) will give a clue to where the likely source could be. The initial step is to eviscerate the bowel and empty the abdominal cavity of fresh blood and blood clots.[33] Again, the mechanism of injury is important, with blunt injury causing haemorrhage mostly from solid organs whereas penetrating injury can also cause true vascular injury. The abdomen is subsequently packed with abdominal swabs and systematically inspected to find the source of the bleeding.[34]

Haemorrhage control

Nicol *et al*[35] lists various techniques for haemorrhage control, i.e. suture, ligation, temporary intravascular shunts (TIVS) and packing. Vascular damage control has traditionally been limited to ligation of the bleeding vessel. Introduction of TIVS and balloon catheter tamponade have shown promising results.

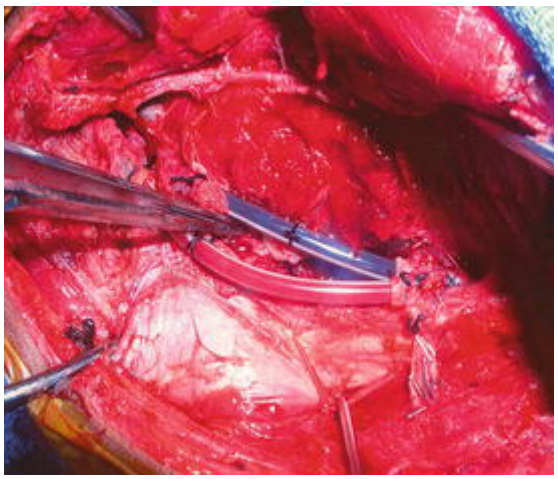

Figure 4 Temporary intravascular shunt (TIVS)

Ball *et al*[34] describes the use of balloon catheter tamponade for inaccessible major vascular injuries, large cardiac injuries, and deep solid organ parenchymal haemorrhage (liver and lungs). Multiple studies from as far back as the 1950's describe the use of occlusive balloon catheters in the management of cardiovascular injuries. Its original use was in controlling bleeding oesophageal varices.[36] Its use has been repeatedly described by percutaneous placement in many areas of the body, into the bleeding tract for haemorrhage control.[37,38] It is however with its use in liver, pelvic and abdominal vascular injuries that this review finds its interest. In 2011 Ball *et al*[39] identified two broad patient groups where balloon catheters are indicated, namely placement for temporary control followed by immediate repair of the injury, and placement into a bleeding site where access is difficult, or repair should not be attempted. He showed very good results with tamponade of liver bleeding (83% successful tamponade) and 67% survival in patients with liver injuries. It was used as a last resort when more conventional methods of haemostasis failed, specifically helpful with deep, central liver gunshot tracts. The catheters were kept in situ for a mean of 53 hours. Additionally, a majority of the patients had diagnostic and therapeutic angiographic interventions, a practice he encourages. When used to arrest iliac/femoral vessel bleeding, immediate repair or shunting was indicated of

the injured vessel. The accepted indications for catheter tamponade are: inaccessible major vascular injuries, large cardiac injuries and deep solid organ parenchymal injuries.[39]

TIVS are synthetic conduits used to temporarily bypass a vascular injury in a patient who has deranged physiology. The definitive repair is done at the next operation. TIVS addresses the initial haemorrhage as well as maintaining perfusion to the distal organs or limbs.[34] Much of the initial research comes from the military use of glass and then more flexible plastic conduits during war time injuries. The majority of TIVS (63% in the US National Data Base) is used following blunt extremity injury with extensive orthopaedic and/or soft tissue injuries.[40] Besides this, other indications are for damage control of peripheral and truncal vascular injury and for temporary stabilisation before transport. In a 2008 review by Ding *et al*[41], he concluded that these shunts can remain in place for prolonged times (12-48hours, longest reported 10 days) without the use of systemic anticoagulation (proximal and distal Heparin infusion at time of insertion could prove beneficial). The duration of patency was also increased by re-establishing venous outflow of the affected limb as well as technical factors like not looping the shunt and appropriate shunt diameter. A 2016 multicentre review also concluded TIVS as a viable option in damage control vascular surgery.[42] They reported a remarkable 96% limb salvage rate with TIVS. They confirmed that a catheter in situ time of no more than 48hours as adequate to preserve shunt patency, as well as finding no significant difference in shunt occlusion between non-commercial and commercial shunts. Oliver *et al*[43] confirmed the appropriateness of using TIVS in a peripheral hospital where vascular expertise is absent and expected definitive repair will be longer than 6 hours after the injury. These patients showed good results when transferred with TIVS in situ to a tertiary setting for definitive repair within 24 hours. The use of prophylactic fasciotomy remains indicated when prolonged ischaemic time is encountered. TIVS of the superior mesenteric artery has

been described by Reilly in 1995.[44] He shunted the SMA of one patient and was able to use an autologous vein graft as definitive repair at the relook surgery. The patient survived to day 63 before aspiration pneumonia caused his death. The use of TIVS in vascular injuries of the viscera isn't common practice.

Bleeding from solid organs can be managed in one of three ways; packing, repair or resection. The management remains very organ specific as well as being influenced by the general condition of the patient. Prolonged repair of solid organ injuries should be avoided in the unstable patient.[45] Packing of solid organ haemorrhage is very effective and controls the bleeding in the majority of cases in the damage control setting.

Apart from being an independent indicator for damage control, patients with high grade liver injuries (especially following blunt trauma) have high mortality rates.[46] The goals when addressing the injured liver in a damage control situation are to primarily halt the bleeding, then to remove devitalised tissue, prevent bile leaks and allow for adequate drainage.[47] It is only in the minority of cases with small liver lacerations that some form of definitive suture can be placed. Perihepatic packing forms the basis for achieving haemostasis in an injured liver. Occasionally intrahepatic packing with a haemostatic agent or omentum into a deep liver laceration can prove effective. Once the packing is in situ and tamponade achieved, special attention is to be given to venous return to the heart. Reduced venous return can be caused by excessive, bulky packing of the liver. In this case the packs need to be adjusted to prevent occlusion of the vena cava while maintaining haemostasis.[47] Excessive packing can also result in splinting of the diaphragm which impairs ventilation, leading to complications such as hypercarbia and atelectasis. Again, repacking is required to correct this. These packs can be left in situ without added risk of sepsis and bile leak

for up to 48hrs, when definitive management is required.[48] Early removal of the packs can result in rebleeding.

If the surgeon is unable to find the origin of the bleeding, applying the Pringle manoeuvre (*Figure 5*) can provide some measure of control, giving the surgeon some time to systematically look for the cause. Bleeding from the “surgical soul” (retrohepatic IVC, hepatic artery and vein, and portal vein) is associated with high mortality rate. Isolation and repair of these vessels is extremely challenging in the most experienced hands. Ligation of the IVC or hepatic artery (not both) can be tolerated. In some cases, it is prudent to leave a retrohepatic hematoma untouched if it isn’t actively bleeding.[49] IVC injuries below this can be explored and either repaired or ligated. It is preferred to leave a lumen of at least 25% of the original diameter when repairing a suprarenal IVC injury.[49] The use of prophylactic bilateral lower limb fasciotomies is not justified with IVC ligation.[50] Indications for exploration of Zone 1 hematomas still remain clear; expanding or pulsating hematoma, any suggestion of enteric injury (bubbles, fluid, bile). No evidence supports the need to expose and repair vena caval wounds that have spontaneously stopped bleeding. Such wounds, especially in the retrohepatic area, may be managed expectantly provided that there is no strong suspicion of an associated injury to a major artery or hollow viscus.[49]

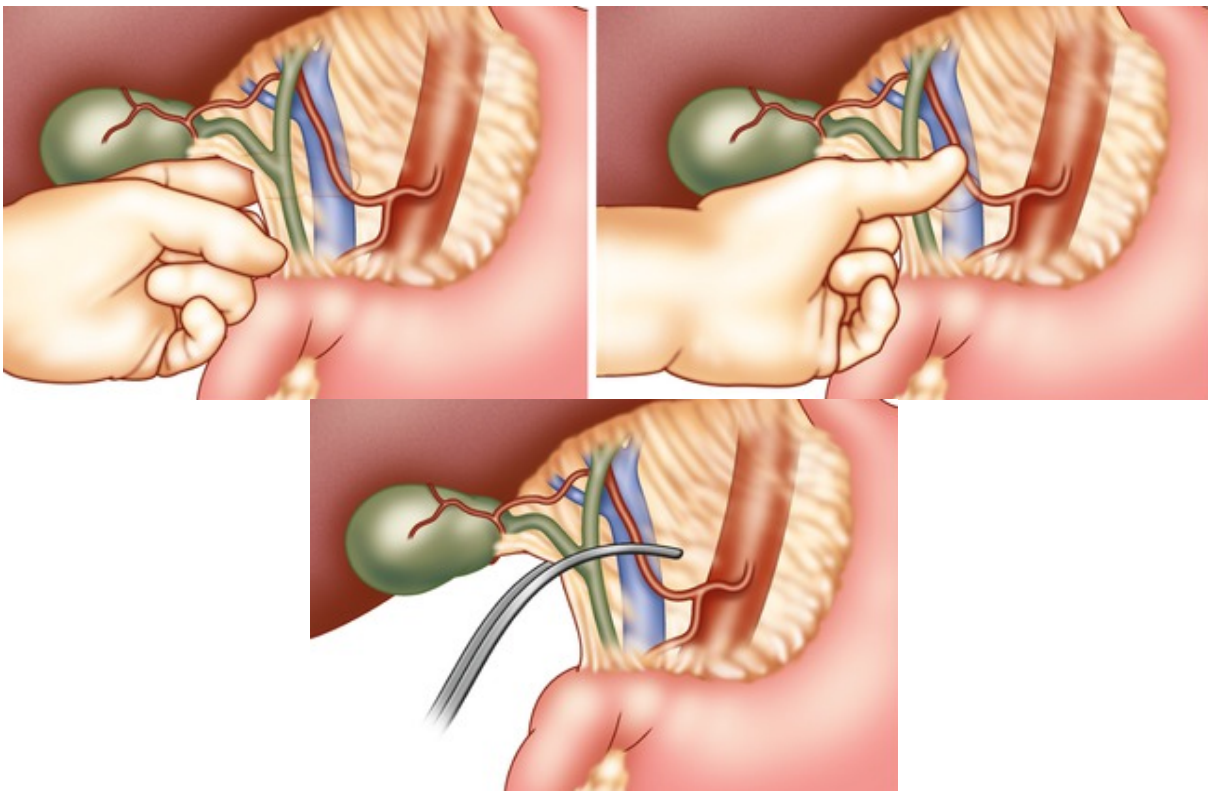

Figure 5 Pringle Manoeuvre

In dire circumstances, complete aortic inflow occlusion can be achieved with direct occlusion of the supra-coeliac aorta. This procedure can be done with exposure of the aorta behind the left lobe of the liver either with direct compression, or with clamping once the aorta has been dissected from the oesophagus. Clamping would free up the assistant's hand to help with the rest of the surgery. Apart from reducing bleeding while the surgeon searches for the source, it also gives the anaesthetist time to catch up with volume replacement. Clamping has shown to augment cerebral and myocardial perfusion in swine models.[51] The exact time of aortic clamping should be noted with the clamp being moved sequentially down the aorta as control of bleeding is gained.

The principles of managing splenic injuries is relatively simple. Splenectomy is an accepted practice in DCS for the bleeding spleen.[35] If the bleeding can be stopped

with a simple suture to a superficial laceration in the spleen, it can be done. The threshold for splenectomy is very low in current practice. No time should be wasted in making this decision. Pancreas injuries are treated with wide local drainage as repair or resection is a timeous excursion not fit for DCS scenarios.[34] The big problems here are the associated duodenal and biliary tree injuries, which will be discussed later.

The management of renal injuries relies on the extent of retroperitoneal bleeding and suspicion of associated injuries. Renal hematomas are explored if arterial injury is expected (expanding or pulsating hematoma). The practice of exposing every penetrating Zone 2 hematoma has been disputed by many.[52] Exposure of stable renal hematomas could lead to time consuming repair or renorrhaphy, or even partial nephrectomies of the injured kidneys, a practice not encouraged in the damage control setting. In patients with functioning opposite kidneys, the common practice would then be to do a nephrectomy of the injured kidney if the hematoma is explored. The availability of preoperative CT imaging, although not common in damage control, can assist with decision making in theatre. Any suspected renal injuries not an immediate threat to the patient's life can be managed at the relaparotomy, with appropriate imaging and the assistance of angiography and specialised urological expertise. Any extravesical bleeding can be controlled with either suture ligation (if intra-abdominal) or pelvic packing (if retroperitoneal).[52]

The management of "surgical bleeding" may not yet be complete following these interventions. It might well be necessary for angiographic intervention in order to obtain complete surgical haemostasis. Injuries where this might be indicated are bleeding from complex liver injuries, retroperitoneum, pelvic and deep muscle bleeding. All are injury locations not easily accessible or amenable to surgical

exploration, especially in the damage control setting.[45] It goes without saying that during this time, close monitoring and continued resuscitation are paramount in order to obtain a good outcome. The introduction of angio-embolisation to treat solid organ haemorrhage as part of DCS 1 was found to reduce mortality significantly.[53] Suen *et al* compared patient outcomes before and after the introduction of angio-embolisation at their institute (controlling for independent predictive factors of mortality). They found a reduction in mortality after the introduction of angio-embolisation, from 18.8% to 3.6% in the first 24hrs after admission.

Contamination control

The second priority during DCS 1 is control of sepsis. Hollow viscous injury causes spillage of bowel content, bile and urine.[45] All these eventually lead to sepsis and complicate the outcome of the patient if left unattended. Cleaning the mess and preventing further spillage are all that's required in the damage control setting. Bowel physiology and its healing capability is massively influenced by the general metabolic condition of a damage control patient. The use of inotropes and resuscitation fluids also make healing very unpredictable due to bowel oedema and vasoconstriction. It follows then that repairing any bowel from stomach to rectum comes with risk of leakage and breakdown. These benefit needs to far outweigh the potential risk in such a delicate situation. With this in mind only the small, well perfused injuries in the small bowel and colon should be repaired. For more severe injuries, especially with devitalised tissue requiring extensive debridement and anastomosis, ligation proves a much more attractive option. Small bowel and colon can be left in discontinuity for up to 48 hours before repair is required.[34]

The stomach is a well perfused organ with robust capabilities to heal. This means that repair with running sutures is indicated with most stomach injuries. Review with possible reinforcement or repeat repair at the relook might still be required for a satisfactory result. Ligation is rarely indicated but can be considered with devastating injuries where the likely outcome will be eventual partial/total gastrectomy. The duodenum is known for its vulnerability and high breakdown rates. Despite this, some form of repair should be attempted even if only viable to the relaparotomy.[54] The most important thing is to provide adequate drainage of these injuries. The involvement of the pancreaticoduodenal complex and sphincters may entail extensive and intricate surgery by hepatobiliary specialists.

Urinary leakage doesn't pose such a threat to patient outcome in DCS 1. If somehow missed in DCS 1, it mostly causes local irritation of surrounding peritoneum. The presence of vacuum dressing over the abdomen in damage control, usually means that most of the extravasated urine is removed from the abdomen.[52] It still remains important to have a high index of suspicion for urinary tract injuries. Bladder injury should be sutured, making sure not to include the ureteric openings in the repair. Injury to the ureters should preferably be drained externally in the damage control setting, although ligation is also accepted. The definitive repair can again be done at relaparotomy with the assistance of a urologist.

Injuries to the gallbladder can be oversewn or drained externally. Cholecystectomy has no place in the damage control setting. Injuries to the biliary tree are very morbid. If ligated initially, complex reconstructive surgery is required at reoperation. Cannulation and external drainage is the preferred option for managing these injuries in DCS 1.[33]

Abdominal closure

This is the last step in DCS 1 before the patient is taken to ICU. Closing the abdominal fascia is not part of damage control.[45] The pathophysiology of major trauma and the treatment thereof lends itself to massive fluid shifts into interstitial and cellular compartments. Bowel oedema and free fluid increase the risk of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS). With the developed understanding of IAH and ACS, the recommended practice is to use a temporary abdominal closure device (TAC). [21,34,35]The medical marketplace offers many commercial TAC options, but the principles can be applied with basic theatre equipment if needed (figure 6). The addition of negative pressure suction gives the added advantage removing any excess exudate and free fluid. The basic principle is drainage of oedema, preventing heat loss and providing a barrier to the atmosphere during DCS 2. Some devices attempt to prevent retraction of the skin and abdominal muscles, with limited success and cosmetic results. The goal is to eventually allow for abdominal closure in DCS 3.

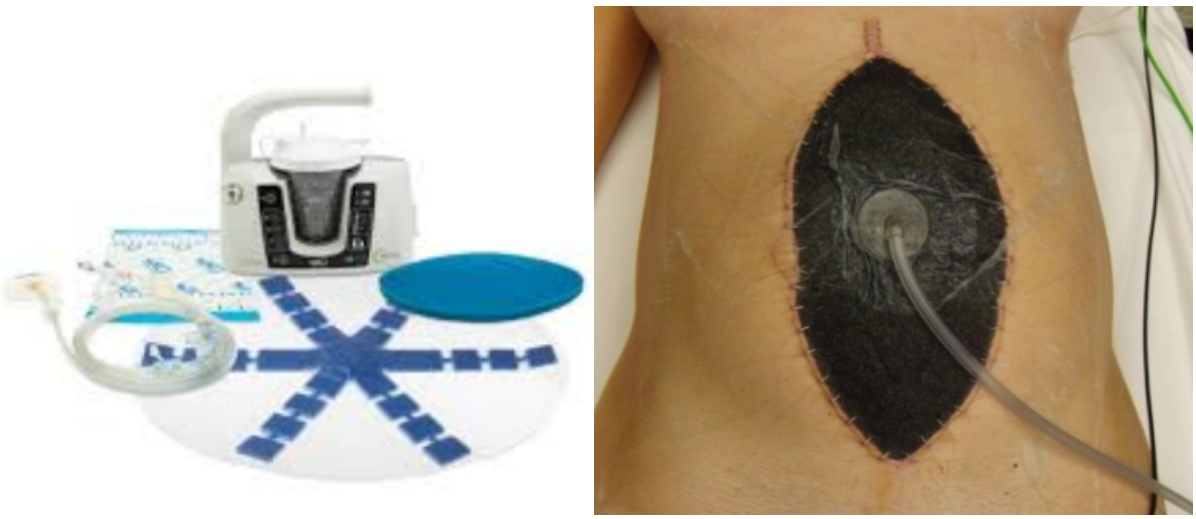

Figure 6 Commercial negative pressure temporary abdominal wall closure device

Damage Control part 2 (DCS 2)

With the surgical bleeding and contamination now under control, the priority shifts to aggressive resuscitation with the eventual goal of restoring some degree of normality with respect to physiology (coagulopathy and acidosis) and hypothermia.[55] This happens in an ICU with the first few hours being specifically labour intensive. Monitoring for changes in patient condition varies per institution. Many different monitoring options exist, in general trading accuracy of measurement for level of invasiveness.[45] The principles for fluid resuscitation are the same as in DC 1, with colloids and blood products preferred to crystalloid use. The endpoints are to have euvolemia with adequate end organ perfusion and oxidation.[55] The specific values or markers of successful resuscitation remain controversial. No specific group of values as endpoints for haemodynamics or physiology have been shown to predict survival. Abramson *et* al[56] however found that clearance of lactate within 24hrs to be an important prognostic factor for survival in injured patients. In his study, 100% of patients survived whose lactate cleared within 24 hrs. When cleared between 24 – 48 hrs, survival was 75%. If not cleared by 48 hrs, survival was only 14%. He also found that 40% of patients who died had achieved previously quoted markers of optimisation (O2 delivery (DO2) to 660 mL/min/m2, O2 consumption (VO2) to 170 mL/min/m2, and cardiac index (CI) of 4.5 L/min), further corroborating how insight into the patient's condition is more important than aiming for arbitrary endpoints of haemodynamics and metabolism.

Hypothermia

Rewarming of the patient forms an important and often neglected part of the solution. Gentilello showed how failure to address hypothermia after DCS is a marker of

inadequate resuscitation or irreversible shock. He also stated (which has been repeatedly confirmed in ensuing literature) the connection between worsening or persistent coagulopathy and hypothermia.[57] On a cellular level, hypothermia impairs thrombin generation and contributes to platelet dysfunction[58]. Rewarming can be divided into passive, active external and active internal rewarming. Passive rewarming is done with the use of blankets and depends on insulating the patient from a cold and wet environment. It requires the shivering reflex be intact. Active external rewarming entails applying heat directly to the skin. It assumes circulation is intact to return warmed blood to the core. Many commercial fluid and forced air warmers are available, but care should be taken not to burn the patient. Maintaining a room temperature of between 23-26˚C also falls under this category. Active internal rewarming entails warming any resus fluid and blood given to the patient. More aggressive ways are to use cardiopulmonary bypass or extracorporeal membrane oxygenation (ECMO) for profound hypothermia.[58] Both massively invasive and associated with significant complications.

Coagulopathy and fluid management

As mentioned earlier, the goal is to restore euvolemia using limited crystalloid resuscitation. DCR principles mentioned in DCS 0 apply here when it comes to specific component blood therapy and colloid infusion. The negative effects of aggressive crystalloid fluid resuscitation are well documented in recent medical literature. When reviewing the German Trauma Registry, Hussmann *et al*[59] concluded significantly more blood transfused, higher rates of coagulopathy and higher lethality in patient groups resuscitated with high fluid volumes (>1.5l) when compared to lower fluid volumes. He supported the concept of low volume resus in trauma patients. Crystalloid resuscitation is associated with a substantial increase in

morbidity, as well as ICU and hospital length of stay. Large fluid shifts with potential for pulmonary oedema and ARDS, as well as oedema of the bowel and abdominal viscera leading to IAH/ACS are dose dependant complications of large volume crystalloid resuscitation.[60] The immunomodulating effects can also increase the risk of septic complications. When using crystalloids, balanced salt solutions are preferred to normal saline, as the latter is associated with hyperchloraemic metabolic acidosis affecting the base deficit and pH of the patient.

The much preferred method is to use goal-directed component therapy and colloids as primary resus fluids. As mentioned in DCR earlier, the inherent acute traumatic coagulopathy along with exsanguination-related DIC and out-dated resus methods cause havoc with haemostasis. Despite surgical bleeding being arrested during DCS 1, this “medical bleeding” could on its own be the cause for patient demise if not addressed early and aggressively. The big difference between DCS 0 and DCS 2 is the setting. The ICU lends itself to a much better monitoring environment and in many cases more specialised personnel and allied services. In the majority of cases the monitoring devices have been inserted and used during DCS 1 in theatre, but now in what is usually a more controlled and focussed environment, goal-directed management can be fine-tuned. Thromboelastography (TEG) is a valuable investigation to guide the directed use of blood and component therapy, with survival benefit when used with fibrinogen and prothrombin concentrate.[61] The use of viscoelastic assays (e.g. TEG) has also shown survival benefit as well as reduced plasma and platelet transfusion loads, when compared with conventional methods of haemostatic testing (INR, fibrinogen level and platelet count).[62]

Correcting the metabolic acidosis is dependent on proper haemostasis and re-establishing adequate tissue perfusion. As mentioned earlier, the early return of a

high lactate to normal is associated with good outcome. No one intervention will achieve this, but more the sum of the different therapies that act together to correct the acidosis. A good knowledge of ventilation principles and use of bicarbonate could help with correcting the pH, but uncorrected underlying pathology will prevail if not addressed. With persistent acidosis despite good, aggressive resuscitation one has to have a low index of suspicion for ongoing bleeding or worsening sepsis. The presence of necrotic tissue (bowel, viscera, limbs) and worsening renal failure are also things to keep in mind. It should go without saying that in a damage control setting with persistent acidosis, there should be a very convincing reason not do an unplanned relook on the patient. Failing to return to theatre to treat a missed injury will prove catastrophic for the patient.[45]

As part of DCS 2, a complete physical examination needs to be done. Relevant imaging should be requested once the patient is stable enough to leave the ICU. Completion of the spinal survey in patients with blunt injury is prudent, as well as doing regular vascular checks of limbs.[45] The involvement of speciality surgical services should be done early in anticipation of the relook surgery where definite repair of injuries is expected. Planning these steps is necessary to achieve the best outcome.

Another reason for unplanned relook is with the development of ACS. The development of organ dysfunction is required to distinguish IAH from ACS. The poor outcomes achieved once ACS has developed make it crucial that accurate intraabdominal pressure monitoring is in place. Simple intermittent urine bladder pressure monitoring with a transducer can diagnose IAH.[63] This management of IAH starts off as conservative bedside interventions, ending with relook and decompression of the abdominal cavity once there is confirmation of ACS.[64] In the

DCS patient this could entail starting with opening the abdominal vacuum dressing in the ICU and monitoring for change in pressure, urine outcome and ease of ventilation. Without immediate relief, the patient should be taken to theatre and the problem addressed.[64]

Damage Control part 3 (DCS 3)

The timing for relook has been generally accepted as being between 24-48 hours.[55] The patient physiology should have been corrected by that stage with normalisation of acidosis, hypothermia and coagulopathy. Sometimes specific injury patterns require earlier take back. If the patient is stable, returning at 24hours would be prudent where bowel has been left in discontinuity. Another situation is where shunting of an artery has been done, with the potential of occlusion and a threatened limb.[45] As mentioned earlier, unplanned relaparotomy is indicated when the patient has ongoing transfusion requirements thought to be from a surgical cause, persistent acidosis and evolving ACS.[55]

The relaparotomy is the first time where some form of unrushed decision making can be made about the surgical procedure. A thorough and extensive handover of the injuries and initial laparotomy needs to be done if the surgical team has changed. All of the surgeons should partake in prioritising and planning the steps to address all the different injuries sustained. The goals of the surgery are for definitive repair of the injuries with complete fascial closure.[55]

On entering the open abdomen, the packs are irrigated and removed carefully to avoid further injury and rebleeding. Any rebleeding that does occur should be

handled with local haemostatic measures. Failure to do so should result in repacking and further definitive therapy reserved for future surgery. If hemostasis is achieved when the packs are removed, a complete re-examination of the abdominal cavity is performed with particular attention paid to the reported injuries. The examination should also note any missed injuries.

Definitive repair of the injuries should now be done. Enteric injuries should preferably be repaired and anastomosed, with few exceptions requiring ostomy diversion. Colonic injuries should be treated with specific care as leaks and breakdowns are devastating. With this in mind, decision making surrounding whether to anastomose or divert colonic injuries has been researched extensively. Outcomes have been shown to be no different when comparing primary anastomosis to diverting colostomy.[65,66] The ability to close abdominal fascia and skin is vitally important to this decision making. If abdominal closure isn't achieved at the first relook, then anastomosis of colonic injuries is associated with an eightfold increase in breakdown. The condition of the abdomen and the extent of the other injuries sustained play a vital role in deciding between anastomosing the colon or fashioning a stoma.

Due to the complexities of these structures and relatively high rate of leaks and breakdown, hepatobiliary repair and reconstruction should be done with the help of a specialised HPB service. Barrie *et al*[67] showed an increase in patient survival odds when HPB services were available for patients with liver and biliary trauma. Feeding tubes and closed suction drains should be placed as indicated.

Vascular injuries initially managed with TIVS will require definitive repair. Grafts can be either synthetic or autologous depending on the nature of the vascular injury and the suitability of veins. The use of systemic anticoagulants has been disputed, with only local anticoagulation being advocated for by most authors.[41] Unfortunately, amputation is also an option. This could be due to initial extensive injury with massive devitalised tissue, where despite rapid restoration of blood supply with TIVS the tissue is unsalvageable. Different scores such as the mangled extremity severity score (MESS) can help guide the surgeon with the difficult decision to amputate on the initial surgery or attempt TIVS.[68] Another reason could be delayed initial revascularisation (i.e. beyond the theoretical 6 hours) where the time of injury is unknown but the limb is “given a chance” but doesn’t survive. Undiagnosed shunt occlusion may occur, with ischaemic tissue discovered at the relook. When combined with orthopaedic injury, a markedly displaced fracture should be repaired under TIVS before definitive vascular repair is done. An immediate repair can be done if the fracture is stable or undisplaced.[69]

Urological injuries can mostly be managed by the trauma surgeon in DCS 1 with shunting, packing, resection or primary repair of the different organs. Definitive repair calls for more proven urologic principles to be implemented.[52] If not explored initially, the renal injury should be defined with CT imaging during DCS 2. Conservative management is becoming a more accepted way for treating an increasing amount of renal parenchymal injuries. Simple injuries to the urogenital system should be managed primarily by the trauma surgeon. Once injuries become complex or complicated, requiring intricate reconstruction or diversion, specialist urological services should be involved to ensure satisfactory outcomes.

Damage Control part 4 (DCS 4)

Abdominal Closure

One of the most common comorbidities is the inability to achieve primary fascial closure, leaving the patient with an open abdomen and all the challenges associated with it. Frequently encountered complications are entero-cutaneous fistulae, intra-abdominal infection, sepsis and ventral hernia.[70] Unfortunately many factors contribute to the failure of primary fascial closure. Dilated and oedematous bowel, ongoing intra-abdominal sepsis, repeated need for relook surgery, and the development of ACS are all possible reasons for the delay and eventual failure to close the abdomen primarily. The closure of an open abdomen has been extensively researched with many suggested treatment protocols. Kreis *et al*[71] found that attempting primary facial closure beyond 5-9days of the initial laparotomy is futile. Miller *et al*[15] reported a decrease in complications associated with fascial closure prior to 8 days (12% vs 52% for delayed closure). A prospective, multicentre study by Matthew *et al*[72] demonstrated reductions in primary fascial closure with delays in returning to theatre following DCS. He suggests returning to theatre no later than 48hrs (preferably within 24hrs). In the event of failed primary closure, the wound is left to granulate after which a skin graft is used to cover the wound. After about 12 – 18 months, the graft and subcutaneous tissue will separate from the underlying abdominal contents, at which time an abdominal wall reconstruction is attempted to repair the ventral hernia.

The huge burden of disease and morbid state that failed abdominal closure presents, has been the fuel which is driving the current research into reducing the DC rates.

The use of DCR principles has been shown to reduce the need for DCS as well as benefits in the ability to close the abdominal wall primarily.[73]\

Conclusion

DCS still carries significant morbidity and mortality rates. The practice has been associated with potentially severe complications, such as intraabdominal sepsis, massive ventral hernias, and entero-atmospheric fistulae. Multiple readmissions and surgeries add to the potential reduced quality of life among survivors.[74] Reports from multiple studies have mortality rates ranging between 18% - 30%.[70]

References

1. Stone HH, Strom PR, Mullins RJ. Management of the major coagulopathy with onset during laparotomy. *Ann Surg*. 1983; 197(5):532-535.

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(3):375-82; discussion 382-3.

3. Pringle JH. V. notes on the arrest of hepatic haemorrhage due to trauma. *Ann Surg*. 1908; 48(4):541-549.

4. Feliciano DV, Mattox KL, Jordan GL, Jr. Intra-abdominal packing for control of hepatic haemorrhage: A reappraisal. *J Trauma*. 1981; 21(4):285-290.

5. Madding GF, Kennedy PA. Trauma to the liver. *Major Probl Clin Surg*. 1971;3:1-130.

6. Lucas CE, Ledgerwood AM. Prospective evaluation of haemostatic techniques for liver injuries. *J Trauma*. 1976; 16(6):442-451.

7. Kashuk JL, Moore EE, Millikan JS, Moore JB. Major abdominal vascular trauma--a unified approach. *J Trauma*. 1982; 22(8):672-679.

8. Rotondo MF, Zonies DH. The damage control sequence and underlying logic. *Surg Clin North Am*. 1997; 77(4):761-777.

9. Johnson JW, Gracias VH, Schwab CW, et al. Evolution in damage control for exsanguinating penetrating abdominal injury. *J Trauma*. 2001; 51(2):261-9; discussion 269-71.

10. Shapiro MB, Jenkins DH, Schwab CW, Rotondo MF. Damage control: Collective review. *J Trauma*. 2000; 49(5):969-978.

11. Richards WO, Scovill W, Shin B, Reed W. Acute renal failure associated with increased intra-abdominal pressure. *Ann Surg*. 1983; 197(2):183-187.

12. Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. *Surg Clin North Am*. 1996; 76(4):833-842.

13. Morris JA, Jr, Eddy VA, Blinman TA, Rutherford EJ, Sharp KW. The staged celiotomy for trauma. Issues in unpacking and reconstruction. *Ann Surg*. 1993; 217(5):576-84; discussion 584-6.

14. Fabian TC, Croce MA, Pritchard FE, et al. Planned ventral hernia. Staged management for acute abdominal wall defects. *Ann Surg*. 1994; 219(6):643-50; discussion 651-3.

15. Miller RS, Morris JA, Jr, Diaz JJ, Jr, Herring MB, May AK. Complications after 344 damage-control open celiotomies. *J Trauma*. 2005; 59(6):1365-71; discussion 1371-4.

16. Roberts DJ, Bobrovitz N, Zygun DA, et al. Indications for use of damage control surgery and damage control interventions in civilian trauma patients: A scoping review. *J Trauma Acute Care Surg*. 2015; 78(6):1187-1196.

17. Mabry RL, Holcomb JB, Baker AM, et al. United States army rangers in Somalia: An analysis of combat casualties on an urban battlefield. *J Trauma*. 2000; 49(3):515-28; discussion 528-9.

18. Blackbourne LH. Combat damage control surgery. *Crit Care Med*. 2008; 36(7 Suppl):S304-10.

19. Neuhaus SJ, Bessell JR. Damage control laparotomy in the Australian military. *ANZ J Surg*. 2004; 74(1-2):18-22.

20. Smith IM, Beech ZK, Lundy JB, Bowley DM. A prospective observational study of abdominal injury management in contemporary military operations: Damage control laparotomy is associated with high survivability and low rates of faecal diversion. *Ann Surg*. 2015; 261(4):765-773.

21. Lamb CM, MacGoey P, Navarro AP, Brooks AJ. Damage control surgery in the era of damage control resuscitation. *Br J Anaesth*. 2014; 113(2):242-249.

22. Roberts DJ, Bobrovitz N, Zygun DA, et al. Indications for use of damage control surgery in civilian trauma patients: A content analysis and expert appropriateness rating study. *Ann Surg*. 2016; 263(5):1018-1027.

23. Krige JE, Nicol AJ, Navsaria PH. Emergency pancreatoduodenectomy for complex injuries of the pancreas and duodenum. *HPB (Oxford)*. 2014; 16(11):1043-1049.

24. Kirkpatrick AW, Roberts DJ, De Waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: Updated consensus definitions and clinical practice guidelines from the world society of the abdominal compartment syndrome. *Intensive Care Med*. 2013; 39(7):1190-1206.

25. Roberts I, Shakur H, Coats T, et al. The CRASH-2 trial: A randomised controlled trial and economic evaluation of the effects of tranexamic acid on death, vascular occlusive events and transfusion requirement in bleeding trauma patients. *Health Technol Assess*. 2013; 17(10):1-79.

26. Ausset S, Glassberg E, Nadler R, et al. Tranexamic acid as part of remote damage-control resuscitation in the prehospital setting: A critical appraisal of the medical literature and available alternatives. *J Trauma Acute Care Surg*. 2015; 78(6 Suppl 1):S70-5.

27. Brohi K, Singh J, Heron M, Coats T. Acute traumatic coagulopathy. *J Trauma*. 2003; 54(6):1127-1130.

28. Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: Directly addressing the early coagulopathy of trauma. *J Trauma*. 2007; 62(2):307-310.

29. Hodgetts TJ, Mahoney PF, Kirkman E. Damage control resuscitation. *J R Army Med Corps*. 2007; 153(4):299-300.

30. Cotton BA, Reddy N, Hatch QM, et al. Damage control resuscitation is associated with a reduction in resuscitation volumes and improvement in survival in 390 damage control laparotomy patients. *Ann Surg*. 2011; 254(4):598-605.

31. Holcomb JB, Tilley BC, Baraniuk S, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: The PROPPR randomized clinical trial. *JAMA*. 2015; 313(5):471-482.

32. Osborne LA, Brenner ML, Prater SJ, Moore LJ. Resuscitative endovascular balloon occlusion of the aorta: current evidence. Emergency Medicine. 2019; 11: 29-38.

33. Hirshberg A MK. *Top knife: The art and craft of trauma surgery.* Shrewsbury: TFM Publishing; 2005.

34. Ball CG. Damage control surgery. *Curr Opin Crit Care*. 2015; 21(6):538-543.

35. Nicol AJ, Navsaria PH, Krige JE. Damage control surgery. *S Afr J Surg*. 2010; 48(1):4-5.

36. MYHRE JR. Balloon tamponade of haemorrhagic oesophageal varices. *Tidsskr Nor Laegeforen*. 1958; 78(11):511-513.

37. Feliciano DV, Burch JM, Mattox KL, Bitondo CG, Fields G. Balloon catheter tamponade in cardiovascular wounds. *Am J Surg*. 1990; 160(6):583-587.

38. Navsaria P, Thoma M, Nicol A. Foley catheter balloon tamponade for life-threatening haemorrhage in penetrating neck trauma. *World J Surg*. 2006; 30(7):1265-1268.

39. Ball CG, Wyrzykowski AD, Nicholas JM, Rozycki GS, Feliciano DV. A decade's experience with balloon catheter tamponade for the emergency control of haemorrhage. *J Trauma*. 2011; 70(2):330-333.

40. 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(7):605-607.

41. 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(9):970-977.

42. Inaba K, Aksoy H, Seamon MJ, et al. Multicenter evaluation of temporary intravascular shunt use in vascular trauma. *J Trauma Acute Care Surg*. 2016; 80(3):359-64; discussion 364-5.

43. Oliver JC, Gill H, Nicol AJ, Edu S, Navsaria PH. Temporary vascular shunting in vascular trauma: A 10-year review from a civilian trauma centre. *S Afr J Surg*. 2013; 51(1):6-10.

44. Reilly PM, Rotondo MF, Carpenter JP, Sherr SA, Schwab CW. Temporary vascular continuity during damage control: Intraluminal shunting for proximal superior mesenteric artery injury. *J Trauma*. 1995; 39(4):757-760.

45. Lamb CM, MacGoey P, Navarro AP, Brooks AJ. Damage control surgery in the era of damage control resuscitation. *Br J Anaesth*. 2014; 113(2):242-249.

46. Hommes M, Chowdhury S, Visconti D, et al. Contemporary damage control surgery outcomes: 80 patients with severe abdominal injuries in the right upper quadrant analysed. *Eur J Trauma Emerg Surg*. 2017.

47. Yu WY, Li QJ, Gong JP. Treatment strategy for hepatic trauma. *Chin J Traumatol*. 2016; 19(3):168-171.

48. Nicol AJ, Hommes M, Primrose R, Navsaria PH, Krige JE. Packing for control of haemorrhage in major liver trauma. *World J Surg*. 2007; 31(3):569-574.

49. Buckman RF, Pathak AS, Badellino MM, Bradley KM. Injuries of the inferior vena cava. *Surg Clin North Am*. 2001; 81(6):1431-1447.

50. Hampton M, Bew D, Edu S, Nicol A, Naidoo N, Navsaria P. An urban trauma centre experience with abdominal vena cava injuries. *S Afr J Surg*. 2016; 54(1):36-41.

51. White JM, Cannon JW, Stannard A, et al. Direct vascular control results in less physiologic derangement than proximal aortic clamping in a porcine model of noncompressible extrathoracic torso haemorrhage. *J Trauma*. 2011; 71(5):1278-86; discussion 1286-7.

52. Smith TG, 3rd, Coburn M. Damage control manoeuvres for urologic trauma. *Urol Clin North Am*. 2013; 40(3):343-350.

53. Suen K, Skandarajah AR, Knowles B, Judson R, Thomson BN. Changes in the management of liver trauma leading to reduced mortality: 15-year experience in a major trauma centre. *ANZ J Surg*. 2016; 86(11):894-899.

54. Ordonez C, Garcia A, Parra MW, et al. Complex penetrating duodenal injuries: Less is better. *J Trauma Acute Care Surg*. 2014; 76(5):1177-1183.

55. Waibel BH, Rotondo MM. Damage control surgery: It's evolution over the last 20 years. *Rev Col Bras Cir*. 2012; 39(4):314-321.

56. Abramson D, Scalea TM, Hitchcock R, Trooskin SZ, Henry SM, Greenspan J. Lactate clearance and survival following injury. *J Trauma*. 1993; 35(4):584-8; discussion 588-9.

57. Gentilello LM. Advances in the management of hypothermia. *Surg Clin North Am*. 1995; 75(2):243-256.

58. Simmons JW, Powell MF. Acute traumatic coagulopathy: Pathophysiology and resuscitation. *Br J Anaesth*. 2016; 117(suppl 3):iii31-iii43.

59. Hussmann B, Lefering R, Waydhas C, et al. Does increased prehospital replacement volume lead to a poor clinical course and an increased mortality? A matched-pair analysis of 1896 patients of the trauma registry of the German society for trauma surgery who were managed by an emergency doctor at the accident site. *Injury*. 2013; 44(5):611-617.

60. Kasotakis G, Sideris A, Yang Y, et al. Aggressive early crystalloid resuscitation adversely affects outcomes in adult blunt trauma patients: An analysis of the glue grant database. *J Trauma Acute Care Surg*. 2013; 74(5):1215-21; discussion 1221-2.

61. Schochl H, Nienaber U, Hofer G, et al. Goal-directed coagulation management of major trauma patients using thromboelastometry (ROTEM)-guided administration of fibrinogen concentrate and prothrombin complex concentrate. *Crit Care*. 2010; 14(2):R55.

62. Gonzalez E, Moore EE, Moore HB, et al. Goal-directed haemostatic resuscitation of trauma-induced coagulopathy: A pragmatic randomized clinical trial comparing a

viscoelastic assay to conventional coagulation assays. *Ann Surg*. 2016; 263(6):1051-1059.

63. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. *Ann Surg*. 1984; 199(1):28-30.

64. Kirkpatrick AW, Roberts DJ, De Waele J, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: Updated consensus definitions and clinical practice guidelines from the world society of the abdominal compartment syndrome. *Intensive Care Med*. 2013; 39(7):1190-1206.

65. Anjaria DJ, Ullmann TM, Lavery R, Livingston DH. Management of colonic injuries in the setting of damage-control laparotomy: One shot to get it right. *J Trauma Acute Care Surg*. 2014; 76(3):594-8; discussion 598-600.

66. Demetriades D, Murray JA, Chan L, et al. Penetrating colon injuries requiring resection: Diversion or primary anastomosis? an AAST prospective multicentre study. *J Trauma*. 2001; 50(5):765-775.

67. Barrie J, Jamdar S, Iniguez MF, et al. Improved outcomes for hepatic trauma in England and wales over a decade of trauma and hepatobiliary surgery centralisation. *Eur J Trauma Emerg Surg*. 2017.

68. Johansen K, Daines M, Howey T, Helfet D, Hansen ST,Jr. Objective criteria accurately predict amputation following lower extremity trauma. *J Trauma*. 1990; 30(5):568-72; discussion 572-3.

69. Beard J. ***Vascular and endovascular surgery A companion to specialist surgical practice****.* 5th Edition ed. Saunders Ltd.; 2013.

70. Zosa BM, Como JJ, Kelly KB, He JC, Claridge JA. Planned ventral hernia following damage control laparotomy in trauma: An added year of recovery but equal long-term outcome. *Hernia*. 2016; 20(2):231-238.

71. Kreis BE, de Mol van Otterloo AJ, Kreis RW. Open abdomen management: A review of its history and a proposed management algorithm. *Med Sci Monit*. 2013; 19:524-533.

72. Pommerening MJ, DuBose JJ, Zielinski MD, et al. Time to first take-back operation predicts successful primary fascial closure in patients undergoing damage control laparotomy. *Surgery*. 2014; 156(2):431-438.

73. Joseph B, Azim A, Zangbar B, et al. Improving mortality in trauma laparotomy through the evolution of damage control resuscitation: Analysis of 1,030 consecutive trauma laparotomies. *J Trauma Acute Care Surg*. 2017; 82(2):328-333.

74. Roberts DJ, Ball CG, Feliciano DV, et al. History of the innovation of damage control for management of trauma patients: 1902-2016. *Ann Surg*. 2017; 265(5):1034-1044.

SUBMISSION READY MANUSCRIPT

Damage Control Laparotomy (DCL) outcomes in a Major Urban Trauma Centre

Trauma remains a worldwide leading cause of morbidity and death. The Western Cape has a particularly high rate of interpersonal trauma, with a high proportion being penetrating in nature. This fact lends itself to specific injury patterns that victims present to hospital with, requiring specialised trauma and surgical care (1). Among these surgical techniques is the concept of Damage Control Surgery (DCS). Originally documented by Pringle in 1902 (2) as a staged laparotomy, the innovation of Damage Control (DC) progressed through the middle and late 1900's. Its use waxed and waned, gaining traction in the Second World War, but then being largely abandoned during the Korean and Vietnam wars where it was seen as a sign of poor surgical skills (3). It wasn't until Stone et al (4) and then, a decade later, Rotondo et al (5) showed its benefit, that DC was finally accepted into mainstream trauma surgery. DC starts in the trauma unit with well-defined resuscitation interventions and goals. Surgical principles include an abbreviated surgery where the priority is to arrest haemorrhage and limit hollow viscous contamination in an attempt to stop or reverse the bloody vicious cycle of acidosis, hypothermia and coagulopathy. Definitive repair of injuries occurs once the metabolic insult and coagulopathy have been reversed in ICU, no later than 48 hours after the first surgery (6) (7). This approach has been shown to significantly reduce mortality when used appropriately (5) (8). DCS is not without its own complications however. Intra-abdominal

collections, entero-atmospheric fistulas and large ventral hernias are well documented complications each with great morbidity (9). The level one trauma centre at Groote Schuur Hospital drains exceptionally high numbers of penetrating trauma, keeping it at the pinnacle of trauma care worldwide. The multitude of severe trauma seen and treated at this trauma centre, make for the perfect research sample from which to identify common factors that could improve the process of identifying, monitoring and treating these patients.

The objectives of this study are to review the outcomes of DCL, identifying pre-operative markers for patients requiring DCL and assess intra-operative parameters determining death or survival likelihood of survival with the aim of early identification of patients requiring DCL. Further aims are to evaluate primary closure rates.

Methods

All patients undergoing DCL for penetrating trauma from May 01, 2015 to July 31, 2017 were reviewed from the prospectively recorded eTHR (electronic health record) data base. Damage control laparotomy was defined as an abbreviated laparotomy that aimed to rapidly and effectively control haemorrhage and/or contamination and which ended with temporary closure of the laparotomy wound. In contrast, a definitive laparotomy was defined as the completion of repairs of all abdominal injuries followed by formal fascial closure of the abdomen during the index operation.

Reviewed data included basic demographics, mechanism of injury, perioperative vitals and biochemical parameters. Injury severity was categorised by the Revised Trauma Score (RTS), Penetrating Abdominal Trauma Index (PATI), Injury Severity Score (ISS) and the Trauma and Injury Severity Score (TRISS). Indications for DCL were recorded as well as length of ICU stay, days of ventilation, number of procedures and primary abdominal closure rates. Complications and mortality were recorded.

Further analysis placed the data into groups of survivors and non-survivors. Statistical analysis will look for significant differences in physiological and injury score markers between survivors and non-survivors. These maybe be used as indicators to identify patients requiring DCR early and monitor their progress accurately.

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) v. 24.0 (SPSS Inc, Chicago, IL, USA). Descriptive statistics will include point estimates (mean, median, mode) and measures of dispersion (standard deviation, range, quantiles) where appropriate.

Nominal categorical variables were analyzed by the chi-squared test for independence or Fisher's exact test (with statistical parameter modification as appropriate) and by non-parametric tests for ordinal data. Numerical variables were

analyzed by parametric and non-parametric test as indicated by the Kolmogorov-Smirnov test for normal data distribution.

A confidence level of 95% was used. An upper and lower bound will be calculated by the method of bootstrapping of 10,00 resamples when required. Unless otherwise indicated, a two-tail test hypothesis will be used with an alpha-value of 0.05 as discriminator for rejection of the null-hypothesis.

Results

Between May 2015 and July 2017 fifty-one (51) patients underwent DCL at Groote Schuur Hospital. Fifty (50) patients were men, ranging in age from 15 to 48 (mean 28.3) years. Forty-seven (92%) of the patients sustained gunshot wounds (GSW) and four (8%) were stabbed. The median number of GSW's per patient was three. Delay to admission averaged 131 minutes (2 hours and 11 minutes), with a total delay to surgery averaging 456 minutes (7 hours and 36 minutes). On admission, the mean systolic blood pressure was 116 mmHg, median heart rate of 109 beats per minutes, median respiratory rate of 22 breaths per minute and mean temperature of 35.1 degrees Celsius. The admission arterial blood gas means were haemoglobin 10.35g/dl, pH 7.27, base deficit -8.25 mEq/l and lactate 6.36. Three preoperative trauma indices were calculated. The mean Revised Trauma Score (RTS) was 7.36, mean Injury Severity Score (ISS) was 21.18 and the probability of survival (TRISS) was 93.76 (Table 1).

The immediate indication for laparotomy was for haemodynamic instability in 27 (53%) patients and peritonism in 22 (43%) patients. In one (2%) patient the indication was based on radiological findings as the patient was intubated and abdominal examination was unreliable. In another patient (2%) an initial decision was made to admit the patient for non-operative management of his injuries. He deteriorated and was taken for surgery. The mean operative time was 156 minutes (2 hours and 36 minutes). A Penetrating Abdominal Trauma Index (PATI) score was calculated from the intraoperative findings, the median score being 28. The median for the total number of packed red cells (PRC) given in the first 24hrs was 6.55 and for intraoperative fresh frozen plasma (FFP) was 2.57. Vasopressors were used in forty-six (90%) patients intraoperatively. The mean intraoperative blood loss was 3256ml. Table 2 depicts the intra-abdominal organ injuries. Table 3 shows the frequencies of the different DCL procedures used to address the injuries found. All patients that survived the index surgery were sent to ICU for continued resuscitation with temporary abdominal closure (TAC) devices in situ. The average length of ICU stay was 6.44 days and average days of ventilation were 4.84 days. Table 4 shows the different complications with their frequencies. Nineteen (37%) patients required a second relook within 30 days with a median of 3 procedures per patient. Primary closure of the abdominal wall was achieved in 35 (69%) patients. 15 (29%) of the patients undergoing DCL died.

A univariate analysis was performed on pre-operative patient parameters comparing survivors and non-survivors (Table 5). Increase in patient's ER temperature proved significant in predicting mortality as in independent variable (OR 2.02; 95%CI 1.12 - 4.32; p 0.04). Increased transfusion of packed red cells was also significant in

predicting death as an independent variable (OR 1.13; 95% CI 1.01 - 1.31; p 0.05). No other pre-operative variable showed significance in predicting death. A multivariate analysis of these same parameters showed patient's ER temperature (OR 2.85; 95% CI 1.30 - 7.65; p 0.02), ER hemoglobin (OR 1.42; 95% CI 1.03 - 1.20; p 0.04) and hemodynamic instability (OR 10.3; 95% CI 1.60 - 93.2; p 0.02) as significant predictors of death when controlling for other variables (Table 6). Of the three trauma scoring systems analysed, only the PATI score showed significance in predicting death (Table 7).

A univariate analysis of intra-operative parameters showed multiple significant parameters between survivors and non-survivors (Table 8). Transfusion requirements proved significant when comparing survivors to non-survivors, with packed red cell (OR 1.18; 95% CI 1.04 – 1.39; p 0.02), fresh frozen plasma (OR 1.44; 95% CI 1.11 – 1.95; p 0.01) and platelet (OR 2.82; 95% CI 1.42 – 6.98; p 0.01) transfusions all showing significance. When comparing patient specific variables, the only significant findings were lowest systolic BP (OR 0.94; 95% CI 0.88 – 0.98; p 0.02), mean arterial pressure (OR 0.90; 95% CI 0.82 – 0.96; p 0.01), lowest pH (OR <0.01; 95% CI <0.01 – 0.04; p 0.01), lowest base excess (OR 0.86; 95% CI 0.74 – 0.98; p 0.03) and lowest bicarbonate (OR 0.80; 95% CI 0.64 – 0.96; p 0.03). A multivariate analysis failed to provide any significant differences between variables of survivors and non-survivors (Table 9).

Discussion

DCS has proven to reduce mortality rates in patients with major intra-abdominal injuries (3)(5). Commonly quoted mortality rates range from 17 – 67% (10). A previous review of DCL for abdominal gunshot wounds at our center reported a mortality rate of 54% (10). Reasons for this high mortality rate, as described in the article, could be due to delay to admission and surgery. The mortality rate of 29% attained in this review is more in line with international standards. Eleven of the 15 deaths (73%) occurred within 48 hrs, most likely as a direct consequence of the injuries sustained. Six deaths were attributed to haemorrhage and another six to septic shock with multi-organ failure. One death is due to self-extubation in the critical care unit resulting in hypoxic brain injury, and another due to refractory hyperkalaemia as a result of acute renal dysfunction.

Patient selection for DCS is critical. Early recognition assures prompt aggressive resuscitation and speedy transfer to theatre. DCR principles have had a drastic effect on patient outcomes, addressing the problems of hypocoagulability, hypothermia and acidosis even before surgery (6) (11). Deciding which patients are candidates for DCS has far reaching consequences. Indications for DCS can be divided into patient's parameters during resuscitation and injury patterns. Table 10 lists some of these (12). Broadly speaking, the indications can be grouped into physiological parameters and injury complexes. Other indications include massive transfusion due to massive ongoing haemorrhage and anticipated prolonged surgery in severely injured patients. Roberts *et al* identified substantial uncertainty around

when DCS is indicated, highlighting the need for further evidence-based consensus indications (3).

On univariate analysis of the pre-operative variables, only admission temperature and day 1 red cell transfusion proved significant. A multivariate regression model further found ER temperature and ER haemoglobin as significant variables. Both ER temperature and ER haemoglobin have positive odds ratios, meaning for each unit of increase (i.e. degree Celsius for temperature and g/dL for haemoglobin) the odds of death increase. For haemoglobin, this can be explained by haemo-concentration expected with acute massive blood loss most likely experienced by the non-survivors over the survivors. This is supported by the significance of pre-operative blood transfusion being a significant factor when comparing survivors to non-survivors in the univariate analysis. The effects of haemorrhagic shock with haemodynamic instability as an indicator for surgery proved significant to increase the odds of death. It is well understood that significant blood loss starts the “bloody vicious cycle” so often described in damage control (13). Large volumes of blood loss also result in the need for massive transfusions. Both these factors serve to explain why haemodynamic unstable patients are at higher odds of death.

The distribution of injuries and DCS techniques to treat these correlate with other studies (8) (15). The most common hollow viscous injury is small bowel followed by colon. For solid organs, the liver is most commonly injured followed by the kidneys and spleen. This then follows that the most common haemostatic technique used was abdominal packing and sepsis control technique was bowel ligation.

Univariate analysis of intra-operative variables revealed the following factors associated with increase odds of death after DCL: shorter time from incident to surgery, higher volume of blood products transfused within the first 24 hours (packed red cells, FFP's and cryoprecipitate), lower systolic blood pressure (SBP) and mean arterial pressure (MAP) as well as lower pH and bicarbonate and higher base excess.

It is well understood that a delay to surgery results in worse outcomes. The average time from incident to delay for the whole study group is 332 minutes. This goes to show the deficiencies our system has with incident reporting, ambulance response time and access to theatre. This undoubtedly leads to a situation where patients arriving at hospital have self-selected. You could argue that the "golden hour" for immediate intervention is no longer relevant in the majority of the patients. The patients are then triaged for theatre according to admission haemodynamics and blood gas parameters, leading to the sicker patients being rushed to theatre, hence the shorter delay having worse outcomes.

The significance of the greater volume of transfused blood products leading to higher chance of death correlates with the earlier discussion that the effect of the "bloody vicious cycle" and massive transfusion has on further hypocoagulability and potential hypothermia. The lower haemodynamic and biochemical parameters proving significant just indicate the extent of the injuries that the non-survivors have, and not necessarily a failure in DCS or the decision-making process. Aoki *et al* used a predictive model for survival by looking at the ability to correct pH at the conclusion

of the DCS and the worst PTT. In their study, they had a 100% mortality rate with pH ≤ 7.2 as well as 100% mortality rate with a worst PTT ≥ 78.7 seconds (16). We only collected the worst blood gas values throughout the surgery and didn't look at the trends of these values during the procedure to evaluate whether they were improving or deteriorating.

DCS is not without its complications. These patients mostly have massive injuries, causing major disturbances to physiology. They all require ventilation and possibly organ support in an ICU setting and are prone to septic complications by the nature of their enteric injuries. The number and variation of complications we encountered (Table 4) are common to these procedures. Our primary fascial closure rate is 69% in keeping with the reported literature (49 - 75%) (17) (18) (19).

The results and consequent deductions should be made with caution given the small sample size of the review. Generalisability is also limited by the local scenario of extreme gang violence and some shortfalls encountered in the public sector with service delivery. Although some findings are in keeping with other research, it must be kept in mind that the study is under powered.

Conclusion

DCL in our setting is associated with a 29% mortality rate and primary abdominal closure rate of 68%. Preoperative severe acidosis, the intraoperative need for a massive transfusion in the first 24 hours and median PATI score of 47 were independent predictors for increased mortality.

Table 1 – Descriptive parameters of the study sample

Variables

Gender	N (%)	Blood Investigations	Mean
Male	50 (98)	Pre-op Hb	10.35
Female	1 (2)	Pre-op Ph	7.27
Age in years (mean)	28.3	Pre-op base deficit	-8.25
Mechanism	N (%)	Pre-op lactate	6.36
GSW's	47 (92)	Trauma indicated	Mean
Stabs	4 (8)	ISS (median)	21
Pre-operative vitals	Mean	TRISS	93.76
Systolic BP	116	RTS	7.36
Pulse rate	109	PATI	28
Respiratory rate	22	Intra-operative parameters	Mean
Temperature	35.1	Temperature	34.9
		pH	7.09
		Base deficit	-12.3
		Lactate	7.11

Table 2 – Incidence of injuries

Injuries	No. of injuries
Small bowel	33
Colon	25
Abdominal vasculature	22
Liver	18
Stomach	14
Kidney	10
Diaphragm	10
Spleen	9
Pancreas	8

Table 3 – Frequency of DCL procedures

DCS procedures	
Abdominal packing	36
Bowel ligation	30
Vascular ligation	9
Splenectomy	9
Nephrectomy	6
Vascular shunting	5
Ureter shunting	1

Table 4 - Complications categorised by Clavien Dindo Classification

Grade	Number (% of complications)	Observed Complications (N)
1	18 (21)	Paralytic ileus (14)
		Superficial SSI (4)
2	17 (20)	Urinary Tract Infection (1)
		Pneumonia (5)
		Deep SSI (5)
		ARDS (1)
		DVT (2)
		Ileus requiring TPN (3)
3a	1 (1)	Intra-abdominal collection (1)
3b	17 (20)	Intra-abdominal collection (4)
		Bowel obstruction (5)
		Open abdominal wound needing SSG (7)
4a	18 (21)	Acute Kidney Injury without dialysis (12)
		Acute Kidney Injury with dialysis (6)
4b		
5	15 (17)	Mortality

Table 5 - Univariate analysis of pre-operative numerical variables

Variable	Group	Mean (SD)	Min / Max	Median	IQR	OR (95% CI)	p-value
Age in years	A****	27.6 (7.6)	15 / 45	27	10.5	1.03 (0.96 - 1.10)	0.41
	D*****	29.8 (10.3)	18 / 48	26.5	13.3		
Minutes to admission	A	150 (177.2)	15 / 930	96	92.5	0.99 (0.98 - 1.00)	0.2
	D	87.9 (39.5)	30 / 150	75	61		
Prehospital heart rate in beats per minute	A	103 (26)	55 / 159	104	40	1.0 (0.97 - 1.03)	0.89
	D	105 (21)	76	140	29		
Prehospital respiratory rate	A	23 (8)	8 / 42	20	9	0.96 (0.86 - 1.06)	0.48
	D	21 (6)	12 / 32	20	6		
Prehospital systolic blood pressure (mmHg)	A	109 (26)	57 / 149	108	50	1.01 (0.98 - 1.04)	0.45
	D	115 (22)	92 / 161	114	20		
ER* heart rate	A	109 (24)	62 / 160	109	29	1.00 (0.98 - 1.03)	0.76
	D	112 (27)	63 / 160	108	33		
ER respiratory rate	A	23 (7)	14 / 40	21	10	1.05 (0.98 - 1.13)	0.15
	D	27 (11)	16 / 57	24	7		
ER systolic blood pressure	A	116 (29)	68 / 167	122	43	1.00 (0.98 - 1.03)	0.83
	D	118 (24)	81 / 163	116	33		
ER temperature	A	34.8 (1.3)	32 / 36.9	35.2	1.5	2.02 (1.12 - 4.32)	**0.04**
	D	35.7 (0.9)	33.5 / 37.5	35.6	1.1		
ER pH	A	7.26 (0.14)	6.87 / 7.47	7.29	0.14	5.14 (0.04 - 1529)	0.53
	D	7.28 (0.10)	6.97 / 7.39	7.31	0.06		
ER base excess	A	-8.1 (6.6)	-29.4	-7.9	7.5	0.99 (0.90 - 1.1)	0.83
	D	-8.5 (4.2)	-15.5	-7.4	5.1		
ER HCO_3^-	A	18.1 (5.0)	8.0 - 30.3	17.6	5.8	0.96 (0.83 - 1.11)	0.61
	D	17.3 (3.2)	9.4 - 21.4	17.5	4		

ER lactate	A	6.9 (5.0)	1.4 - 22.4	5.9	5.5	0.91 (0.74 - 1.06)	0.26
	D	5.3 (2.7)	1.2 - 10.9	4.2	3.6		
ER white cell count	A	12.3 (6.5)	2.5 - 32.6	11.6	8.2	1.03 (0.94 - 1.14)	0.5
	D	14.0 (8.8)	0.8 - 28.3	13.8	12.6		
ER hemoglobin	A	10.0 (2.3)	6.0 - 15.5	9.8	3.1	1.29 (0.98 - 1.78)	0.09
	D	11.2 (2.0)	8.0 - 13.7	11.4	4		
ER PRBCs**	A	1.1 (1.4)	0 - 6	1	2	1.17 (0.76 - 1.78)	0.46
	D	1.4 (1.5)	0 - 6	1	2		
Time to surgery	A	338.3 (431.4)	30 - 2160	231	341.5	1.0 (0.99 - 1.0)	0.08
	D	179.9 (93.1)	110 - 395	135	71		
Operative time	A	150.3 (81.9)	65 - 495	125	67.5	1.0 (1.0 -1.0)	0.45
	D	169.3 (78.4)	60 - 320	160	80		
Day 1 PRBC units	A	5.3 (3.6)	0 - 15	3.5	3.5	1.13 (1.01 - 1.31)	**0.05**
	D	9.2 (8.1)	0 - 29	7	6		

* Lowest emergency room value

** Emergency room packed red blood cell units

*** Fresh frozen plasma

**** Alive

***** Deceased

Table 6 - Multivariate model of preoperative variables

Variable	Odds ratio (95% CI)	*p* value
ER temperature	2.85 (1.30 - 7.65)	**0.02**
Hemodynamic responder	4.31 (0.70 - 36.9)	0.14
Hemodynamic instability	10.3 (1.60 - 93.2)	**0.02**
ER hemoglobin	1.42 (1.03 - 1.20)	**0.04**
ER respiratory rate	1.08 (0.99 - 1.20)	0.09

Table 7 - Univariate analysis of the three trauma scores

Score	Group	Min - max	Median	IQR	OR (95% CI)	*p* value
RTS	A	3.8028 - 7.8408	7.8408	0.7326	0.65 (0.22 - 1.36)	0.33
	D	5.9672 - 7.8408	7.8408	0.2908		
PATI	A	8 – 59	24	13	1.06 (1.02 - 1.12)	**0.01**
	D	9 – 55	47	24		
ISS	A	9 – 43	17	9	1.03 (0.96 - 1.10)	0.39
	D	9 -42	22	11		

Table 8 - Univariable analysis of numerical variables

Variable	Group	Median	IQR	OR (95% CI)	p-value
Surgical delay from admission	A	231	303	0.99 (0.99 - 1.00)	0.07
	D	138	66.5		
Surgical delay from incident	A	360	445	0.99 (0.99 - 1.00)	**0.05**
	D	265	58.2		
Indication for laparotomy (hemodynamic instability)	A			2.94 (0.82 - 12.32)	0.11
	D				
Operative time	A	138	75	1.00 (0.99 - 1.01)	0.55
	D	158	68.8		
PRC in first 24hrs	A	5	4	1.18 (1.04 - 1.39)	**0.02**
	D	7.5	6		
FFP during surgery	A	2	3	1.44 (1.11 - 1.95)	**0.01**
	D	4	3.75		
Platelets during surgery	A	0	1	2.82 (1.42 - 6.98)	**0.01**
	D	1	2		
Cryoprecipitate during surgery	A	0	0	1.01 (0.36 - 2.50)	0.98
	D	0	0.75		
Vasopressor during surgery	A			1.58 (0.21 - 32.38)	0.7
	D				
Blood loss	A	2200	2200	1.00 (0.99 - 1.00)	0.14
	D	2100	3762		
Lowest systolic BP	A	70	15	0.94 (0.88 - 0.98)	**0.02**
	D	62.5	26.2		
Lowest MAP	A	45	10	0.90 (0.82 - 0.96)	**0.01**
	D	37.5	20		
Highest pulse rate	A	135	20		0.16

	D	135	17.5	0.98 (0.95 - 1.00)	
Lowest temperature	A	35	1.34	1.05 (0.64 - 1.72)	0.85
	D	35	1.14		
Lowest pH	A	7.14	0.09	< 0.01 (<0.01 - 0.04)	**0.01**
	D	7.05	0.11		
Highest lactate	A	7.01	2.53	1.06 (0.84 - 1.34)	0.64
	D	7.4	3.16		
Lowest base excess	A	-11.4	4.23	0.86 (0.74 - 0.98)	**0.03**
	D	-14.9	5.65		
Lowest HCO3	A	15.2	3.07	0.80 (0.64 - 0.96)	**0.03**
	D	12.7	3.82		

Table 9 - Multivariable model of preoperative variables

Variable	Odds ratio (95% CI)	*p* value
PRC in first 24hrs	1.01 (0.83 - 1.28)	0.9
FFP during surgery	1.17 (0.74 - 1.91)	0.52
Platelets during surgery	1.65 (0.71 - 5.92)	0.31
Lowest systolic BP	0.95 (0.88 - 1.00)	0.13
Lowest pH	<0.01 (<0.01 - 0.04)	0.06
Lowest base excess	1.52 (0.78 - 3.38)	0.23
Lowest HCO3	0.85 (0.28 - 2.09)	0.73

Table 10 – Indications for Damage Control Surgery

Physiological parameters
Hypothermia <35°C Acidosis pH <7.2 or base deficit >8 Coagulopathy Haemodynamic instability or profound hypoperfusion
Injury complexes
High energy blunt torso injury Multiple penetrating torso injuries Combined visceral and major vascular injuries Injuries across body cavities with competing treatment priorities

References

1. Trauma Surveillance in Cape Town South Africa: An Analysis of 9236 Consecutive Trauma Centre Admisssions. A, Nicol. 2014 Jun(6), JAMA Surg, pp. 549-56.

2. Notes on the arrest of hepatic hemorrhage due to trauma. JHV, Pringle. 1908, Vol. 48.

3. History of the Innovation of Damage Control for Management of Trauma Patients: 1902 - 2016. DJ, Roberts. s.l. : Ann Surg, 2017, Vol. 265.

4. Management of the major coagulopathy with onset during laparotomy. HH, Stone. 5, s.l. : Ann Surg, 1983, Vol. 197.

5. "Damage Control": An approach for improved survival in exsanguinating penetrating abdominal injury. MF, Rotondo. 3, s.l. : J Trauma, 1993, Vol. 35.

6. Damage control surgery in the era of damage control resuscitation. CM, Lamb. 2, s.l. : Br J Anaesth, 2014, Vol. 113.

7. Damage Control Surgery. CG, Ball. 6, s.l. : Curr Opin Crit Care, 2015, Vol. 21.

8. A Prospective Observational Study of Abdominal Injury Management in Contemporary Military Operations. IM, Smith. 4, s.l. : Ann Surg, 2015, Vol. 261.

9. Complications after 344 damage-control open celiotomies. RS, Miller. 6, s.l. : J Trauma, 2005, Vol. 59.

10. Damage Control: Collective Review. MB, Shapiro. 5, s.l. : Teh Journal of Trauma Injury, Infection and Critical Care, 2000, Vol. 49.

11. Indications, mortality and long-term outcomes of 50 consecutive patients undergoing damage control laparotomy for abdominal gunshot wounds. K, Twier. 2013.

12. Improving mortality in trauma laparotomy through the evolution of damage control resuscitation: Analysis of 1030 consecutive trauma laparotomies. B, Joseph. 2, s.l. : J Trauma Acute Care Surg, 2017, Vol. 82.

13. Damage control surgery: it's evolution over the last 20 years. BH, Waibel. 4, s.l. : Rev Col Bras Cir, 2012, Vol. 39.

14. Major abdominal vascular trauma - a unified approach. JL, Kashuk. 8, s.l. : J Trauma, 1982, Vol. 22.

15. Traumatic colon injury in damage control laparotomy - A multicenter trial: Is it safe to do a delayed anastomosis? LC, Tabete. 4, s.l. : J Trauma Acute Care Surg, 2017, Vol. 82.

16. Predictive Model for Survival at the Conclusion of a Damage Control Laparotomy. N, Aoki. s.l. : The American Journal of Surgery, 2001, Vol. 180.

17. Planned ventral hernia following damage control laparotomy in trauma: an added year of recovery but equal long-term outcome. BM, Zosa. s.l. : Hernia, 2016, Vol. 20.

18. Control the damage: morbidity and mortality after emergency trauma laparotomy. JA, Harvin. s.l. : The American Journal of Surgery, 2016.

19. Primary skin closure after damage control laparotomy. MJ, Pommerening. s.l. : British Journal of Surgery, 2014, Vol. 102.

APPENDIX

Appendix A - Human Research Ethics Clearance

UNIVERSITY OF CAPE TOWN
Faculty of Health Sciences
Human Research Ethics Committee

Room E53-46 Old Main Building
Groote Schuur Hospital
Observatory 7925
Telephone (021) 406 6626
Email: shureta.thomas@uct.ac.za
Website: www.health.uct.ac.za/fhs/research/humanethics/forms

19 June 2017

HREC REF: 404/2017

Prof P Navsaria
General Surgery
Trauma Unit

Dear Prof Navsaria

PROJECT TITLE: REVIEW OF DAMAGE CONTROL LAPAROTOMY (DCL) OUTCOMES IN A MAJOR URBAN TRAUMA CENTRE-(HMed-candidate Dr A Kruger)

Thank you for submitting your study to the Faculty of Health Sciences Human Research Ethics Committee.

It is a pleasure to inform you that the HREC **has formally approved** the above-mentioned study.

- Please update the protocol to reflect the Helsinki Declaration 2013.

Approval is granted for one year until the 30 June 2018.

Please submit a progress form, using the standardised Annual Report Form if the study continues beyond the approval period. Please submit a Standard Closure form if the study is completed within the approval period.
(Forms can be found on our website: www.health.uct.ac.za/fhs/research/humanethics/forms)

Please quote the HREC REF in all your correspondence.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please note that for all studies approved by the HREC, the principal investigator must obtain appropriate institutional approval before the research may occur.

Yours sincerely

signature removed

PROFESSOR M BLOCKMAN
CHAIRPERSON, FHS HUMAN RESEARCH ETHICS COMMITTEE
Federal Wide Assurance Number: FWA00001637.
Institutional Review Board (IRB) number: IRB00001938
This serves to confirm that the University of Cape Town Human Research Ethics Committee complies to the Ethics Standards for Clinical Research with a new drug in patients, based on the Medical