AN AUDIT OF ABDOMINAL VENA CAVA INJURIES IN AN URBAN TRAUMA CENTRE

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CONTENTS

DECLARATION

I, Dr Mark Ian Hampton, hereby declare that the work on which this dissertation is based is my original work and that neither the whole work or any part of it has been, is being, or is to be submitted for another degree in this or any other university.

Signature:. Signed by candidate

Date: 01 September 2013

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ABSTRACT

AN AUDIT OF ABDOMINAL VENA CAVA INJURIES IN AN URBAN TRAUMA CENTRE

Aim: To present the surgical management of injuries to the abdominal vena cava (AVC) and to identify clinical and physiological factors and management strategies that affect outcome.

Methods: A retrospective review of AVC injuries in patients attending the Trauma Unit at Groote Schuur Hospital from January 2003 to December 2011.

Demographic data, mechanism and agent of injury, level of injury, physiological parameters, associated injuries, trauma scores, management strategy, morbidity and mortality, and length of hospital stay were taken from the Trauma Unit's operative databank at Groote Schuur Hospital.

Results: Thirty-five patients with AVC injuries were identified. There were 33 penetrating injuries (94.29%) with gunshot wounds accounting for 28 of these (84.84%). There were 19 (54.29%) infrarenal, 9 (25.71%) juxtarenal, 3 (8.57%) suprarenal and 4 (11.42%) retrohepatic AVC injuries. Most patients were treated with AVC ligation (62.86%) and damage control surgery (71.42%).

There were 17 (48.57%) deaths.

There were significant differences in preoperative systolic blood pressure

(*p=0.0435*), number of red cell units (*p=0.001*), serum lactate (*p=0.0.0073*), arterial pH *(p=0.0015*) and preoperative temperature (*p=0.0004*) between survivors and non-survivors. There was also a significant difference in the management strategy used between the two groups (*p<0.0001*).

There was no difference in the applied trauma scores, level of injury and the number of associated injuries between survivors and non-survivors

Conclusion: AVC injuries are associated with high mortality. Patients presenting with clinical and physiological evidence of shock requiring damage control surgery appear more likely to suffer worse outcomes, particularly when multiple physiological derangements are present. Patients who demise often have severe associated injuries.

CHAPTER ONE

LITERATURE REVIEW

Introduction

Injuries to the abdominal vena cava are rare. They occur more commonly following penetrating (0.5 %– 5%) than blunt (0.6 % – 1%) trauma.[1]

These injuries are associated with a high mortality**.**The best survival rates reported in modern trauma centres are approximately33%. [1]

The incidence of trauma to the abdominal vena cava (AVC) appears to be increasing with some centres reporting that it constitutes up to 40% of all abdominal vascular injuries.[1]

Both general and trauma surgeons are occasionally faced with managing patients with major injuries to the AVC.

The management is usually challenging based on the fact that these injuries are rarely isolated and are often associated with serious adjacent solid and/or hollow visceral injuries as well as devastating neurovascular injuries. Patients are frequently haemodynamically unstable and require the utilization of multiple hospital resources including blood products, anaesthetic care, theatre time and often prolonged ICU stay which often involves organ supportive measures to ensure optimal outcomes.

General surgeons manage the majority of South African trauma patients as there are relatively few specialist trauma surgeons in our country. It has always been necessary for South African-trained general surgeons to be comfortable inmanaging all types of major abdominal vascular trauma.

The management principles of vascular trauma in haemodynamically unstable patients have evolved over the last 20 to 30 years. The concept of "damage control" surgery has become the guiding principlein conventional trauma practice. Complex time consuming anatomical repairs of major vascular injuries are foregone and temporary measures such as shunting, or non-corrective measures such as ligation are instead utilized. These techniques are relatively easy to execute and do not take much time.

The majority of patients with AVC injuries, currently, meet the criteria for damage control surgery, and ligation of the abdominal cava is a frequent treatment strategy. Although there is little doubt that this is a life saving measure, the associated morbidity after caval ligation has not been clearly elucidated. The incidence of deep venous thrombosis (DVT), chronic venous disease of the lower limbs and renal failure related to juxta- and supra renal AVC ligation is not known.

History of AVC injuries

The first report of a Vena Caval ligation was by Kocher, recorded in1883. Bilroth performed the procedure in 1885.[2] Ligation was performed for intraoperative caval injuries during surgery for malignant disorders in two patients. Both of these patients demised.

The first record of an infrarenal vena caval ligation with a successful outcome was by Bottini.[2]Detrie reported the first survivor after a suprarenal ligation[3].

By 1949 there were 136 reports of caval ligations in the literature.Not all of these procedures were performed for injuries to the AVC. Many were

electively performed in patients thought to be at high risk for pulmonary embolism in the era before therapeutic anticoagulationwas available.[2]

DeBakey et al reported the first large series of AVC injuries in 1978. They reported on 301 patients who had been identified with caval injuries over the preceding 30 years. The majority (234) were treated with repair while only 32 received caval ligation. Initial mortality rates in the 1950's approached 100%. [4]

AVC ligation was an accepted practice in selected patients during the Second World War. However, military data from the Vietnam Vascular registry in the 1970's showed an increase in lower limb oedema and vascular insufficiency and a trend away from AVC ligation began. AVC ligation became more popular once again towards the end of the 1980's and early 1990's with the advent of damage control surgery, backed up by civilian series that showed acceptable limb salvage and short-term outcomes with caval ligation.

"Damage Control Surgery" was widely adopted and applied from 1993.[3] The concept of damage control resides on the fact that a patient who has sustained a major polytrauma has not only suffered from anatomical damage, but has also undergone a major physiological insult. A cascade of pro- and anti-inflammatory mediators are released at the time of trauma and the patient undergoes major changes in the coagulation, complement and immune systems in conjunction with the anatomical injuries which may be present. Patients in this situation are highly susceptible to hypothermia due to prolonged exposure to the elements, breakdown of tissue barriers, resuscitation with cool fluids and blood products and during surgical exploration and exposure of body cavities.

Patients experiencing major polytrauma are often shocked for prolonged periods. This equates to tissue hypoxaemia as a result of an inadequate delivery of oxygen (DO_2) to tissue. The consequence of this is anaerobic respiration of the tissues with production of excess hydrogen ions and a metabolic acidosis.

Hypothermia and acidosis present in a polytrauma patient in whom the inflammatory cascade has been initiated leads to what is known as the "Coagulopathy of Trauma Shock".[5] In historical trauma series, this entity was not fully appreciated.The focus of trauma surgery in polytrauma patients was on performing anatomically corrective repairs to damaged structures irrespective of whether there was any evidence of coagulopathy[6]. Current trauma practices encourage the early identification of patients at risk of coagulopathy to enable appropriate selection for the utilization of damage control measures. These are as follows:

- Control ofbleeding by simple repairof vital vessels or ligating vessels that can be sacrificed, packing or resection of solid organs, and packing of non-surgical bleeding sites
- Restoring organ / extremity perfusion by simple repair or temporary shunting of vessels, with or without a fasciotomy of the involved limb when these are operated upon.
- Limit contamination by temporarily tying off or stapling the ends of injured bowel, and debriding and removing devitalized tissue
- Protecting the abdominal contents with an open abdomen dressing
- Transport of the patient to ICU for correction of the physiological insult and coagulopathy

- Relook laparotomy once the coagulopathy has been corrected for definitive anatomical repairs and formal abdominal closure.[6]

In a series by Feliciano et al, outcomes of patients with vascular injuries were evaluated in the periods before and after adoption of damage control measures.[3] All injuries, with the exception of aortic and AVC injuries, showed an improved survival after the implementation of these measures.

Ligation of the AVC is now an accepted practice in patients who meet the criteria for the implementation of damage control measures because of major physiological derangements.

Recent series by large trauma centres report that a high percentage of patients with inferior vena cava injuries are being managed with ligation. Navsaria et al managed 63% of patients with caval injuries in this manner[7]. Feliciano et al reported a ligation rate of over 40% in their large series of caval injuries.[3] This is in contrast to earlier series in the pre-"damage control" era where more repairs andfewer ligations were performed. In Davis et al's review of vascular trauma it was noted that AVC ligations increased from 15.1% in the period from 1989 to 1992 (pre-"damage control"era) to 27.4% in the period from 1993 to 1998.[8]

Anatomy of the Abdominal Vena Cava

The AVC is the largest vein in the body. It has no valves and originates at the L5 vertebral body by the confluenceof the right and left common iliac veins. At this point it lies posterior to the right common iliac artery and about 2.5 cm to the right of the midline and aorta. It returns blood from the lower limbs, most

of the abdominal wall and abdomino-pelvic viscera to the right atrium. It ascends on the right psoas muscle to the right of the midline and passes through the vena caval hiatusin the diaphragm at the level of T8. After a short supraphrenic course it pierces the fibrous pericardium and enters the inferior part of the right atrium.

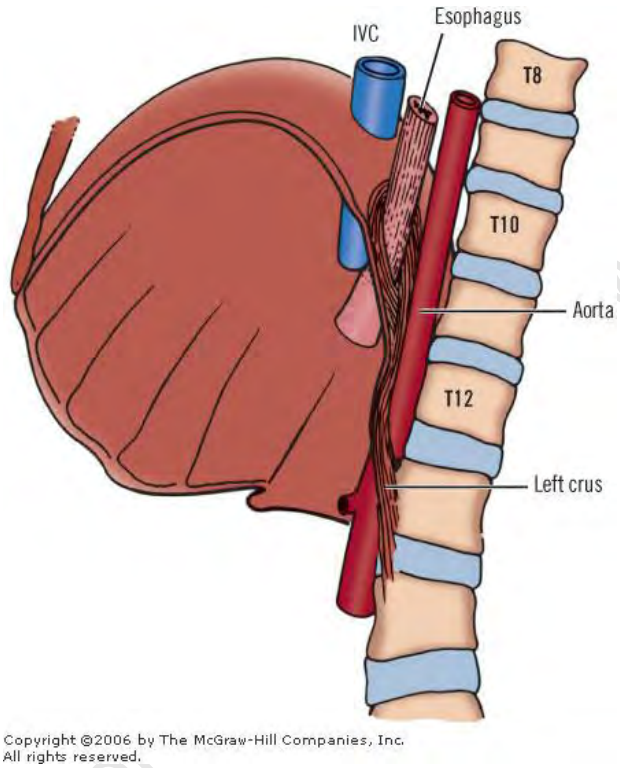

Figure 1. Posterolateral relations of the AVC at the Diaphragmatic hiatus[31]

As the AVC ascends to the vena caval hiatusits posterior relations include the right psoas muscle, the right sympathetic trunk, the right renal artery, the right adrenal gland, the right celiac ganglion, and the right diaphragmatic crus.

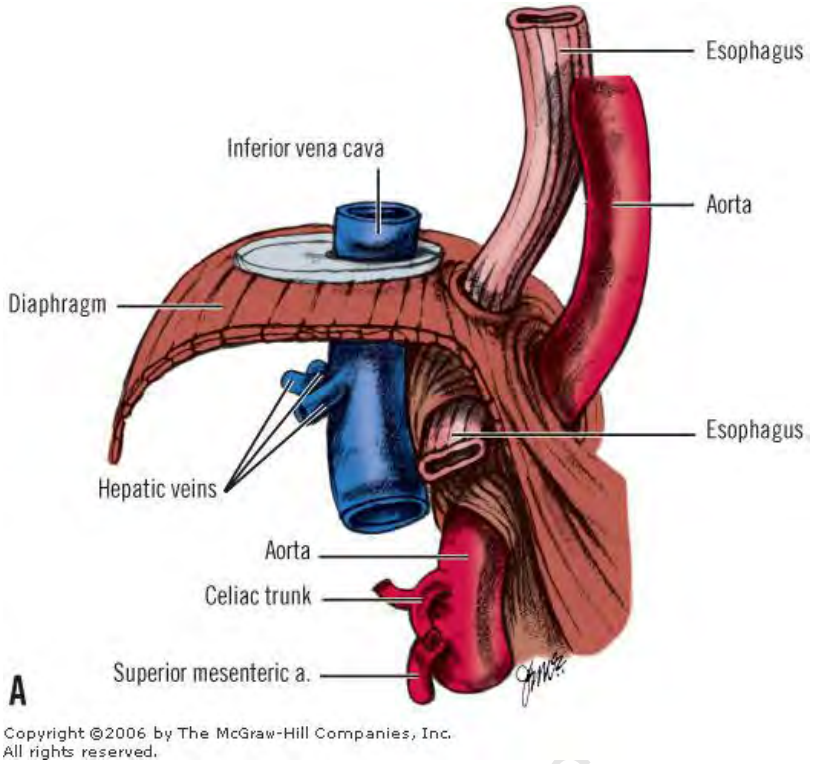

Figure 2. Medial relations of the AVC at the Diaphragmatic hiatus[31]

The anterior relations of the AVC as it ascends include the peritoneum, the superior mesenteric vessels in the root of the mesentery, the third part of the duodenum, the head of the pancreas with the bile duct and portal vein in between. As the AVC courses above the duodenum, it lies posterior to the foramen of Winslow. Thereafter it enters a groove in the posterior surface of the liver between the right and caudate lobes. To the right of the AVC lies the right ureter and kidney and the second part of the duodenum. To its left lies the aorta.

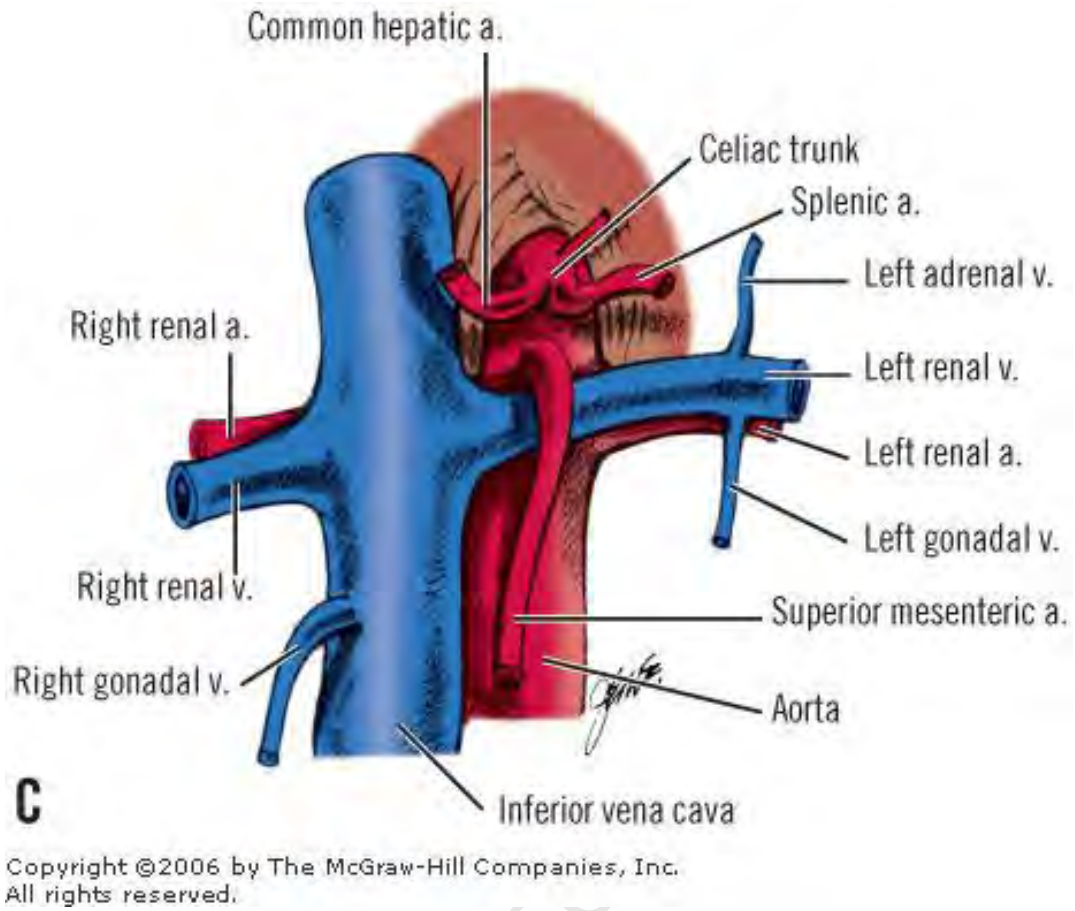

Figure 3. Medial vascular relations of the AVC at the level of the renal veins[31]

There are 7 major tributaries to the AVC. These include

- The right and left common iliac veins which form the AVCat the level of L5
- The 3rd and 4th lumbar veins which are segmental and communicate with the ascending lumbar veins and vertebral venous plexus
- The right gonadal vein
- The right renal vein that drains in to the AVC at the level of L2. It lies anterior to the right renal artery
- The left renal vein that originates in the hilum of the left kidney at the level of L2. It passes anterior to the left renal artery andcrosses

theaorta just inferior to the origin of the superior mesenteric artery to join the AVC just to the right of the midline. It receives the left gonadal and adrenal veins just after its origin.

- The azygous vein connects the inferior vena cava to the superior vena cava. It commonly arises from the posterior aspect of the AVC just above the level of the right renal vein, but may begin as a continuation of the right subcostal vein or from the junction of this vein and the right ascending lumbar vein. The azygous vein enters the thorax via the aortic hiatus. It anastomoses with the inferior hemiazygous vein in the chest which commonly arises from the posterior surface of the left renal vein
- The right inferior phrenic vein drains blood from the abdominal surface of the right hemidiaphragm and usually drains directly in to the AVC
- The hepatic veins are short vessels which drain into the AVC just as it passes through the vena caval foramen in the diaphragm

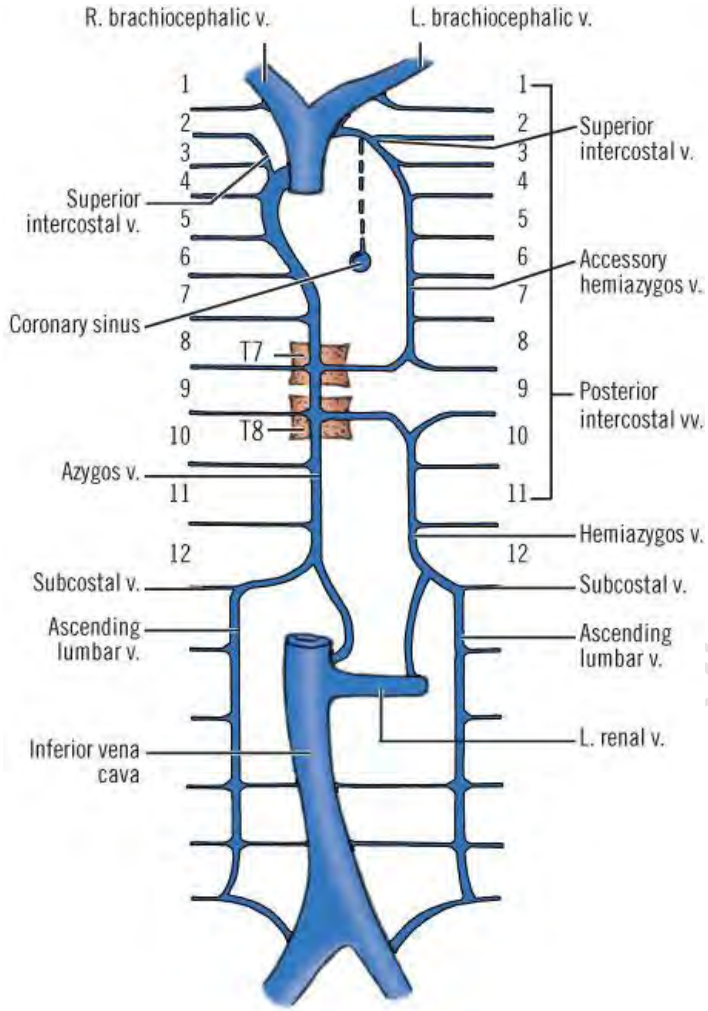

Figure 4. Azygous venous network[31]

There are 3 potential collateral pathwaysfor venous blood to reach the right atrium when the AVC becomes obstructed.[20]

These include:

- The superficial and inferior epigastric veins in theanterior abdominal wall. Blood drains into the thoracoepigastric veins, superior epigastric veins and in to the superior vena cava via the intercostal and subclavian veins.
- The lumbar venous tributaries that drain into the vertebral venous plexus of veins.

- The superficial abdominal and thoracic veins that connect the superficial circumflex iliac veins to the axillary veins.

In occasional circumstances, congenital vena caval anomalies may pose difficulties to the management of injuries to it. There are 3 major congenital AVC anomalies. These are:

- AVC absence in the intrahepatic segment due to failure of the right subcarinal vein tributaries to connect with the hepatic veins in the 7th gestational week.
- AVC duplication with a persistent left sided embryonic cava, which is the most common anomaly. During embryological development there are two vena cavae, one each on the right and left side.

The right embryonic cava persists as the mature inferior vena cava whereas the left embryonic AVC should degenerate. The left sided embryonic cava may persist as a large or small vessel that originates at the left common iliac vein and joins the left renal vein.[21]

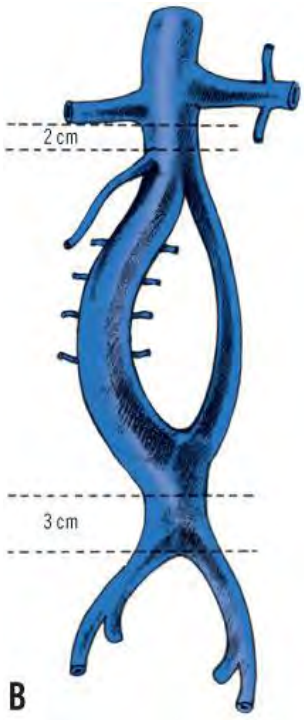

Figure 5. AVC duplication[31]

- Pre-ureteric AVC that occurs on the right side only. The right ureter passes behind and around the AVC and may become obstructed in this position.[22]

Clinical Presentation and Diagnosis

Patients with abdominal vascular injuries, including those with AVC injuries, may present in one of 2 ways; those who are haemodynamically stable and those who are unstable.

In stable patients with AVC injuries, the implication is that the injury to the cava has undergone tamponade in the retroperitoneal space and there is no

active bleeding. Exploratory laparotomy may be indicated in these patients for signs of peritonitis due to associated injuries, or for deterioration in clinical abdominal signs over time, at which time the AVC injury is uncovered during exploration of the retroperitoneal haematoma. Alternatively, in stable patients with no immediate indication for laparotomy, but where an indication for a contrasted Computed Tomography of the abdomenexists, a caval injury could be identified.

Unstable patients can be divided into those who respond transiently to fluid resuscitation and those who do not respond at all. Laparotomy is immediately indicated once the source of bleeding has been localized to the peritoneal cavity. This may be clinically obvious in cases of penetrating trauma but in cases of patients with blunt polytrauma the site of haemorrhage may be elsewhere such as either the right or left chest. Emergency department diagnostic protocols such as Focused Abdominal Sonar in Trauma (FAST) or Diagnostic Peritoneal Lavage (DPL) can help to localize the site of bleeding to the abdomen.[23] In the absence of an adequate tamponade,active bleeding will be encountered on entering the abdomen at the site of the caval injury.

The most common clinical scenario in which the trauma surgeon encounters an AVC injury is where a retroperitoneal haematoma is encountered during laparotomy.[3,7]The surgeon is well advised to institute strategies whereby haemostasis will be optimized and blood loss will be minimized once the haematoma is explored. Injudicious medial rotation of the right colon before haemostatic adjuncts are instituted could lead to abrupt exsanguination and intra-operative death. The site of injury to the AVC is almost always obscured from vision by active bleeding, making repair virtually impossible without some

means of haemorrhage control. The key to all successful venous repairs relies on adequate controlof bleeding while the repair is being performed, adequate exposure of the operative field,and visualization of the edges of the damaged vein. Techniques include the utility of swab-holding forceps to apply caval compressionproximal and distal to the siteof injury. The use of a side-biting Satinsky clamp is often very useful. The segment of the AVC containing the injury is placed within the jaws of the instrument, thereby isolating the injury. Repair of the injury can then be performed whilst the injury is within the jaws of this instrument. Additional adjuncts to control of haemorrhage include isolation of the proximal and distal AVC with vascular clamps and cross-clamping of the supra-coeliacabdominal aorta.[24]

Management

There are essentially two definitive methods of managing AVC injuries that could form part of the surgical strategy in dealing with multiply injured patients. These include primary repair of the injury and ligation of the abdominal cava.

Strategies used in managing patients with multiple abdominal and extra abdominal injuries include:

- Definitive surgery: all injuries are definitively treated and no further surgery is needed
- Damage control surgery: definitive anatomically corrective repairs are foregone and basic life, and limb, saving techniques are used.Repeat surgery to carry out definitive repairs is planned when the patient's physiological profile has improved.

Occasionally adjuncts to standard damage control techniques are deemed necessary by the operating surgeon. These are more likely to be used when dealing with injuries to the retro hepatic cava and hepatic veins. In an unpublished series, Navsaria et al reported one survivor of a retro hepatic AVC injury using perihepatic packing to achieve tamponade. Other techniques include atriocaval shunting and thoracotomy.

In a 10-year review of 47 consecutive AVC injuries by Hansen et al, 10 patients were found to have received emergency department thoracotomies(EDT). All but one demised.[11] Kuehne et al reported that 25 patients in their series of 158 patients with AVC injuries required EDT's with aortic cross clamping for profound hypotension. All but onedemisedmaking the use of EDT statistically predictive of a mortal outcome.[9] This outcome has been supported by the findings of Asensio et al in their review of their series of patients with all vascular trauma. They reported survival rates following EDT ranging from 2 to 10%.[1]

There are varying reports in the literature evaluating whether the management strategy used affects the outcome of the patient. In Feliciano's series of 100 patients with AVC injuries the subgroup of infrarenal injuries (the largest subgroup comprising 51 patients) was analyzed to detect differences between the patients who were treated with caval ligation and those whose AVC injuries were primarily repaired. There was significantly higher use of packed red cells and a higher overall mortality in the group receiving ligation of the cava. There was also a trend towards shorter length of ICU and hospital stay and lower ISS score in those patients managed with primary repair.[3]

This perhaps suggests that the patients receiving AVC repair were less likely to be as severely injured as those receiving ligation.

These findings are in contrast to those reported by Navsaria et al. In their series 48 patients with AVC injuries a statistical correlation between ligation or repair and outcome was not found.[7]In this series, none of the patients being managed with repair of their caval injury had complex venous repair or patch venoplasty. It seems likely that the outcome of patients with this injury is determined more by the pre-operative physiological status rather than the method used to manage it.[3]

Prognostic indicators

The primary outcome measure against which all management strategies of AVC injuries are judged in the trauma literature is mortality. Secondary outcome measures include hospital and ICU length of stay and complications of management.

Demographic, clinical, physiological and management variables have been assessed as well as injury-specific variables including the mechanism of injury, the level of injury and the presence and severity of associated injuries.

Analysis of demographic data in the majority of reports in the literature shows that the profile of the patient most likely to be treated for an AVC injury is a young adult male.[3,7,9,10,11]

Mechanism of Injury

AVC injuries can be due to penetrating (stab, gunshot or shotgun wounds) or blunt (pedestrian or passenger vehicle accidents) mechanisms.

Most series report that penetrating injuries are significantly more common than blunt,[3,7,10,12] with gunshot and shotgun wounds causing more damage and being more likely to lacerate the AVC. [1,13]

Most of the more recent series report that of all penetrating wounds, most are caused by low velocity gunshots [3,7,12].Clarke et al published a comparative series that evaluated two separate time periods ten years apart. They evaluated how the mechanism of AVC injury had evolved over time and found that significantly more injuries were caused by firearms and significantly less injuries were caused by stab wounds in the later period.[14] Davis et al. reported a higher incidence of intra operative exsanguination from penetrating AVC injuries in the period from 1993 to 1998 (9.5%) when compared to the period from 1989 to 1993 (1.9%). They concluded that this was due to the fact that the firearms being used by perpetrators of interpersonal violence where becoming more available, more powerful and capable of more extensive damage over time.[8]

It appears that blunt injuries, which are more likely to be associated with a retrohepatic level of AVC injury and also with injuries to the liver and or the hepatic veins, have a higher attendant rate of mortality than penetrating injuries.[10] This is likely due to the higher energy transfer that occurs during a motor vehicle accident where the number of intra- and extra abdominal injuries is greater.

Associated Injuries

The number, type, and severity of injuries associated with AVC trauma and their influence on outcome have been evaluated in various ways.

Various trauma scores have been used in polytraumapatients in an attempt to determine the likelihood of mortality and morbidity with a given set of injuries. The trauma scores commonly used in series of AVC injuries include the Revised Trauma Score (RTS), the Penetrating Abdominal Trauma Index (PATI) and Injury Severity Score (ISS).

The Revised Trauma Score (RTS) is a physiological system that aims to predict the probability of survival in trauma patients. It uses the GCS, systolic blood pressure and respiratory rate as the only variables. Each variable is multiplied by aweighted constant, with the GCS-constant making the contribution from the GCS to the overall score the most influential variable.

An RTS score of 4 correlates to a probability of survival of 60.5%, while a score of 3 denotes a probability of 36.1%.[15] Although the RTS has been validated, the fact that it is so heavily influenced by the GCS makes its application to severe traumatic injuries where a head injury has not occurred, less appropriate. Many trauma patients with severe injuries have been sedated, intubated and ventilated by paramedic staff prior to arrival at a trauma unit, which makes accurate determination of the GCS difficult. In a deeply sedated patient who is being ventilated, the determination of respiratory rate is also not accurate.

The PATI score was founded by Moore et al in 1981. This system was developed to quantify the risk of post-operative complications in trauma patients. The PATI score examines 14 intra-abdominal organs and a score of 1 to 5 is assigned according to the severity of each injured organ. The score is then multiplied by a risk factor of 1 to 5 according to the specific organ that is injured based on the influence on survival that an injury to that particular organ would have. The final score is obtained by summing the individual organ scores. Scores of greater than 25 are associated with a postoperative complication rate of approximately 50%. PATI scores are limited by the fact that only one injury score per organ can be assigned and the impact of less severe, but no less serious injuries, are at risk of being ignored.[16]

The ISS is an anatomical scoring system that provides an overall score for patients with multiple injuries. Each injury is assigned an Abbreviated Injury Scale (AIS) score and is allocated to one of 6 body regions (Head, Face, Chest, Abdomen, Extremities including the pelvis, and external). Only the highest AIS in each body region is used and only the 3 most severely injured body regions are used in the calculation. Scores in each region are squared and summed to produce the ISS. The ISS aims to predict likelihood of mortality and correlates to patient ages at which the mortality rate for a given score is more than 50%. For example, for patients between the ages of 15 and 44, an ISS of 40 corresponds to a likelihood of death of 50%, while for patients aged 65 and over, an ISS of 20 corresponds to the same likelihood. The weaknesses of the ISS are that the impact of less severe injuries are not

considered in the overall risk to the patient, and that different combinations of injuries can produce the same ISS.[17]

The clinical utility and predictive value of these scores have been assessed in 2 recent reports. Feliciano et al reported that patients with infrarenal injuries had significantly lower ISS scores compared to patients with injuries to the AVC at more proximal levels, and that those with infrarenal injuries were more likely to have higher RTS scores than the other groups.[3] Navsaria et al found that there was no difference in the RTS score nor the PATI score between survivors and non survivors, but there was a significant difference in the ISS scores between these groups, with those surviving having lower scores.[7] Kuehne et al similarly reported a statistically significant difference in the ISS scores of survivors and non-survivors in their series of 158 patients with AVC injury.[9]

Navsaria et al reported that the mean number of associated injuries was 3.1 in their series.These injuries most commonly involved the liver, small bowel (jejunum and ileum), duodenum and large bowel.[7] Rosengart et al found that the mean number of associated injuries in survivors of AVC injuries was three and in non survivors, four. Eighty per cent of patients with four or more associated injuries did not survive, while there was a mortality of 33% for those with three or less associated injuries.[10]

However, later series did not find statistically significant evidence of this.[3]

The effect of associated intra abdominal vascular injuries on outcome have been evaluated in a number of series. Degiannis et al found that the number of associated intra abdominal vascular injuries had a direct influence on

mortality.[18] Kuehne et al similarly reported that associated injury to the abdominal aorta was a significant predictor of outcome.[9]

Level of Injury

The level of AVC injury can be classified in various ways, the simplest method being to separate them using the level of the renal veins as the point of referencei.e. infra- and suprarenal injuries. Most series have, however subdivided AVC injuries into infrarenal, juxtarenal (injury within 2.5 cm above or below the renal veins)[13], suprarenal (a short segment of the AVC between a point 2.5 cm above the right renal vein and the right adrenal vein) and retro hepatic (between the right adrenal vein and inferior phrenic vein).[13] Using the simplest division, infra- vs. suprarenal, it is clear from some series that suprarenal AVC injury carries a worse prognosis.[7,10,18] This is largely due to the high concentration of vital vascular and other structures in more proximal locations as well as attendant technical difficulties in obtaining adequate surgical exposure in these areas. The fact that trauma to the AVC in these proximal locations is more likely to be dueto either blunt injury or high energy projectile injury, where energy transfer and surrounding tissue injury is more extensive, also contributes to the higher mortality rate seen in proximal AVC injuries. Feliciano et al reported that retro hepatic injuries had a significantly worse outcome than injuries at other levels.[3] Kuehne et al found that survival was highest in patients with infrarenal injuries and that mortality was highest in patients with retrohepatic injuries. This was attributed to the fact that the

AVC is readily surgically accessible which simplifies exposure and facilitates prompt vascular control.[9]

In contrast to the above, Navsaria et al did not find a statistical correlation between survival and the level of injury, even after reclassifying patients in their series into the two basic categories of infra- and suprarenal levels.[7]

Systolic Blood Pressure

Many of the published series of AVC injuries have evaluated physiological variables as predictors of outcome. Systolic blood pressure on arrival in the emergency department, haemoglobin level as well as arterial blood gas parameters including lactate have been assessed.

Many authors have shown a significant difference between survivors and non-survivors regarding the presence of preoperative hypotension. This was reported by Buckman et al, Kuehne et al as well as by Hansen et al.[11,9] Kuehne et al also reported a significant difference between survivors and non survivors regarding the lowest preoperative systolic blood pressure recorded.[9] Navsaria et al reported a trend towards preoperative hypotension in the group of non-survivors and Feliciano et al showed a trend towards significant preoperative hypotension in the subgroup of patients with infrarenal injuries who received caval ligation, the subgroup of patients who were more severely injured.[3,7] Degiannis et al reported that persistent shock significantly predicted a mortal outcome.

Acidosis, Lactate and Base deficit

Arterial blood gas measurements are often used in the evaluation of trauma patients in the emergency department to help quantify the extent of tissue hypoperfusion that the patient is being subjected to. The presence of severe acidosis accompanied with other parameters such as hypotension or hypothermia could influence the surgical management of the patient and a strategy of damage control would often be chosen under these circumstances. Variables including arterial pH, base deficit and lactate have been evaluated in various series as independent predictors of outcome. Base deficit, bicarbonate level and pH were assessed between survivors and non-survivors in the series by Hansen et al.[11] No significant difference was found between the groups regarding base deficit and pH but a difference was found in the bicarbonate levels. Kuehne's group did find statistical significance in pH values between survivors and non-survivors. The mean pH for survivors was 7.23 while the mean for non-survivors was 7.04.[9]

Nicol et al evaluated the effect of pH on outcome in severely injured patients requiring damage control treatment. They found a linear correlation between a decrease in pH and the rate of mortality. At a pH of 7.0 the mortality in this series was approximately 50%.[19] Feliciano et al reported no statistical difference between survivors and non-survivors when base deficit was assessed.[3]

Rosengart et al analyzed base deficit and lactate levels in their series of 37 patients with AVC injuries and found a trend towards higher measurements in non-survivors although this was not statistically significant. They did find,

however, that a base deficit and lactate level of 4 or less accurately predicted survival.[10]

Hypothermia

Together with acidosis, hypothermia is a key component that leads to the coagulopathy seen in patients with severe injuries.[5]Temperature has been evaluated in some series of AVC injuries to ascertain whether the presence and degree of hypothermia could independently predict outcome. Kuehne et al found that the average temperature during surgery differed significantly between survivors and non-survivors. Survivors had an average temperature of 35.1°C whereas non-survivors averaged 33.3°C.[9] Nicol et al evaluated the effect of core temperature on outcomes in patients requiring a damage control procedure. They found a linear relationship between a decrease in core temperature and mortality up to a point where the core temperature reached 32°C. When the core temperature dropped below this, the mortality rate went up exponentially. They concluded that below a core temperature of 32°C, survival is unlikely.[19]

Packed Red Cell Requirement

The number of packed red cells that a patient requires during the first 24 hours of management has been assessed as an independent factor for outcome in patients with AVC injuries. It seems intuitive that the more blood products a patient needs, the more severe their injuries and the worse their outcome would be. Rosengart et al found a trend towards increasing mortalityin patients who received 20 or more units of blood with a mortality

rate of 93% in this group, compared to 30% for patients who received less than this.[10]

Kuehne, Hansen and Navsaria reported non-survivors of AVC injuries were likely to require significantly more units of red cells than survivors.[7,9,11]

Mortality

Deaths in patients who have been subjected to major abdominal trauma where damage control principles have been applied may be assessed as being due either to haemorrhage occurring at the time of the index procedure, coagulopathy occurring in the first five days after the index procedure, sepsis if death occurred later than this, or due to a treatment decision to withdraw care.

Injuries to the AVC carry a high associated mortality. This is often due to the injury to the cava itself, which in its own right is often lethal. The surgeon treating a caval injury may be unable to control haemorrhage from the damaged vein with subsequent exsanguination and intra-operative death. In situations where the caval injury can be controlled, death may still result from associated injuries. These could be other vascular injuries where the patient demises because of failure to control haemorrhage during the index procedure.Approximately 10% of patients with AVC wounds have a second major vascular injury, usually to the aorta or portal vein. Various reports have examined the influence that cumulative abdominal vascular injuries have of outcome. Graham et al reported a mortality rate of more than 50% with more than one associated injury and 75% in the presence of more than two.[4] In the large series by Feliciano et al there was no significant difference in the

number of associated vascular injuries in patients with penetrating AVC injuries between the group managed with repair versus the group managed with ligation.[3]

The presence of an associated liver injury was assessed to determine whether the presence thereof influenced the likelihood of death due to haemorrhage in this series. A statistically significant association was not found, but the relationship tended to approach significance (p=0.08)

Patients who survive their index procedure may still develop the coagulopathy of trauma shock (ACOTS)[5], often precipitated by the combination of hypothermia and acidosis in a polytrauma patient.[5] This problem would present as ongoing “non surgical” bleeding, which refers to bleeding from damaged peritoneal surfaces, injured solid organ parenchyma and sites of visceral repairs. It is largely managed by correction of physiologic parameters including temperature and pH, as well as targeted administration of blood products. Death that occurs in the first two to five days after an index damage control procedure is largely due to this coagulopathy.

Patients who successfully undergo damage control surgery and who have either avoided or survived the coagulopathy of trauma shock are still at risk of mortality. Deaths that occur after a period of five to seven days has passed, usually while the patient is still in ICU, are often due to sepsis. Sepsis may be a direct surgical complication or may be an acquired problem secondary to critical illness.

Lastly, there is a small subset of patients in whom death is the result of a decision by the critical care and trauma teams to withdraw supportive therapy. This situation is commonly encountered in patients who are not coagulopathic or septic, but in whom brain death has occurred.

Special Considerations

1. Laparoscopic repairs
2. Emergency stenting of the AVC
3. Temporary balloon occlusion of the AVC
4. Retrohepatic caval injuries

   -atriocaval shunt

   -packing

   -stent-graft for traumatic aortocaval fistulae

   -deep hypothermic arrest on bypass

Laparoscopic AVC repairs

There are some experimental reports of laparoscopic techniques used for the repair of AVC injuries. These techniques include suturing and the application of Chitosan based haemostatic dressings. The role of laparoscopy in trauma is expanding but is as yet ill-definedin the management of traumatic vascular injuries. In the setting of iatrogenic caval injury during elective laparoscopic surgery it would seem feasible to perform a laparoscopic-based repair if the surgeon has the necessary skill to do this.[25]

Emergency stenting of the AVC

The role of endovascular techniques for management of caval injuries has been described in 3 case reports.[26] All have been described in the setting of an iatrogenic caval injury during elective surgery. One such report describes the use of balloon occlusion-isolation of a segment of injured inferior vena cava during a posterior lumbar interbody fusion at L3-4. An iatrogenic caval perforation was detected during on-table fluoroscopic screening performed for a suspected venous injury. After the insertion of balloon occluders placed in to the cava proximally and distally to the injury through the lumbar incision, the patient was turned supine and taken to the endovascular suite where a 44mm thoracic aortic stent-graft was introduced via a transfemoral approach and placed to cover the caval tear. There was a good technical result and no subsequent endoleaks were reported. The patient recovered well.[26]

There are some major limitations to this technique:

- It is only feasible in haemodynamically stable patients who can tolerate time consuming venographic studies
- The equipment and stent-grafts used are not vein-specific
- Follow up protocols after stent-grafting are not defined
- The risk of progressive caval narrowing and subsequent thrombo-embolic events is not quantified

Temporary balloon occlusion of the AVC

The use of temporary balloon occlusion of an injury to the Abdominal Vena Cava (AVC) has been described in a case report by Bui et al. They described this technique in a stable patient with a penetrating injury to the infrarenal

AVC following a single abdominal gunshot wound. The patient was investigated pre-operatively with a Computed Tomogram (CT) of his abdomen which revealed a large right-sided retroperitoneal haematoma with contrast extravasation from the AVC. The patient was taken to theatre immediately and had percutaneous placement of 7 French sheaths into both femoral veins. Contrasted venography was then performed to confirm the AVC injury. The right-sided sheath was exchanged for a 14 French sheath and a 27mm occluding balloon (Reliant, Medtronic, MN) was introduced into the AVC and inflated across the injury. Midline laparotomy was then performed. The retroperitoneal injury was entered without the risk of haemorrhage. The injury to the cava was clearly identified and repaired with simple suturing. The patient was discharged after a short duration of stay. There were no complications during subsequent follow up.[32]

The role of endovascular therapy for caval injuries isill-defined. Although unlikely to play a role in the management of the unstable trauma patient, there is perhaps a role in the management stable patients with AVC injuries.

Retrohepatic caval injuries

Very few injuries demand as much respect as injuries to the retrohepatic vena cava and hepatic veins. This segment of the AVC is extremely delicate, difficult to access and to expose adequately, and difficult to treat. Inan actively bleeding unstable patient this becomes even more challenging.

Patients with these injuries can either present intraoperatively with exsanguinating haemorrhage or with a stable haematoma.

There is no debate that in patients who are exsanguinating from a retrohepatic caval injury some form of active management must be undertaken to save the life of the patient whether this entails perihepatic packing, attempted suture repair, atriocaval shunting or some other form of treatment. Some controversy does exist over what to do if one encounters a stable retroperitoneal haematoma. The concerns one would have in opening a stable haematoma in this area would be the probability of provoking a major life-threatening haemorrhage as well as the risk of air embolism. The disadvantage of opting to leave the retrohepatic haematoma alone and observe it would be the risk of spontaneous disruption of the haematoma at a later stage with exsanguinating haemorrhage in an uncontrolled situation.[9]It is not known what the risk of this scenario is but it is thought to be uncommon.A case report by Khan et al describes the conservative management of a stable patient with a penetrating gunshot wound through the retrohepatic vena cava.[27]

The options for active management of retrohepatic caval injuries include packing, atriocaval shunting and temporary total hepatic isolation with repair.[9] These techniques are controversial but there are reports of some successful outcomes with their use.

In order to obtain the best exposure for these techniques, andtohave the best chance of success, the abdominal incision should be extended to a right thoraco-abdominal or median sternotomy incision and the hepatic ligaments should be divided.

Total temporary hepatic isolation involves the placement of 4 vascular clamps onto the AVC above and below the injury, the portal triad and the supra-celiac aorta. This allows one some time to expose the injury to the retrohepatic cava and repair it. The risks of hepatic isolation are sudden precipitous disruptions in venous return to the right heart and ischaemic injuries to the kidneys, liver and gut.

The atriocaval shunt involves the placement of a 32F intercostal chest tube or 9 mm endotracheal tube down the inferior vena cava through a purse string in the right atrial appendage. The cuff is inflated above the level of the injury and Rummel tourniquets are placed superiorly above the supra-diaphragmatic cava and inferiorly around the abdominal cava. This allows the surgeon the opportunity to repair the injured cava. This technique has many critics but there are some reports of patients having done well when this technique was appropriately applied.[9]

Mattox et al reported a series of 31 patients treated with atriocaval shunting for retrohepatic AVC injuries in which the overall survival for the procedure was 19%. Factors that were uniformly associated with a mortal outcome were the need for thoracotomy, the need for hepatic resection or where there were technical problems with the shunt.[28]

Perihepatic packing with or without other adjuncts such as Foley catheter or Sengstaken tubes for further tamponade, with delayed exploration is likely to be the most commonly used damage control technique for retrohepatic caval injuries. It can provide temporary control of haemorrhage and allow correction of the patient's physiological derangements in the ICU if the tamponade effects of the packs are adequate to control bleeding. It also allows the

opportunity to perform selective hepatic angio-embolization for control of associated arterial bleeding. On return to theatre for pack removal definitive repair of the retrohepatic caval injury may be undertaken in a much more controlled environment.[9]

Waldrup et al have described the use of aortic stent-graft for the exclusion of a traumatic supracoeliac aortocaval fistula in a patient with penetrating thoraco-abdominal trauma. This injury was not initially identified and was diagnosed with an arteriogram on day 19 post initial laparotomy.This was requested to for the investigation of resistant abdominal compartment syndrome and an ongoing fall in haematocrit.

An AneurRx aortic extension cuff was advanced under fluoroscopic guidance and deployed over the fistula. Completion angiography showed that the fistula had been obliterated. The stent was patent after one year follow up.[33]

The precise indication for aortic stent-grafting for traumatic aorto-caval fistulae remains ill-defined, but it may offer an attractive alternative for the management of proximal injuries that would otherwise necessitate open surgery in difficult circumstances.

Controversies and concerns

1. Use of prophylactic lower limb fasciotomies for caval ligations
2. Use of anticoagulation after AVC repairs and ligations

Use of prophylactic lower limb fasciotomies for caval ligations

The issue of whether to perform prophylactic bilateral lower limb fasciotomies in cases of AVC ligation is controversial with no clear evidence-based

guidelines in the literature. No authors have recommended routine use of this procedure, but some have recommended its selective use.[3] However, in Navsaria et als series in which 40% of AVC injuries were managed with ligation, no patient received a fasciotomy and no subsequent adverse effects were recorded. In another more recent unpublished series by the same author where 61% of AVC injuries were treated with ligation; again, not one patient required a fasciotomy.[7]

Proponents of a more aggressive approach to the use of fasciotomies argue that the risk of lower limb compartment syndrome occurring secondary to acute obstruction of lower limb venous return, coupled with the generalized capillary leakage secondary to the major physiological insult that the patient has undergone, warrants the utilization of the procedure.[3] Some have used measurement of lower limb compartment pressures to guide the use of fasciotomy, while others have used their clinical impression of raised compartment pressure only.[3]

Opponents of the liberal use of fasciotomy have sited large series in the literature in which fasciotomy has not been at all necessary. An added concern is that once the lower limb has been subjected to the chronic venous hypertension which is an inevitable consequence following AVC ligation, delayed or non-healing of the fasciotomy wound could become a significant issue.

The most recent recommendations regarding the use of selective lower limb fasciotomies comes from Feliciano`s group who recommend that fasciotomy should be done where:

- The compartment pressure in the lower limb is more than 25 mmHg

- Hard, non-compressible compartments have been identified[1]

In these cases bilateral 4 compartment lower limb fasciotomies should be done. In this series, for those patients who had received lower limb fasciotomy, delayed primary closure was possible in 40%, partial closure with skin grafting in 10% and no closure with skin grafting in 20%. Thirty per cent of patients demised before an attempt could be made at wound closure.[3]

Use of anticoagulation after AVC repairs and ligations

Venous thrombo-embolic (VTE) complications after AVC injuries managed with ligation or repair are expected, but the incidence is unknown. A review of 308 patients by Singer et al identified that the rate of VTE was not statistically different between patients managed with ligation and those managed with repair. The overall rate of VTE in the series was 2.3%, which was less than expected. Five (1.6%) cases of deep venous thrombosis (DVT) and 2 cases (0.6%) of pulmonary embolism (PE) were documented.[29]

There is no level 1 or 2 evidence in the literature regarding the use of anticoagulation for patients with major venous injuries. The American College of Chest Physicians (ACCP) have proposed some general anticoagulation guidelines for these scenarios.

The 2012 ACCP guidelines recommend commencement of a low molecular weight heparin (LMWH) in patients who have sustained major trauma as soon as it is considered safe to do so.[30] In immobile patients thromboprophyllaxis should be continued until hospital discharge. Where LMWH is contraindicated an acceptable alternative is to use a combination of graduated compression stockings (GCS) and a pneumatic compression device (PCD). Most patients

with caval injuries would fall into this category and should thus receive thromboprophyllaxis as recommended.

The use of vena cava filters (VCF) is controversial in trauma patients. Although not recommended in the current ACCP guidelines, use of VCF's has been reported in the literature for conditions where:

- There was a contraindication to LMWH e.g. Heparin Induced Thrombocytopaenia (HIT)
- Patients developed bleeding complications while on LMWH
- Patients who developed VTE while on treatment with LMWH

There are studies in the literature that showed a decrease in the incidence of PE where VCF's were prophylactically used. Concerns about the safety and early and long-term complications with the use of VCF's have limited their use.[30]

Long-term morbidity after ligation of the AVC

Complete long-term follow-up in trauma patients is notoriously difficult. Many series in the literature on AVC trauma do not report on follow-up. Feliciano et al reported follow-up on 7 patients who had had AVC ligations. At a mean of 42 months, no patient had any evidence of lower limb swelling or dysfunction. This is the largest set of followed-up patients in the trauma literature to date with this injury.[3]

CHAPTER TWO

AIM

The aims of this study were to:

- Review and report on the surgical management of abdominal vena cava injuries in the Trauma Centre at Groote Schuur Hospital.

- Compare data and outcomes in this review to data from a previous study from the same unit

- Document the incidence of lower limb fasciotomy

- Identify significant parameters that were different between survivors and non survivors of AVC injuries

- Document any lower limb venous morbidity in a selected sample of survivors following AVC ligation

CHAPTER THREE

METHODS

The study design was a retrospective review of AVC injuries that were managed in the Trauma Centre at Groote Schuur Hospital from January 2003 to December 2011.

Approval from the Department of Surgery Research Committee and Faculty of Health Sciences Human Research Ethics Committee was obtained prior to accessing data.

Patients were identified from the Trauma Centre's operative logbook and their records were obtained from the General Hospital filing department or from the Medico legal department. All patients with generalized peritonitis and/or an abdominal cause for haemodynamic instability had an emergency laparotomy after resuscitation along ATLS principles.

Data points that were obtained from the patients' medical records included:

Temporal data

- Date of admission
- Date of ICU discharge
- Date of Hospital discharge
- Date of death

Demographic data

- Age
- Gender

First recorded physiological data at admission

- Systolic blood pressure
- Haemoglobin concentration
- Arterial pH
- Serum lactate concentration
- Base deficit
- Temperature

Injury data

- Mechanism of injury – blunt or penetrating
- Mechanism of penetrating trauma – gun shot, stab wound or shotgun wound
- Level of injury as described in the operation note – infrarenal, juxtarenal, suprarenal or retrohepatic
- Associated intra- and extra abdominal injuries as depicted in the medical records and operation notes.

Management data

- Number of packed red cell units required in the first 24 hours
- Ligation or repair of the AVC injury
- Whether the surgical strategy used was damage control or definitive management
- Whether a consultant trauma surgeon was present at the operation or not

Morbidity and Mortality data

- Documented complications following management of the AVC injury
- Time and cause of death as described in the operation notes and medical records

Data was captured on proforma and converted to an Excel spreadsheet.

Statistical analysis was performed using the program, STATA Version 11.0. (Statacorp Lp, 2009).

For descriptive purposes, mean ± SD were calculated for continuous variables, and proportions were used for categorical variables. To determine the difference in the mean continuous variables, student t-tests and One-way ANOVA tests were applied when appropriate. Chi-squared and Fishers exact tests were used for determining associations between categorical variables.

Statistical significance was defined at a level of <0.05.

CHAPTER FOUR

RESULTS

During the period from 01-01-2003 until 31-12-2011 thirty-five patients with AVC injuries were identified. There were 29 males and 6 females with a mean age of 27.17 years and a range of 15 to 45 years.

Total	n=35
Male	29
Female	6
Mean age	27.17 ± 7.69

Table 1. Demographic data

Thirty-three of the injuries were caused by penetrating trauma (94.29%) of which gun shot wounds accounted for 28 and stab wounds for 4. There was one shotgun wound.

There were two blunt injuries to the AVC (5.71%) caused by motor vehicle accidents.

Total	n=35
Blunt	2
Penetrating	33
Gunshot wounds	27
Stabs	4
Shotgun wounds	1

Table 2. Mechanism of Injury

Infrarenal injuries made up 19 of the injuries, Juxtarenal 9, Suprarenal 3 and retrohepatic injuries made up 4.

AVC Injury	Number (%)
Infrarenal	19 (54.29)
Juxtarenal	9 (25.71)
Suprarenal	3 (8.57)
Retrohepatic	4 (11.43)

Table 3. Level of Injury

There were 17 deaths (48.57%) in this series, while 18 (51.43%) patients survived.

The mean values of the first recorded physiological parameters of the patients as they came in to the Trauma unit were as follows:

First recorded parameter	Mean ± SD
Systolic blood pressure (mm Hg)	113.71 ± 34.15
Lactate (mmol/l)	3.81 ± 2.27
pH	7.29 ± 0.11
Temperature (°C)	35.69 ± 1.15
Base deficit	-7.22 ± 5.37
Haemoglobin (g%)	10.12 ± 2.67

Table 4. Physiological parameters with means

Three Trauma Scoring systems were used in this study; the Revised Trauma Score (RTS), the Penetrating Abdominal Trauma Index (PATI) and the Injury Severity Score (ISS). Mean scores with standard deviation and ranges were calculated. The mean RTS was 6.931, the mean PATI 41.52 and the mean ISS 33.71.

Scores	Mean ± SD	Min.	Max.
RTS	6.931 ± 1.48	2.6	7.841
PATI	41.52 ± 13.06	12	61
ISS	33.71 ± 9.06	25	54

Table 5. Trauma scores

The number and type of associated intra- and extra abdominal injuries was recorded. All patients had a least one associated intra-abdominal injury with more than half of the patients sustaining at least 2 intra-abdominal injuries. Liver injuries were the most common associated intra-abdominal injury with 18, followed by large bowel injuries with 13. There were a further 12 associated duodenal and 12 small bowel injuries.

Associated Intra-abdominal injuries	Frequency (N)	Percentage (%)	Cumulative (%)
1	3	8.57	8.57
2	9	25.71	34.29
3	11	31.43	65.71
4	5	14.29	80.00
5	4	11.43	91.43
6	2	5.71	97.14
7	1	2.86	100.00
Total	35	100.00	

Table 6. Frequency of associated intra-abdominal injuries

Associated intra-abdominal injury	Number (n)
Liver	18
Large bowel	13
Duodenum	12
Small bowel	12
Kidney	8
Stomach	5
Diaphragm	5
Mesentery	4
Ureter	4
Pancreas	4
Gall bladder	3
Urethra	1
Bile duct	1

Table 7. Numbers of intra-abdominal injuries

Extra-abdominal injuries were less common. A total of 62.86% of patients had at least one such injury.

Associated extra-abdominal injuries	Frequency (N)	Percentage (%)	Cumulative (%)
0	13	37.14	37.14
1	13	37.14	74.29
2	6	17.14	91.43
3	2	5.71	97.14
8	1	2.86	100.00
Total	35	100.00	

Table 8. Frequency of extra-abdominal injuries

Patients with the highest number of extra-abdominal injuries had most commonly sustained blunt trauma due to motor vehicle accidents and the injuries involved were mostly compound long bone fractures.

A trauma consultant surgeon was present at 30 of the 35 operations performed (85.7%). A senior trauma registrar was present in all the cases where a consultant was not present.

In 25 of the procedures a surgical strategy of damage control surgery was used and in the remainder of cases a definitive approach was chosen.

During the first 24 hours of management, patients required a mean of 10.44 units of packed red cells with a range of 2 to 43 units.

	n
Trauma consultant present	30
No trauma consultant (senior registrar present)	5
Damage Control	25
Definitive Surgery	10
Mean red cell transfusion	10.44 (2 – 43) units

Table 9. Surgical strategies

AVC injuries were managed with ligation in 22 patients (62.86%). In four patients, intraoperative deathoccurred due toexsanguination after caval ligation had been performed. These patients had extensive associated intra-abdominal injuries which accounted for their demise.One patient had

sustained a close-range shotgun wound to his left flank and had multiple penetrating injuries to his abdominal aorta, left renal artery and vein. The other three patients had multiple injuries to hollow viscera with gross peritoneal soiling associated with their caval injuries. Repair of the AVC was performed in 6 patients (17.14%). No complex repairs or venoplasties were utilized.

Treatment	Number (%)
Ligation	22 (62.86)
Repair	6 (17.14)

Table 10. Management of injuries

Of the 17 deaths, 8 occurred intraoperatively due to exsanguination with a further 3 occurring within 48 hours of presentation due to uncorrectable coagulopathy. Five more patients demised after a time of 48 hours had passed since their initial presentation due to multiple organ dysfunction syndrome (MODS) with sepsis, while care was withdrawn by the treating physicians in one patient who had sustained a severe hypoxaemic brain injury

Cause of Death	n = 17
Intra-operative haemorrhage	8
Coagulopathy	3
Sepsis	5
Care withdrawn	1

Table 11. Mortality

There was an overall mortality rate of 48.57% in this series. Significant differences between survivors and non survivors in this series were found in systolic BP, the number of packed red cells required in the first 24 hours, serum lactate concentration, pH on the initial arterial blood gas analysis, first recorded temperature and haemoglobin concentration.

	Survivors	Non-survivors	*p* value
Systolic blood pressure (mean mm Hg)	124.94	101.82	0.0435
Packed red cells (mean number)	6.44 units	14.94	0.0001
Lactate (mean)	2.86 mmol/l	5.05 mmol/l	0.0073
pH (mean)	7.34	7.23	0.0015
Temperature (mean °C)	36.32	34.98	0.0004
Haemoglobin (mean)	11.25 g/dL	8.91 g/dL	0.0094

Table 12. Significant differences between survivor and non-survivor groups

There was no statistical association between the groups of survivors compared to non-survivors when the mechanism of injury, the agent of injury, the level of the injury or the trauma score values were evaluated.

	Survivors	Non-survivors	*p* value
Mechanism of trauma:			0.486
• Blunt	2	0	
• Penetrating	16	17	
Agent of penetrating injury:			0.275
• Stab	3	1	
• Gun shot	13	15	
• Shot gun	0	1	
Level of injury:			0.248
• Infrarenal	11	7	
• Juxtarenal	2	7	
• Suprarenal	2	1	
• Retrohepatic	2	2	
Trauma scores:			
• RTS (mean)	7.28	6.44	0.095
• PATI (mean)	38.56	44.29	0.213
• ISS (mean)	32.56	34.94	0.248

Table 13. Non-significant differences between survivors and non-survivors

The number of associated intra-abdominal injuries was not statistically different between the groups of survivors versus non-survivors *(p* = 0.165).

There was also no statistical difference between the groups that survived or demised regarding the number of associated extra-abdominal injuries (p = 0.384).

Differences in physiological parameters were analyzed between survivors and non-survivors to detect whether values outside of the normal established range for a particular parameter would significantly predict an adverse outcome, in this case death, in this series of patients.

Parameters analyzed in this way included systolic blood pressure, temperature and lactate.

Normal ranges and values were as follows:

- Systolic blood pressure above 90 mm Hg
- Temperature above 36°C
- Lactate above below 2.5 mmol/l
- Base deficit below -4.0

In this series of patients, a lactate level of more than 2.5 mmol/l was found to be significantly associated with an adverse outcome (*p* = 0.042), although there were patients that survived with levels in this range. There was a significant association between a temperature of below 36.0°C and the outcome of death (p = 0.042) although 6 survivors had a temperature of below 36°C on arrival. A significant association was not found between a systolic blood pressure of less than 90 mm Hg with mortality (p = 0.146). This may be

due to variations in a particular patients ability to haemodynamically compensate for a specific injury.

There was a trend towards a significant difference between the groups of survivors and non-survivors when a base deficit of greater or less than -4 was evaluated (p = 0.057).

The outcome of patients in whom a systolic blood pressure below 80 mmHg, lactate above 2.5 mmol/l and a temperature of below 36.0°C were all present was significantly different from those in who did not have this combination of derangements in this series, with death being more common in this group (p = 0.019).

Follow up and Morbidity

Follow up of patients who were discharged after successful management of their injuries was completed in 7 of the 18 survivors in this series (39%). The remaining patients were discharged to their local hospitals. Duration of follow up of the former group varied based on their individual problems. Some were seen once in the Trauma outpatient clinic after one month and discharged while another is undergoing long term follow up and has been seen annually for the last 3 years.

Of the 7 patients that were followed up, 5 had been managed with AVC ligation and 2 with repair of the injury.

General morbidity

The general morbidities in the group of survivors in this series varied. We expected that the rate of post-operative complications in this group of patients would be significant as a direct consequence of the high number of associated injuries that were encountered during surgical management.

Three patients developed bowel obstruction in the post surgical period. One was managed conservatively with extended bowel rest and total parenteral nutrition (TPN) after which resolution of the obstruction was achieved.
The other two patients required repeat surgery. Both were managed with laparotomies and adhesiolysis of obstructing bands. Neither of these patients required bowel resection and both subsequently achieved resolution of their obstructions.

One patient with apenetratingretrohepatic AVC injury, presenting as a contained haematoma, also had a penetrating injury to the common bile duct (CBD) that was missed at the index surgery. The patient had initially been managed according to damage control principles and the CBD injury was diagnosed at the repeat laparotomy performed 48 hours later. Repair of the injury was attempted, but the patient subsequently developed a CBD stricture and eventually required an hepaticojejunostomy.

One patient, a 34-year-old diabetic was the victim of multiple thoraco-abdominal gun shot wounds. He sustained a penetrating injury to the infrarenal AVC, as well as perforations of the caecum, multiple segments of

small bowel and transverse colon. The cava was managed with ligation and he received damage control measures for the remaining injuries at his index procedure. After his definitive surgery he required continuing ICU cardiorespiratory support. He subsequently developed multiple enterocutaneous fistulae and intra abdominal collections that were managed with surgical drainage and a laparostomy dressing. He had a massive haemorrhage into his laparostomy dressing at approximately one month after his injury and demised, the cause of which was unknown.

One patient, a 34 year old male, was the victim of an abdominal gunshot and sustained intra abdominal vascular as well as bowel injuries. His AVC was lacerated below the renal veins and there was an intimal flap injury to his left common iliac artery. There were also perforations of the caecum and sigmoid colon. The hollow viscus injuries were repaired, the AVC was ligated and the arterial injury was managed definitively with resection of the injured segment and use of an interposition 6mm gortex graft. After a period of 4 days in ICU, the patient was discharged to the ward for ongoing care. He developed signs of abdominal sepsis two weeks later and was investigated with a CT scan of the abdomen that revealed a large perigraft collection that contained air locules. The problem of graft sepsis was diagnosed. He was immediately taken back to theatre for excision of the graft and a fem-fem crossover bypass. He did well after this and was discharged. He had no complaints at his clinic visit one month later.

Venous Thromboembolism

Three patients developed deep vein thrombosis (DVT) during their index admission. All of these DVTs were diagnosed utilizing a combination of clinical suspicion as well as compression ultrasound in 2 and a CT venogram in the third. No patient was empirically screened for DVT in this series.

There was no association between the risk of DVT and the strategy used to repair an AVC injury (ligation or repair) in this series (p=0.481) although the small number of cases may have influenced this.

There was one non-occlusive common femoral DVT, one iliofemoral DVT and one infrarenal vena caval free-floating thrombus that had been diagnosed in a patient who had been managed with a venous repair of a penetrating injury to the retrohepatic AVC.

All three of these patients were followed up after discharge at the trauma clinic in order to manage the ongoing oral anticoagulation as well as to evaluate for evidence of chronic venous insufficiency.

The former two patients were managed with extended oral anticoagulation and compression stockings and reported no symptoms of chronic venous insufficiency at their subsequent clinic visits. Both have since stopped their Warfarin.

The patient who developed free-floating caval thrombus was managed with placement of a removable IVC filter that was inserted under fluoroscopic guidance via a (deleted) jugular approach. The filter was left in situ for a period of 8 weeks while anticoagulation was established and was subsequently retrieved.

This patient has been seen in follow up clinic frequently and is now undergoing annual venous duplex surveillance of his AVC and deep leg veins. The first duplex performed at one year post injury was normal with no evidence of chronic venous disease. His oral anticoagulation was stopped at this visit. Although the duplex performed one year later (shown below) did show evidence of post phlebitic changes in the deep veins of the thighs and AVC, the patient was well and complained only of occasional swelling symptoms in his legs. He continues to be followed up and uses compression stockings.

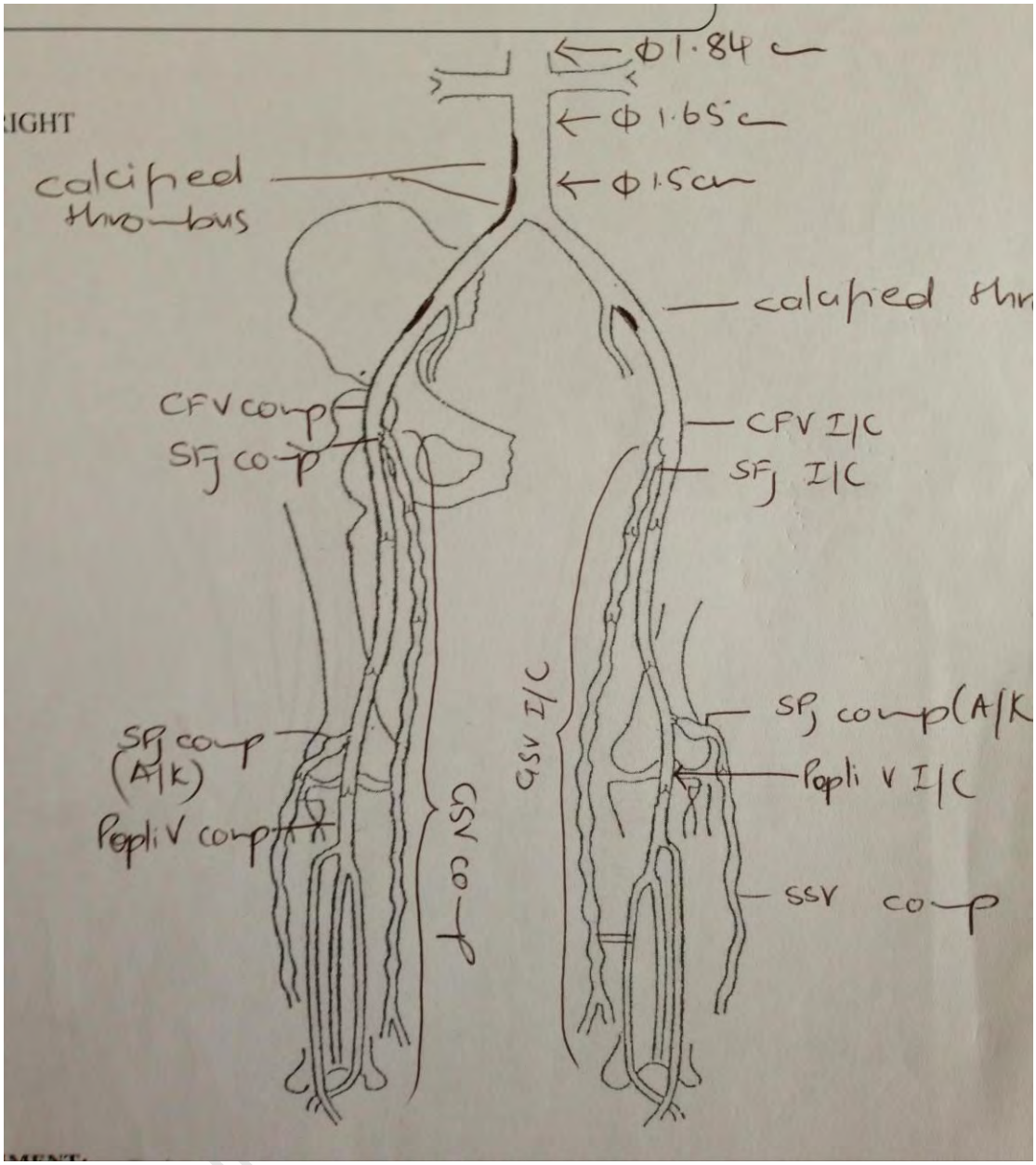

Figure 6. Venous Duplex performed 2 years after repair of Retrohepatic AVC injury and placement of vena caval filter

CHAPTER FIVE

DISCUSSION

Abdominal caval injuries injuries continue to be associated with a high mortality rate. One third of patients will die of their injuries before reaching hospital and a further 30% will die within 24 hours of hospital admission due to exsanguination or the resultant coagulopathy associated with major trauma.[13]A subset of patients will develop the multi organ dysfunction syndrome (MODS)and succumb to the consequences of the associated abdominal and extra abdominal injuries.

Although the mortality rate in this series of 48.57% is comparable to other reported series, of concern to us is the increased rate of mortality we have seen compared to the initial report by Navsaria et al from our own institution.[7] There are no clearly identifiable reasons why this should be so, except for the fact that it is possible that our Paramedic service has improved to such an extent that patients who would previously have demised before reaching hospital are now reaching us sooner, meaning that we are possibly treating patients who are initially more unstable than they were in the past. On comparingpenetrating AVC injuries attributable to the different injuring agents, the numbers are not different between Navasaria's original study and this one. The number of gun shot wounds is similar and therefore the increase in the mortality rate we have seen in this series is not due to a higher number of gun shot wounds. We were not able to evaluate whether the types of handguns used by the perpetrators of gun violence in this series was any different to those used in the original series. The possibility exists that more powerful

handguns with an increased potential to cause more extensive injury are now more freely available.

Various studies, including our own,demonstrate that basic physiological parameters, which should be measured on all trauma patients at admission, vary significantly between the group of patients who will survive and those who will not. We have also been able to show that abnormalities in certain of these basic parameters were significantly predictive of an adverse outcome in our group of patients especially where patients had combinations of physiological derangements.

We feel that damage control principles should be readily utilized in patients with injuries to the AVC due to the high likelihood of associated major injuries and we feel that early recognition of these patients in the emergency department is possible by using simple measurements of the patients vital signs, haemoglobin and lactate concentration. An abbreviated laparotomy in an unstable, bleeding patient is the optimal management and this demands rapid and definitive control of major haemorrhage.

We found that in this series, the majority of injuries (approximately 80%) involved the infra- and juxtarenal cava. Although a statistically significant association between the likelihood of survival and the level of injury was not found, of the patients that survived, a high percentage of them had infrarenal injuries (66.67%).

There were slightly fewer infrarenal injuries in this series than those found in the original series by Navsaria et al, where more than 85% of injuries were infra- or juxtarenal, although this change is unlikely to represent true statistical difference.[7]

Ligation of the AVC rapidly achieves the first goal of damage control surgery, which is to stop major bleeding. We found that the group of patients that did not survive in this series was more likely to have been managed with caval ligation ($p < 0.0001$), which may indicate that ligations were performed in only the most unstable patients with the worst injuries.

Infrarenal caval ligation is generally simple and we advocate its use as a life-saving means of haemorrhage control in the appropriate circumstances. The rate of ligation in this series, 62.86%, is similar to the ligation rate in the previous series of AVC injuries from our institution by Navsaria et al.[7] We do feel that there is a definite role for caval repair when the patient is physiologically and haemodynamically stable and will tolerate a longer operation and in this series we found that where the abdominal vena caval injury was managed with primary repair, the patient was more likely to have been part of the group of survivors ($p = 0.005$).

For injuries to the retrohepatic cava we have had some success in performing perihepatic packing for initial haemorrhage control via tamponade, and subsequently performing a delayed retrohepatic caval repair. We have achieved this without the use of deep hypothermic arrest and bypass, but we

acknowledge that some retrohepatic caval injuries may well be better served using this technique. We have not utilized atriocaval shunting nor have we had any experience using laparoscopic methods of caval repair. We feel that laparoscopic methods of repair are only really suitable in the setting of iatrogenic caval injury during an elective laparoscopic operation where the patient has not been subjected to a major physiological insult. We feel the same way regarding the use of endovascular stent-grafting of the cava. Although these are elegant techniques and have been shown in some case reports to be feasible in certain circumstances, we do not support their use for patients who have sustained multiple injuries who require damage control surgery. In our series we found significant differences in systolic blood pressure, the number of packed red cell units required by the patient in the first 24 hours, the pH and lactate on the initial blood gas, the core temperature and haemoglobin levels between survivors and non survivors. This is in accordance with other published series.[3,7]

We were unable to find statistically significant associations between these groups when the mechanism of injury, the agent causing the penetrating injury, the level of injury and the trauma scores were analyzed. This is in contrast to the previous series from our institution by Navsaria et al where the ISS scores were significantly different between survivors and non survivors.[7] This may again indicate improvements in paramedic services where sicker patients reach our services more quickly and changes in referral patterns whereby secondary drainage hospitals are bypassed and polytrauma patients are brought directly to our unit. There was also no difference between

survivors and non survivors in this series in terms of the number of associated injuries, nor was there a difference to the outcome whether a trauma consultant surgeon was present at the operation or not, also in accordance with the original study from our institution.

On further analysis of the data in this series we found that a lactate level above 2.5 mmol/l on the initial gas was significantly associated with an adverse outcome as did a core temperature of below 36°C. A derangement in the base deficit trended towards significance for an adverse outcome. When patients had more than one baseline physiological derangement, this was significantly associated with an outcome of death. We suggest that rapid identification of these derangements in polytrauma patients with targeted resuscitative therapy and early surgery could help to lower the high mortality rate for patients with this devastating injury in the future.

This leads to the question as to whether futile damage control operations could be avoided by opting to not actively manage profoundly hyperlactataemic and hypothermic patients.

In order to address this question, Nicol et al devised a formula incorporating the age of the patient, the pH and temperature to identify patients in whom active treatment would be futile.[19]

There has been some controversy in the literature regarding the use of prophylactic lower limb fasciotomies in situations where caval ligation has

been performed. This intervention has never been performed in two series of AVC injuries from our institution, both reporting AVC ligation rates in excess of 60%. We feel that in general, there are enough extra-caval collateral venous pathways available for blood to pass through on its return from the lower limbs to the right side of the heart. These pathways have been demonstrated in radiological studies.[20] We feel that the occurrence of an acute obstruction to venous outflow from the leg, which is severe enough to result in a lower limb compartment syndrome, is extremely rare and therefore prophylactic fasciotomy of the lower limb is not justified.

Although 2 of our surviving patients who had been managed with AVC ligation developed DVTs, both were managed conservatively and neither patient had any evidence of a post thrombotic syndrome at follow up. There was no statistical association between AVC ligation and an increased risk of DVT in this series.

CHAPTER SIX

CONCLUSION

Patients with injuries to the AVC often present trauma teams and surgeons with difficult management problems. They are usually haemodynamically unstable and are often at risk of the coagulpathy of trauma which is now a well-described and often fatal entity.

Patients who present wih more than one isolated physiological derangement on arrival in the emergency unit appear to have the highest risk of death.

We, and others, have described ligation of the vena cava as a reasonable and time-efficient technique to arrest haemorrhage from a blunt or penetrating injury to it, which is the cornerstone tenet of damage control surgery. We believe that some of the concerns regarding chronic venous insufficiency after caval ligation have been overstated in other series and we feel the use thereof is justified by the equivalent survival rate and low venous complication rates shown in this series of patients when compred to others.

We srongly advocate pre-hospital triage of patients with potential major abdominal vascular injuries with simple measures such as vital signs and baseline haemoglobin so that delays at centres without the necessary means to manage such patients can be avoided.

The majority of these patients require management at a dedicated trauma centre wth early institution of damage control principles if they are to have a chance of surviving this potentially devastating injury.

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ADDENDA

UNIVERSITY OF CAPE TOWN

Department of Surgery

Departmental Research Committee
Professor Anwar Suleman Mall
J-45 Room Old Main Building, Groote Schuur Hospital,
Observatory 7925, South Africa
Tel (021) 406-6148/6230/6227 FAX (021) 448-6461
Email: Anwar.Mall@uct.ac.za

8th May 2012

Dr M Hampton
Department of Surgery
Division of General Surgery
Groote Schuur Hospital
University of Cape Town

Dear Dr Hampton,

RE: PROJECT 2012/038

PROJECT TITLE: **Morbidity associated with traumatic IVC injuries in the damage control era**

The above proposal was reviewed by the Department of Surgery Research Committee and I am pleased to inform you that the committee approved the study.

Please use the above project number in all future correspondence.

Yours sincerely

PROFESSOR ANWAR S MALL
CHAIRMAN: RESEARCH COMMITTEE

Addendum 1. Research Committee approval

UNIVERSITY OF CAPE TOWN

Faculty of Health Science
Faculty of Health Sciences Research Ethics Committe
Room E52-24 Groote Schuur Hospital Old Main Buildin
Observatory 792
Telephone (021) 406-6338 • Facsimile (021) 406 641
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24 July 2012

HREC REF: 366/2012

Dr M Hampton
Department of General Surgery
J45
OMB

Dear Dr Hampton

PROJECT TITLE: MORBIDITY ASSOCIATED WITH TRAUMATIC IVC INJURIES IN THE DAMAGE CONTROL ERA

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

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

Approval is granted for one year till the 28 July 2013.

Please submit a progress form, using the standardised Annual Report Form (FHS016), if the study continues beyond the approval period. Please submit a Standard Closure form (FHS010)if the study is completed within the approval period.

Please obtain permission from Dr Bruwer Patel to access patient's records for research purposes.

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

Please quote the REC. REF in all your correspondence.

Yours sincerely

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS

Federal Wide Assurance Number: FWA00001637
Institutional Review Board (IRB) number: IRB00001938

Addendum 2. Ethics Committee approval

RESEARCH PROPOSAL FOR MMED (SURG).

MORBIDITY ASSOCIATED WITH TRAUMATIC IVC INJURIES IN THE DAMAGE CONTROL ERA

INVESTIGATOR: Dr Mark Ian Hampton
Registrar Dept. of General Surgery

SUPERVISOR: A/Prof Pradeep H Navsaria

INTRODUCTION:

Traumatic injuries to the IVC occur infrequently in the civilian setting and may be caused by both blunt and penetrating mechanisms. These injuries constitute a small percentage of all vascular traumas seen in our unit but are associated with high mortality and morbidity due not only to the caval injury alone, but also to the injuries invariably associated with it.
The practice of ligating the IVC has become accepted in the modern management of devastating caval haemorrhages, especially in a damage control scenario.
Although it can be argued that this practice may be life-saving, it is not known what the immediate and long term effects are with specific reference to renal function with supra-renal caval ligation and the development of lower extremity post thrombotic syndrome with caval ligation at any level.
The risk of developing lower extremity compartment syndrome after caval ligation has been raised as a concern and has led to selective prophylactic lower limb fasciotomy use in various large reported series. This is not a generally accepted practice and has never been performed in our unit.
The purpose of this study is to review our Trauma unit's management and outcomes of these injuries over an eight-year period with specific emphasis on the group of patients who were managed with caval ligation as opposed to lateral repair.
We propose to selectively follow a subgroup of these patients and perform a standardized examination to identify evidence of post thrombotic syndromes.

AIMS AND OBJECTIVES:

The aim of this study is to examine the incidence, presentation, management and outcomes of IVC injuries in our centre; to specifically document our unit's limited use of prophylactic lower limb fasciotomy in caval ligation; to evaluate renal function and the need for renal replacement therapy in patients with supra-renal caval ligation and to determine the incidence of post thrombotic syndrome in selected patients with caval ligation.

PATIENTS AND METHODS:

Patients will be identified by a review of the Trauma unit's operative note filing system from the period of 01-01-2004 until 31-01-2011. Patient charts will then be retrospectively analysed.
Demographic data, mechanism of injury, associated intra- and extra-abdominal injuries, blood product requirement in the 1st 24 hours, type of surgical procedure and management of the caval injury will be reviewed. Evidence of Acute Coagulopathy of Trauma Shock (ACOTS) will be sought by means of preoperative arterial blood gas analysis, temperature on presentation and reporting in the operative notes.
The Injury Severity Score (ISS), Penetrating Abdominal Trauma Index (PATI) and the Revised Trauma Scores (RTS) will be calculated.
ICU management will also be reviewed in terms of the need for renal replacement therapy, repeated surgical procedures and length of stay.
A selected group of patients with caval ligations will be followed up with a questionnaire, clinical examination and venous duplex scanning to identify evidence of lower extremity post thrombotic syndrome.

LITERATURE REVIEW:

The trauma surgical literature contains various reports of IVC injury management and outcomes. The majority of these are made up of retrospective series which compare ligation versus lateral repair. The largest reported series consists of 100 consecutive patients with IVC injuries and 25 with caval ligations, but most series contain between 30 and 50 patients. There are three case reports of caval injuries managed by endovascular means.
Reporting on the selective use of prophylactic lower limb fasciotomies varies widely in the literature.
Very few series document long term follow up of patients after caval ligation with the largest groups of follow up varying between 5 and 7 patients.

ETHICS:

This study aims to review our existing practice in the management of IVC injuries and aims to identify modifiable factors which may lead to a decreased incidence of lower limb post thrombotic syndrome.

This study adheres to the Declaration of Helsinki 2000.

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Addendum 3. Proposal document

Western Cape Government
Health

GROOTE SCHUUR HOSPITAL
Enquiries: Dr Bhavna Patel
E-mail : Bhavna.Patel@westerncape.gov.za

Dr Mark Hampton
Department of Surgery
J45 – Old Main Building

E-mail: drmarkhampton@icloud.com & Pradeep.Navsaria@uct.ac.za

Dear Dr Hampton

RESEARCH: Morbidity Associated with Traumatic IVC Injuries in the Damage Control Era

Your recent letter to the hospital refers.

You are hereby granted permission to proceed with your research.

Please note the following:

a) Your research may not interfere with normal patient care
b) Hospital staff may not be asked to assist with the research.
c) No hospital consumables and stationary may be used.
d) **No patient folders may be removed from the premises or be inaccessible.**
e) Please introduce yourself to the person in charge of an area before commencing.
f) Confidentiality must be maintained at all times.

I would like to wish you every success with the project.

Yours sincerely

DR BHAVNA PATEL
SENIOR MANAGER: MEDICAL SERVICES
Date: 11th February 2013

c.c. Dr B. Jacobs

Addendum 4. Permission to review folders

	N = 35
Male	29
Female	6
Mean age	27.17 ± 7.69

Table 14. Demographic data

	N=35
Blunt	2
Penetrating	33 • Gun shot wounds: 27 • Stabs: 4 • Shotgun wounds: 1

Table 15. Mechanism of Injury

AVC Injury	Number (%)
Infrarenal	19 (54.29)
Juxtarenal	9 (25.71)
Suprarenal	3 (8.57)
Retrohepatic	4 (11.43)

Table 16. Level of Injury

First recorded parameter	Mean ± SD
Systolic blood pressure (mm Hg)	113.71 ± 34.15
Lactate (mmol/l)	3.81 ± 2.27
pH	7.29 ± 0.11
Temperature (°C)	35.69 ± 1.15
Base deficit	-7.22 ± 5.37
Haemoglobin (g%)	10.12 ± 2.67

Table 17. Physiological parameters with means

Scores	Mean ± SD	Min.	Max.
RTS	6.931 ± 1.48	2.6	7.841
PATI	41.52 ± 13.06	12	61
ISS	33.71 ± 9.06	25	54

Table 18. Trauma scores

Number: intra-abdominal injuries	Frequency (N)	Percentage (%)	Cumulative (%)
1	3	8.57	8.57
2	9	25.71	34.29
3	11	31.43	65.71
4	5	14.29	80.00
5	4	11.43	91.43
6	2	5.71	97.14
7	1	2.86	100.00
Total	35	100.00	

Table 19. Frequency of associated intra-abdominal injuries

Associated intra-abdominal injury	Number (N)
Liver	18
Large bowel	13
Duodenum	12
Small bowel	12
Kidney	8
Stomach	5
Diaphragm	5
Mesentry	4
Ureter	4
Pancreas	4
Gall bladder	3
Urethra	1
Bile duct	1

Table 20. Numbers of intra-abdominal injuries

Number: extra-abdominal injuries	Frequency (N)	Percentage (%)	Cumulative (%)
0	13	37.14	37.14
1	13	37.14	74.29
2	6	17.14	91.43
3	2	5.71	97.14
8	1	2.86	100.00
Total	34	100.00	

Table 21. Frequency of extra-abdominal injuries

	n
Trauma consultant present	30
No trauma consultant (senior registrar present)	5
Damage Control	25
Definitive Surgery	10
Mean red cell transfusion	10.44 (2 – 43) units

Table 22. Surgical strategies

Treatment	Number (%)
Ligation	22 (62.86)
Repair	6 (17.14)

Table 23. Management of injuries

Cause of Death	n = 17
Intra-operative haemorrhage	8
Coagulopathy	3
Sepsis	5
Care withdrawn	1

Table 24. Mortality

	Survivors	Non-survivors	*p* value
Systolic BP (mean mm Hg)	124.94	101.82	0.0435
Packed red cells (mean number)	6.44 units	14.94	0.0001
Lactate (mean)	2.86 mmol/l	5.05 mmol/l	0.0073
pH (mean)	7.34	7.23	0.0015
Temperature (mean °C)	36.32	34.98	0.0004
Haemoglobin (mean)	11.25 g/dL	8.91 g/dL	0.0094

Table 25. Significant differences between survivor and non-survivor groups

	Survivors	Non-survivors	*p* value
Mechanism of trauma:			0.486
• Blunt	2	0	
• Penetrating	16	17	
Agent of penetrating injury:			0.275
• Stab	3	1	
• Gun shot	13	15	
• Shot gun	0	1	
Level of injury:			0.248
• Infrarenal	11	7	
• Juxtarenal	2	7	
• Suprarenal	2	1	
• Retrohepatic	2	2	
Trauma scores:			
• RTS (mean)	7.28	6.44	0.095
• PATI (mean)	38.56	44.29	0.213
• ISS (mean)	32.56	34.94	0.248

Table 26. Non significant differences between survivors and non-survivors

Addendum 5. Tabulated results