ASSESSMENT OF DIAGNOSTIC MODALITIES IN PENETRATING CARDIAC TRAUMA FOR THE HAEMODYNAMICALLY STABLE PATIENT

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A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, in partial fulfillment of the requirements for the Degree of Master of Medicine in Surgery.

Johannesburg 2014

DECLARATION

I, Daniel Johnathan David Surridge, declare that this research report is my own work. It is being submitted for the degree of Master of Medicine in General Surgery at the University of the Witwatersrand, Johannesburg. It has not been submitted for any degree or examination at this or any other University.

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Daniel Johnathan David Surridge

31st January 2014

DEDICATION

To my darling wife, Alice, whose unwavering support and tolerance (even to my wild surgical ranting at all hours of the day and night) has made it possible to specialize in surgery.

To my family for always having complete confidence in my abilities despite the available evidence and for investing in my dreams at no matter what cost.

To my many mentors who have taken my numerous breathtaking blunders and given them some small degree of finesse.

CONGRESS PRESENTATIONS FROM THIS STUDY

Oral presentations

1) Precordial Stab Wounds in the Developing World: a Retrospective Analysis (preliminary study): Annual meeting of the Surgical Research Society, Bloemfontein 27-28/06/2007
2) Precordial Stab Wounds in the Developing World: a Retrospective Analysis (preliminary study): Bert Myburgh Research Day 28/09/2007
3) Resolving the Dilemma of the Stable Stabbed Heart (extended evidence): ASSA-SAGES meeting, Cape Town 7-10/08/2009
4) Solving the Dilemma of the Stable Stabbed Heart (final evidence): Annual meeting of the Surgical Research Society, Stellenbosch 12-13/07/2012
5) Troponin T as a Screening Tool for Occult Cardiac Injury (final evidence): Bert Myburgh Research Day 28/11/2012

Poster presentations

1) Troponin T as a Screening Tool for Occult Cardiac Injury (final evidence): Faculty Day University of the Witwatersrand Faculty of Health Sciences 19/09/2012

International presentations

1) Troponin T as a Screening Tool for Occult Cardiac Injury (final evidence): Annual clinical meeting of the American College of Surgeons, Chicago, Illinois 30/09-4/10/2012 (see Appendix D)

ABSTRACT

Introduction: One in 11.5 patients with a thoracic wound has cardiac involvement with potentially life-threatening consequences. Therefore, cardiac injury must be assumed in every patient with a penetrating chest lesion, even if the patient is haemodynamically stable. A need exists to diagnose or screen for “occult” cardiac injury.

Methods: A retrospective analysis was conducted in patients with a penetrating injury to the chest at Charlotte Maxeke Johannesburg Academic Hospital Trauma Unit from 1 January 2007 to 30 June 2010. Data was compared between patients with and without cardiac injury. Clinical examination and special investigations were assessed for sensitivity, specificity, positive and negative predictive values.

Results: Of 7 781 major injuries assessed, 1 591 (20%) sustained a penetrating injury to the chest. All cardiac injury was incurred through a precordial wound. Two investigations were found to be both significant and useful. Transthoracic echocardiography (TTE) had a sensitivity of 100% and specificity of 95%. Serial Troponin T (Trop T) levels showed a peak at 4 hours and by 6 hours post admission the specificity and negative predictive values were 100%.

Conclusion: Of the investigations examined, TTE was found to have the best results. The need for specialised equipment and training make TTE less practical in a resource-limited environment. Serial Trop T shows a high negative predictive value and is a cost effective screening test for penetrating cardiac injury.

ACKNOWLEDGEMENTS

- Prof J Goosen, MBChB; FCS(SA)

  Principle Specialist Surgeon, University of the Witwatersrand

  Head of Trauma Unit, Charlotte Maxeke Johannesburg Academic Hospital

  Supervisor.
- Dr MS Moeng, MBChB; FCS(SA)

  Senior Specialist Surgeon, University of the Witwatersrand & Charlotte Maxeke Johannesburg Academic Hospital
- Prof G Candy, PhD

  Research Manager, Department of Surgery, University of the Witwatersrand

  Supervisor.
- Dr D Kruger, PhD

  Research Coordinator, Department of Surgery, University of the Witwatersrand

  Proofreading and editing
- The collection, analysis and write-up of data for this study have received no funding.
- Finance was kindly provided by the Health Sciences Research Office and the Department of Surgery of the University of the Witwatersrand as well as the Trauma Unit of Charlotte Maxeke Johannesburg Academic Hospital for the presentation of this study at the Annual clinical meeting of the American College of Surgeons in Chicago, Illinois, 2012.

TABLE OF CONTENTS

LIST OF FIGURES AND TABLES

LIST OF ABBREVIATIONS

ABG	Arterial blood gas
CK	Creatinine Kinase
CKMB	Creatinine Kinase M and B fractions
CMJAH	Charlotte Maxeke Johannesburg Academic Hospital
CT	Computer assisted tomography
CXR	Chest x-ray
ECHO	Echocardiogram
ER	Emergency room
ICU	Intensive care unit
M:F	Male to female ratio
NPV	Negative predictive value
P1	Priority one
PPV	Positive predictive value
TEE	Transoesophageal echocardiogram
Trop I	Troponin I
Trop T	Troponin T
TTE	Transthoracic echocardiogram
US	Ultrasound

CHAPTER 1 – INTRODUCTION

Penetrating cardiac injury is a life-threatening event. This condition is so severe that 70-90% of such patients die before reaching hospital and 80% of those who make it to a hospital present in a haemodynamically unstable state (1, 2). Penetrating cardiac injury constituted 0.5% of all trauma admissions to a single level 1 trauma center in the United States of America assessed over a 7 year period in the 1990's (3). These statistics depend on the level of pre-hospital care and the demographics of the population serviced by the health care facility (4-6). For example, it has been found that areas with better developed prehospital services have worse inhospital results for cardiac injury despite more advanced hospital services and this is thought to be due to increased numbers of moribund patients reaching the Emergency room (ER) alive than has ever been seen before (3). One can speculate that a greater number of trauma admissions for penetrating cardiac injury may be found in South Africa today as access to emergency care and private transport has increased along with rapid urbanisation.This has, however, not been assessed in the literature.

Moreover, patients who have sustained a penetrating injury to the heart do not always present with overt haemodynamic instability or signs of pericardial tamponade (4, 7-9). Approximately 20% present with normal vital signs (6) and some presenting in shock may completely respond to fluid resuscitation (10). In these haemodynamically stable patients, penetrating cardiac injury may be missed (11, 12). Furthermore, such patients may present with delayed tamponade (13) and rapid haemodynamic decompensation. Others may present later still with constrictive pericarditis (10). The true incidence of these complications is unknown but has been estimated at 9% (10). *Demetriades et al* in 1983 reported 5 missed injuries out of 125 hospitalised patients who rapidly decompensated and died (6). *Harris et al* showed that 2/3 patients who had been stabilised and admitted for observation of clotted haemopericardium eventually decompensated requiring life-saving surgery (4). It is impossible to know what the risk factors for these lethal complications are as patients have been reported to complicate late (13) and only present in the mortuary (11). Since the true incidence of missed injuries is unknown (10) and the risk of a specific injury cannot be accurately assessed (11, 13), the true impact of missed cardiac injury is not known. What is known is that these patients can decompensate catastrophically early or late and even develop postpericardial syndrome, i.e. constrictive pericarditis and effusion (10). Although, both of these conditions can prove fatal and pose their own diagnostic difficulties, they are beyond the scope of this study.

Therefore, from the above, it must be noted that a missed cardiac injury can be fatal and progression can be averted or monitored and managed if accurate, early diagnosis is made. Avoidable mortality as high as 9% has been reported (11).

Essentially, every haemodynamically stable patient with a penetrating chest injury requires clinical assessment and investigation on suspicion of having a cardiac injury (4). In our institution, we adhere to Advanced Trauma Life Support (ATLS®) principles to assess and resuscitate patients with a penetrating chest injury. Although no formal management algorithm is used, most resuscitation procedures would follow the stepwise approach laid out in Figure 1.1.

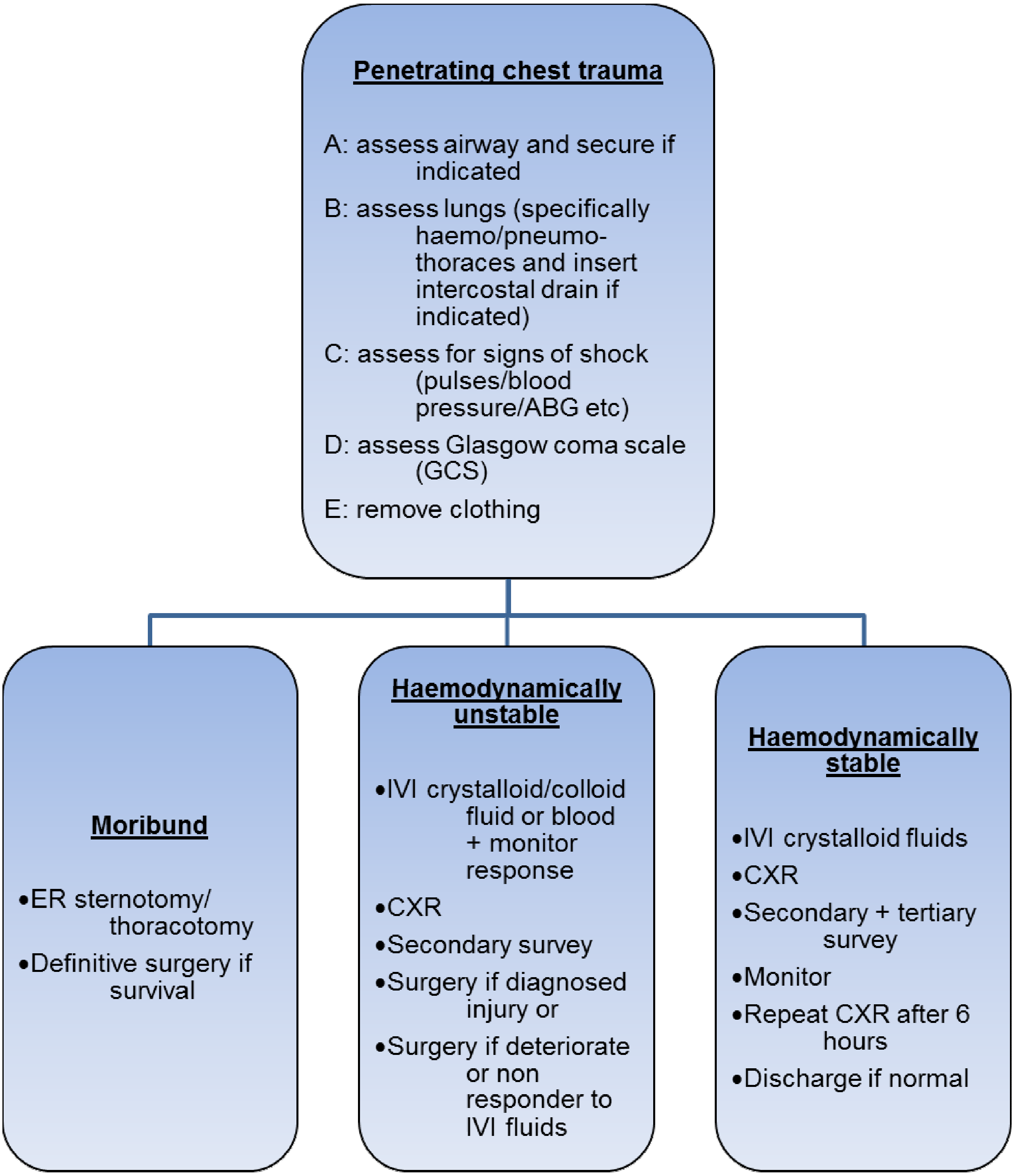


Figure 1.1. Informal algorithm for management of penetrating chest trauma at CMJAH

From the discussion above, it would appear that life threatening penetrating cardiac injury can present a diagnostic dilemma in a stable patient or a patient who responds to fluid resuscitation. With improvement in prehospital emergency services and access to private transport, more severe cases are arriving in ERs (3) resulting in front room heroics and giving the impression that this injury presents dramatically. This may result in more subtle signs being missed and patients being misdiagnosed. The busy ER does not always allow for subtle clinical signs to be noted and, thus, cardiac injury should always be suspected in a patient who has sustained a stabbed chest (5).

With the plethora of investigations for this injury, it is difficult to know when to apply specific tests, especially in situations where they are not readily available. Any algorithm for assessment of these patients should bear the above in mind. This may call for clinical decision making or referral to other institutions for further assessment. Even in specialist centres, expertise for less invasive TTE may not be available in the middle of the night. Subxiphoid window is an invasive test, especially for injuries that do not penetrate past subdermal tissue. Therefore, blanket statements in the literature that all stabbed chests must be fully investigated (5) can prove logistically impossible.

How then can the haemodynamically stable patient with a penetrating chest injury best be managed? This question is more pertinent than ever in the resource-constrained and expertise-limited situation of South Africa. Therefore, this study aimed to investigate whether there is a readily available test for the diagnosis of penetrating cardiac injury in haemodynamically stable patients to enable accurate management of these patients before they decompensate.

Chapter 2 – LITERATURE REVIEW

A patient with a penetrating cardiac injury can present anywhere on a clinical spectrum from complete haemodynamic stability to cardiac arrest (9). The diagnosis of a penetrating cardiac injury could be made immediately in the case of a haemodynamically unstable patient requiring emergent treatment, such as a sternotomy or thoracotomy. In the case of the patient who responds to fluid resuscitation or presents with stable vital signs, several clinical and special investigations are at the disposal of the clinician to determine penetrating cardiac injury. These investigations are discussed in the following sections.

2.1 Clinical examination

Suspicion for a penetrating cardiac injury should be raised in any patient with a chest lesion (5). Patients with a parasternal or precordial penetration have a 60% chance of cardiac injury (14). The precordium is defined as the area between the midclavicular lines, below the clavicles and above the costal margin. Moreover, 85% of confirmed cardiac injuries result from lesions in this area (15).

A cardiac injury can present in any of 3 scenarios:

1) *Cardiac tamponade*: Blood collects in the pericardium and tamponades the heart (5).
2) *Haemothorax*: if the laceration to the pericardium communicates with the pleura, the heart will bleed freely into the pleural cavity (5).
3) *Haemopericardium*: if the myocardial laceration clots and seals quickly, then the heart will not bleed freely into the pericardium and tamponade but may bleed at a later stage (4, 8, 16). This is more likely with a thick walled, low pressure chamber

such as the right ventricle (4, 6) and was found in almost 25% of patients (4). There is slightly more association with patients who present in a haemodynamically stable condition with *Harris et al* reporting 48% of haemodynamically stable patients having clotted lacerations as opposed to 17% of moribund patients(4). Up to 2/3 patients with this injury will later bleed requiring surgery (4).

Each scenario has a different clinical presentation and implication on special investigations and will be discussed further below.

The clinical signs of tamponade consist of Beck's triad of hypotension, high central venous pressure and muffled heart sounds on chest auscultation (17). Approximately 80% of patients who have sustained a penetrating cardiac injury and survive to reach hospital will present with some degree of cardiac tamponade (5). The incidence of the latter is so high as cardiac tamponade is thought to confer some degree of protection to the patient in tamponading the bleed (9, 14, 18). Conversely, some data suggests that tamponade has no effect on outcome (19) and, if the tamponade is too tense, may even lead to diastolic compromise (5) and acute failure. Since the literature offers no consensus, it seems reasonable to conclude that tamponade seems to offer protection up to a point, past which it is deleterious (5, 9).

Beck's triad will be elicited in 10-90% of tamponade cases (5, 7, 16). However, it can present subtly and easily be missed (6, 20) and the raised central venous pressures of pericardial tamponade may be masked by the low pressures of shock (5). Therefore, the clinical identification of cardiac tamponade may be found in as few as 22% of patients with cardiac injury (6).

Another sign of cardiac tamponade is neck vein distension on expiration, or Kussmaul's sign, which is found in a minority of patients (9).

Haemothorax presents with reduced breath sounds on the affected side and dullness to percussion. In the case of penetrating thoracic injury, this poses a diagnostic dilemma: where does the bleeding come from? If the origin is cardiac, then most patients' present haemodynamically unstable as the heart is bleeding freely into the thoracic cavity (5). If a haemothorax is found in a patient with a penetrating cardiac injury, it can further confuse diagnosis as ultrasound and echocardiography can be less reliable (21, 26)(see below).

Patients who present with cardiac stab wounds which spontaneously seal (clot), present on a spectrum anywhere between no clinical signs to overt tamponade. Essentially these are patients with tamponade and a clot and do not differ clinically.

Therefore, a small population of patients with a penetrating cardiac injury will present with measured normal haemodynamic parameters and without any obvious clinical signs to suggest their occult lesion (6). The true incidence of such patients is unknown and they may decompensate later (10, 16, 22). Admission and observation of these patients for clinical deterioration is futile as decompensation may be precipitous and disastrous (4). On the other hand, some patients may not deteriorate until days later (22) and even present to the mortuary (11). Since these injuries are by definition "missed", it is imposiible to assess the number of patients who deteriorate. All that can be said is that a missed injury can be fatal (10, 11, 16, 22).

It is not possible to predict which patients will deteriorate and conservative management can have lethal consequences (4). The only risk factor for late deterioration has been found to be an increased incidence of delayed pericarditis in patients who underwent a pericardiocentesis (4).

The information above highlights the importance of special examinations for diagnosis. Even in the haemodynamically stable patient this may not be as reliable or simple as is often assumed.

2.2 Chest x-ray (CXR)

The performance of a chest x-ray (CXR) is mandatory in all patients who present to the emergency room(ER) following trauma to the chest. Pericardial tamponade may present as a globular heart shadow or widened superior mediastinum (5, 6), signs which may be missed on a supine film. Pneumopericardium is also a characteristic finding on an CXR for cardiac injury (5). Haemothorax can be seen easily in the erect film but may be missed if the haemothorax is bilateral on a supine view. CXR findings may, however, be non-specific and have a true positive rate of 50% (5). Some studies have found CXR to be falsely negative in 80% of patients with cardiac injury (23). No recent data for the predictive value of a CXR in penetrating cardiac injury exists. A normal CXR, therefore, does not rule out a cardiac injury (24).

2.3 Electrocardiograph (ECG)

Electrocardiography (ECG) is a very non-specific investigation for penetrating cardiac injury (16) but demonstrates better sensitivity and specificity in patients with injury to the coronary vessels when compared with myocardial and pericardial injury only (25). Typical findings include low voltages, S-T segment elevation or inverted T-waves (5, 24). However, normal ECGs have been found in 85% of penetrating cardiac injury cases (26, 27).

2.4 Serum markers

Troponin T (Trop T) is a protein component of cardiac muscle and a useful marker for diagnosing myocardial ischemia. Raised Trop T levels have occasionally been found in patients with penetrating cardiac injury (25). These levels are not regularly tested as the time from drawing blood to obtaining the result may be excessive and it is unknown whether the enzyme leak will take time to become detectable by serum assay (as is the case with myocardial infarct) or whether the serum levels would be immediately measurable. Upon immunocytochemical examination of weapons involved in cardiac assault, Trop T has been shown to be present on the blade (28). This may point to a more immediate rise in titres than is seen following ischaemic cell lysis in myocardial infarction. All of the above studies were performed at a time when Troponin I and Highly Sensitive Trop T were not available. It is not known what relevance these newer tests would have. Immediate point-care-testing for Trop T is available in most ERs now.

Troponin I (Trop I), another cardiac protein, has recently been introduced as a marker of myocardial ischaemia but has not been investigated in traumatic cardiac injury.

2.5 Ultrasound (US)

Focused assessment with sonography in trauma (FAST) is used to demonstrate pericardial fluid and is accurate in the absence of haemothorax (4, 29). Harris *et al*. demonstrated a false negative rate of 11% with FAST in patients with penetrating cardiac injury and no haemothorax (4). Other studies suggest approximately 100% sensitivity and accuracy of cardiac ultrasound (30, 31). It must be borne in mind, however, that decompression of the pericardial sac into the pleural cavity can result in a falsely negative test (32).

Approximately 80% of patients who survive a stabbed heart long enough to reach the ER will have some degree of pericardial tamponade (5) and 10-90% of these will have clinical signs discussed above (5, 7, 16). Patients with a normal ultrasound but clinical signs of tamponade should be escalated for farther investigation and treatment (4). Patients with normal vital signs and a negative cardiac ultrasound are suitable for non-operative management (4). However, approximately 20% of patients with a stabbed heart will not have tamponade (5).

2.6 Echocardiography

Echocardiography (ECHO) has gained popularity as an accurate diagnostic tool in penetrating cardiac injury and provides additional information on internal cardiac injuries

(26). Therefore, ECHO has a benefit over US in that it does not only identify pericardial fluid but also specific injury sites. Initial studies showed that ECHO had a sensitivity of 56%, specificity of 22% and accuracy of 90% (21). However, when patients with concurrent haemothoraces were excluded from this group, the sensitivity rose to 100%, specificity to 91% and accuracy to 91% (21). This compares favourably with subxiphoid pericardial window.

Two types of ECHO are available:

1) The trans-oesophageal echocardiogram (TEE) provides a high degree of resolution (7, 14, 21, 33, 34). It can be used not only to diagnose a stabbed heart but also to delineate intracardiac injury (33) and complex multiple injury (35). This is especially useful intraoperatively.
2) The transthoracic echocardiogram (TTE) is proving to be very useful as it is not as invasive as a TEE but is unable to describe complex injuries with as high a degree of resolution (26, 33). FAST, TEE and TTE are operator dependant and require specialised training and expensive equipment which may not be available in the emergency department at the time of presentation of the patient (16).

2.7 Computer Assisted Tomography (CT)

Computer assisted tomography (CT) has been mentioned in the literature for use in the diagnosis of haemopericardium in haemodynamically stable patients (8) but the sensitivity and specificity of this modality has not been assessed in regards to cardiac injuries. It is very useful in the diagnosis of associated injuries, for example to the lungs and great vessels) (8).

CT is not used routinely in our centre to diagnose cardiac injury. It is used to delineate other mediastinal and thoracic trauma in haemodynamically normal individuals who have sustained a penetrating wound of the chest. Our experience has been that the risk of sudden decompensation and death in the CT scanner, which is located some distance from the resuscitation bays, negates the benefit of information that may be obtained in a patient where a cardiac injury is suspected or already diagnosed. These patients are investigated either in the resuscitation bay or explored in theatre.

2.8 Pericardiocentesis

Pericardiocentesis has largely fallen away as a diagnostic modality in penetrating cardiac trauma. From the early description as a life-saving diagnostic manoeuvre (36), it's use has largely been discarded as false positive rates may be as high as 50% and false negative of 37% with potential for (additional) damage to the heart (5, 6). There is a place for pericardiocentesis as a temporizing measure before definitive management can be instituted (38). More recently, echocardiographic and ultrasound guidance of pericardiocentesis catheter placement has been introduced showing reduction in complications and more accurate placement (39). The benefit of visualizing the location of the fluid allows the clinician to place the pericardial catheter accurately and without added injury to the heart (38). There may exist a purely diagnostic function for pericardiocentesis if the origin of the pericardial fluid is in doubt (eg tuberculosis, malignancy etc).

2.9 Subxiphoid window

As a diagnostic tool, subxiphoid window is the gold standard in penetrating cardiac injury. It is a reliable, minor procedure that requires a general anaesthetic (9). However, it requires the presence of blood in the pericardium to be positive and some authors have reported up to 20% false negative findings where cardiac injury existed without haemopericardium (37). There has been criticism in well organised trauma centres that it delays definitive repair (5) but this is probably negligible.

ECHO provides similar results to pericardial window (7, 9, 21) and is less invasive. This has led to decreased use of pericardial window as ECHO technology has been introduced (9).

2.10 Thoracoscopy

Thoracoscopy has a sensitivity of 100% and a specificity 96% which makes it an accurate tool with the additional advantages of assessing other thoracic structures that may have been injured and allowing for repair of injuries found (25). However, it requires single lung ventilation and specialised skills and equipment. It can, therefore, only be performed on stable patients and in centres equipped for such procedures (25). There are no reports in the literature of thoracoscopic repair of cardiac injuries.

2.11 Thoracotomy

Thoracotomy allows both full assessment and management of penetrating cardiac injuries. It is very invasive and carries significant complications (6). Its use should probably be reserved for management of cardiac injury rather than diagnosis as it is extremely invasive. However, thoracotomy alone does not provide information on intracardiac injuries and intraoperative TEE may be employed to map these (33, 35).

CHAPTER 3 – AIM OF THE STUDY

A number of cardiac injuries are being missed as they present with a penetrating wound to the chest in a haemodynamically normal patient and with no clinical signs of cardiac tamponade. Therefore, this study aimed to assess whether there is readily available test for the diagnosis of penetrating cardiac injury in such haemodynamically stable patients so as to accurately manage them before they decompensate.

Specifically, the objectives of this study were:

1) To determine the usefulness of the clinical assessment of penetrating chest injury, specifically referring to Beck's triad and Kussmaul's sign, in identifying cardiac injury.
2) To determine the usefulness of special investigations in the diagnosis of penetrating cardiac injury, specifically CXR, ECG, ultrasound, Trop T levels, Trop I levels, cardiac enzymes, CT and ECHO.
3) To provide a simple and useful algorithm with which to approach these patients.

CHAPTER 4 – METHODOLOGY

At Charlotte Maxeke Johannesburg Academic Hospital (CMJAH) Trauma Unit, all patients who have sustained a penetrating injury to the chest are assessed in resuscitation bays and findings are recorded on a resuscitation form (see appendix A). Each resuscitation form consists of four pages. The first page captures the demographic data for the patient as well as the mechanism of injury, mode of transportation, pre-hospital vital signs and arrival times. The second page captures the patient's vitals on arrival, airway, breathing and circulatory control, systemic examination, resuscitation fluid volume and special investigations. Page three captures a schematic representation of the injuries, arterial blood gasses (ABG) and neurological assessment. The last page consists a flow chart of vital signs and fluid and drugs administration during stay in the ER. Laboratory tests were followed up on the National Health Laboratory Services (NHLS) database.

A retrospective analysis was conducted of the resuscitation form for each patient who sustained a penetrating injury to the chest and presented to CMJAH Trauma Unit from 1 January 2007 to 30 June 2010.

Patients were included who had sustained penetrating thoracic trauma. Data was compared between patients with and patients without a proven cardiac injury. As it is not protocol to observe penetrating cardiac injury at CMJAH, cardiac injury was only deemed proven at open surgery. Patients are observed in the ER for 6 hours and suspicious injuries were admitted for observation as necessary. These cases were brought for follow up at a clinic 1 week after discharge. Mortuary records were unavailable for missed injuries and no patient was documented to have returned to our unit with a complication in the study period.

During analysis, it became clear that no proven cardiac trauma was found in any patient in whom the chest wall injury had occurred outside the precordium (the precordium was defined as the area between the midclavicular lines, below the clavicles and above the costal margin). Therefore, patients with these injuries were excluded.

The following parameters were examined (see Appendix B):

1) Demographics: age; sex; mechanism of injury (stab/gunshot)
2) Clinical: anatomical site of injury; Beck's triad; Kussmaul sign
3) Special investigations: CXR; ECG; ultrasound; Trop T; Trop I; cardiac enzymes; CT; ECHO

Since the above tests are interpreted qualitatively, a univariate analysis was performed to determine the sensitivity (sens), specificity (spec), positive (PPV) and negative (NPV) predictive values. Each of these are presented for every parameter and analysed in search of a useful tool.

Each categorical data set was subjected to a Chi-squared test to determine statistical significance. If a particular data set was not large enough in numbers, the Yates correction was employed to one degree of continuity. If the total number of patients was less than 10, the Fisher Exact test was applied. The p-value was then calculated and presented with the results. This statistical analysis was performed using Clinstat GmbH, Cologne, Germany. A p-value of less or equal to 0.05 was considered to be statistically significant.

As this is a retrospective study of data prospectively collected in the CMJAH Trauma Unit, some data points were missing. Where this was found to be the case, statistical tests for these variables were performed only on patients with the available data points. Each investigation was analysed separately resulting in each investigation having a different set of patients depending on which patients had the necessary data points. (eg only 165 patients had an ECG. Therefore only 165 out of a possible 816 patients were examined for ECG whereas 782 patients had CXR). This was deemed appropriate as no multivariate analysis was conducted.

Ethical clearance was approved unconditionally from the Human Research Ethics Committee (HREC) of the University of the Witwatersrand on 2/02/2009 (ref number M090122). See Appendix C.

CHAPTER 5 – RESULTS

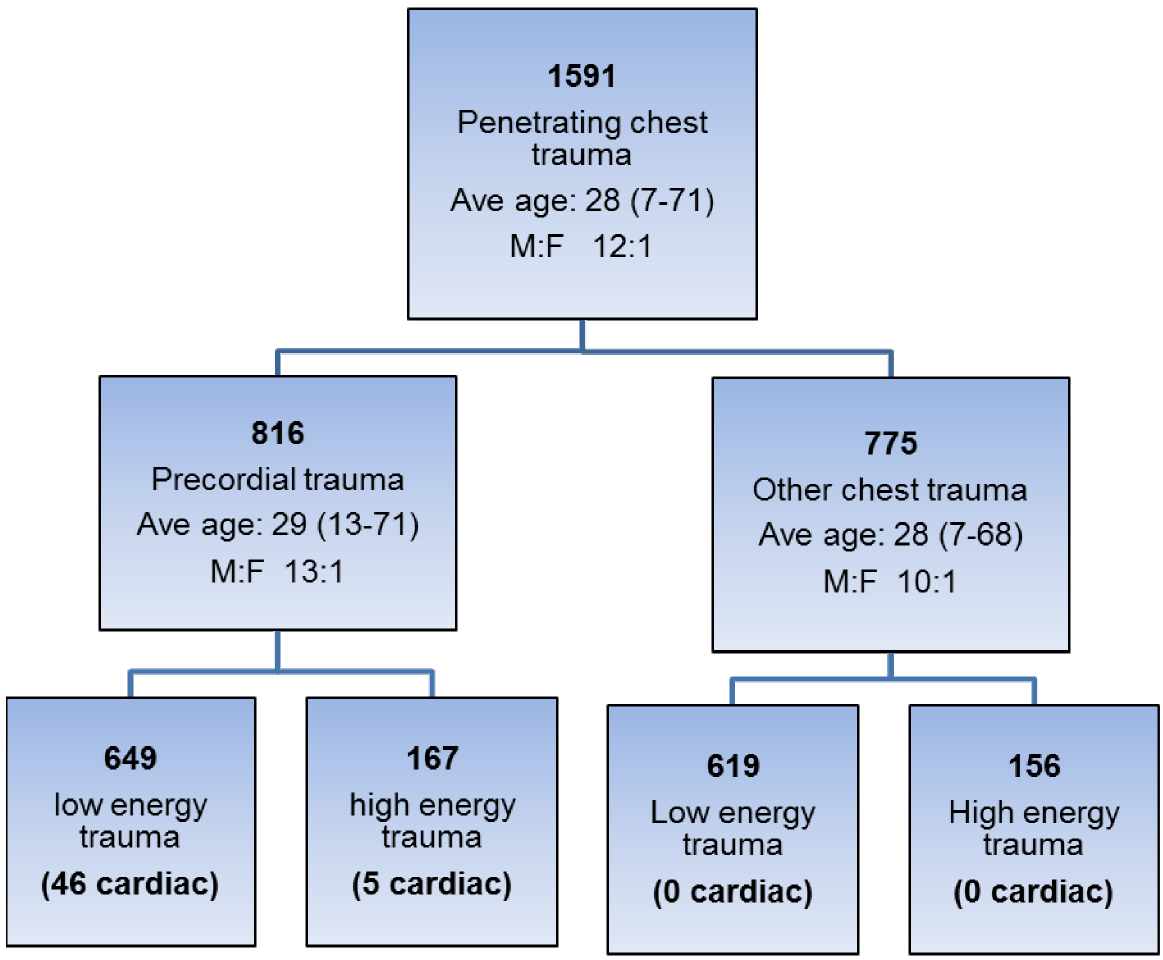
5.1 Demographic data

From January 2007 to the end of June 2010, a total of 7 781 priority one (P1) patients presented to the trauma unit of CMJAH. The resuscitation forms for these patients were assessed and 1 591 were found to have sustained a penetrating injury to the chest (20%). These patients had an average age of 28 years (range 7-71) and a male to female ratio (M:F) of 12:1. Of these, 1 268 patients suffered a stab injury and 323 were shot.

The diagram in Figure 5.1 shows the breakdown of the 1 591 patients with penetrating chest trauma. Of these patients, 816 had injuries to the precordium with an average age of 29 years (range 13-71) and a M:F of 13:1. The precordium is defined as the area between the midclavicular lines, below the clavicles and above the costal margin. Furthermore, 649 patients were stabbed and 167 shot. Chest trauma outside of the precordium was found in 775 patients with an average age of 28 years (range 7-68) and M:F of 10:1, 619 suffering low energy injury and 153 high energy.

Cardiac injury was sustained in 51 patients with an average age of 30 years (range 17-57) and M:F 25:1. Of these, 46 patients were stabbed and 5 shot. As cardiac injuries were found only in the group of patients who had sustained a precordial injury, the group without penetration of the precordium was removed from further analysis.

The study group thus consists of 816 patients with precordial trauma and 6.25% incidence of cardiac injury.



Number of cardiac injuries indicated in bold and bracketed

Figure 5.1. Subsets of study patients presenting with penetrating chest trauma based on anatomical site of injury

5.2 Clinical data

5.2.1 Beck's triad

Data for clinical examination of patients is filled in on a table on the resuscitation form with a separate space for chest and cardiac examination. Tick boxes to indicate positive or negative findings are included for muffled heart sounds. If the box was not ticked, it was assumed that the treating doctor did not look for this sign and the patient was removed from the data set.

The last page of the resuscitation form contains a flow chart of vital signs. If a systolic blood pressure of less than 90 mmHg was recorded at any time in the resuscitation bay, then the patient was regarded as having had an episode of hypotension.

In those patients who had a CVP catheter inserted, the CVP was regarded as positive for tamponade if it was greater than 15 cmH_2O as per protocol at CMJAH.

When the data for Beck's triad of signs in cardiac tamponade were analyzed, only 5 patients had a full complement of data. This resulted in a sensitivity of 33%, specificity of 100%, positive predictive value (PPV) of 100% and negative predictive value (NPV) of 50%. These findings are not statistically significant with a p-value of 1.000.

If the CVP level was substituted by the clinical sign of distended neck veins, then 19 patients have a full complement of data. This results in a sensitivity of 25%, specificity of 100%, PPV of 100% and NPV of 83.3. These findings are not statistically significant with a p-value of 0.4667.

We also assessed the individual components of Beck's triad separately. These individual results for sensitivity, specificity, PPV and NPV of the muffled heart sounds, hypotension, CVP and distended neck veins are shown in Table 5.1 below.

Hypotension and raised CVP are objective signs while the presence of muffled heart sounds is a subjective sign that can be difficult to elicit especially in the noise and excitement of the resuscitation room. When Beck's triad using distended neck veins was recalculated with the distant heart sounds removed, a total of 726 patients were included in the analysis, giving a sensitivity of 23.1%, specificity of 98.1%, PPV 40.9% and NPV 95.7% (Table 5.1). These results are significant with a p-value of <0.0001.

5.2.2 Kussmaul's Sign

As with muffled heart sounds, distended neck veins are included in a tick box. Kussmaul's sign is elicited with neck vein distension on expiration. The resuscitation form only allows for recording neck vein distension. No comment on the relationship of vein diameter to respiration was made. Therefore, Kussmaul's sign was not further assessed in this study

5.2.3 Pulsus paradoxus

Only 1 patient was noted to have been assessed for pulsus paradoxus which was a true negative result.

Table 5.1. Accuracy of using clinical signs in diagnosing cardiac injury

	N	Sens (%)	Spec (%)	PPV (%)	NPV (%)	P-value
Muffled heart sounds	19	75	66.7	37.5	90.9	0.3537
Hypotension	815	30.8	92.0	17.9	95.9	<0.0001
CVP (>15 cm H_2O)	32	62.5	91.7	71.4	88	0.007
Distended neck veins	726	30.8	92	17.9	95.9	<0.0001
Beck's triad	5	33.3	100	100	50	1.000
Beck's using distended neck veins	19	25	100	100	83.3	0.4667
Beck's using distended neck veins but excluding muffled heart sounds	726	23.1	98.1	40.9	95.7	<0.0001
Pulsus paradoxus	1	Undefined	100	undefined	Undefined	Not relevant

Abbreviations: Sens: sensitivity; Spec: specificity; PPV: positive predictive value; NPV: negative predictive value

5.3 Special investigations

Depending on the specific circumstances of the patients involved, certain special investigations were performed according to Trauma Unit treatment protocols and at the discretion of the treating physician. If these were found to indicate cardiac injury, they were recorded as positive. Those not showing cardiac involvement were recorded as negative.

The sensitivity, specificity, PPV and NPV for each was calculated against statistical significance. The special investigations included CXR, ECG, US, echocardiography, CT scan, cardiac enzymes, trop T, trop I, subxiphoid window and pericardiocentesis. These results are discussed below.

5.3.1 CXR

Unless the patient was too unstable on arrival, every patient in the resuscitation area received a CXR once the primary survey was completed. If the CXR at resuscitation was normal and the patient well, it was repeated after 6 hours. A straight left heart border, globular cardiac shadow, widened superior mediastinum or pneumomediastinum was regarded as positive for cardiac injury (and prompted farther investigation of the patient) in this study.

CXR was performed in 782 patients which resulted in a sensitivity of 22.86%, specificity of 97.32%, PPV of 28.57% and NPV of 96.42%. These findings were significant with a p-value of <0.0001.

5.3.2 ECG

ST-segment changes, inverted T waves and low voltages are regarded as positive changes on ECG. A total of 165 patients had an ECG which yielded a sensitivity of 27.27%, specificity of 90.91%, PPV of 17.65% and NPV of 94.59%. These results are, however, not statistically significant with a p-value of 0.1604.

5.3.3 US

FAST including an ultrasound of the pericardium was documented in 203 patients. The presence of any fluid discernible on ultrasound (diagnostic thickness of fluid noted on reports) in the pericardial space was regarded as positive. As no ultrasound machine was available in our casualty, all FAST are performed by a radiologist using a portable ultrasound machine as part of the secondary survey. This gives a sensitivity of 41.67%, specificity of 97.38%, PPV of 50% and NPV of 96.37%. The p-value was <0.0001.

Since review of the literature indicates that the presence of a haemo- or pneumothorax may adversely affect the accuracy of a FAST (4, 30-32), these patients were removed. The sample group then consisted of 100 patients. A sensitivity of 66.67%, specificity of 97.87%, PPV of 66.67% and NPV of 97.87% were found. These results are statistically significant as the p-value was <0.0001.

As no surgeon performed FAST was performed, this modality could not be assessed.

5.3.4 Echocardiography

ECHO is not routinely performed in our unit and as a result only 25 patients had TTE recorded, giving a sensitivity of 100%, specificity of 95%, PPV of 83.33% and NPV of 100% (p<0.0001). As the presence of a haemo- or pneumothorax may reduce the sensitivity and specificity of ECHO (21, 26), the patients without haemo- or pneumothoraces were analysed and a sensitivity of 100%, specificity of 90.91%, PPV of 80% and NPV of 100% were seen (p= 0.001).

5.3.5 CT scan

CT chest was reported in 138 patients' resuscitation forms. The sensitivity was 66.67%, specificity 94.81%, PPV 22.22% and NPV 99.22% (p = 0.002). CT angiography was not distinguished from routine CT chest with intravenous contrast.

5.3.6 Cardiac enzymes

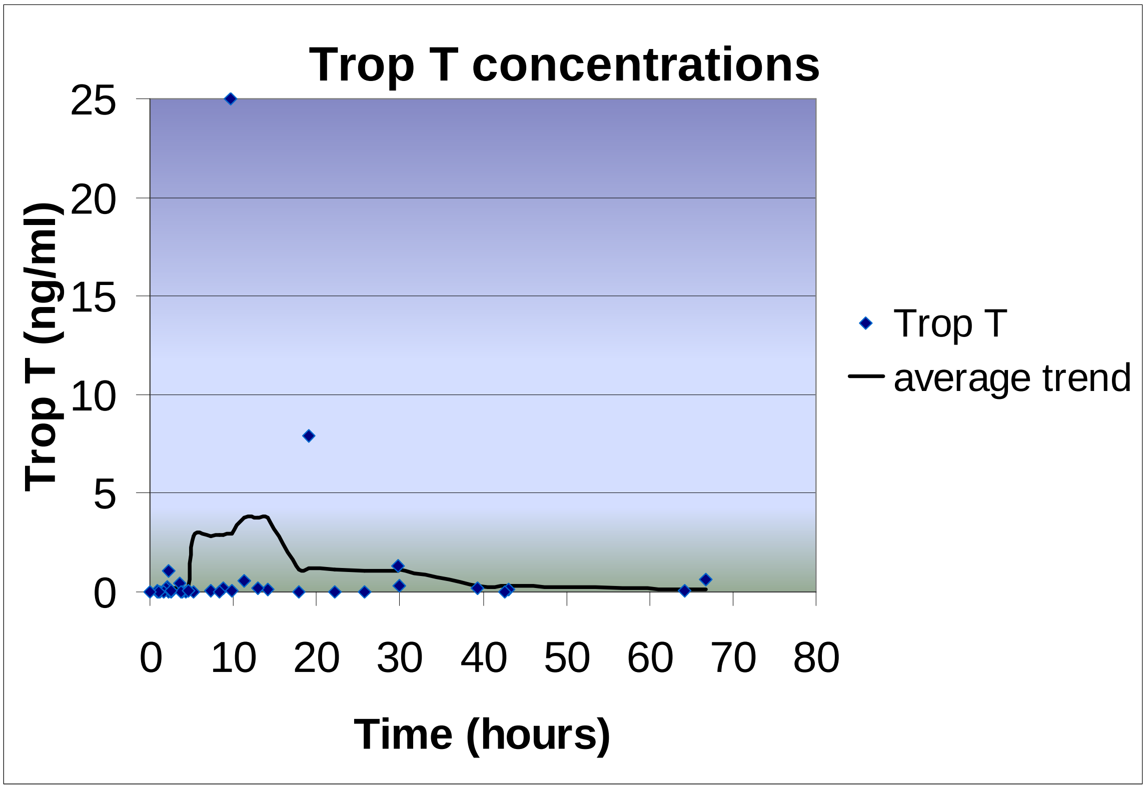
Creatinine kinase (CK) is positive if more than 174 unit/L. If the M and B isomers are more than 24 unit/L and make up more than 6% of the CK concentration, it was regarded as cardiac specific.

Cardiac enzymes were taken in 206 patients. CK gives a sensitivity of 90%, specificity of 15.34%, PPV of 15.34% and NPV of 90%. Of these, 174 patients have M and B fractions of CK (CKMB) measured. The CKMB, therefore, gives sensitivity 85.19%, specificity 29.05%, PPV 17.97% and NPV 91.49%. Where this fraction was more than 6%, then the sensitivity was 74.07%, specificity 33.11%, PPV 16.81% and NPV 87.50%. None of these results are statistically significant.

5.3.7 Troponin T

A total of 255 patients with penetrating precordial trauma had a Trop T taken at resuscitation. Trop T showed a sensitivity of 59.26%, specificity of 93.86%, PPV of 53.33% and NPV of 95.11 ($p<0.0001$). These results were calculated using only the first chronological Trop T in the patient's list of blood tests.

Nine patients with cardiac injury had more than one Trop T blood test taken in the resuscitation bay. Of these, most had a string of varying positive results but 4 initially presented with a negative Trop T and later developed a positive result. There is no space on the resuscitation form to note the time of incident. However, if the time from arrival at the ER to the time of Trop T sampling was plotted against the value of the Trop T, it was found that there is a peak in the trop T value at approximately 10 hours (n=27) (see Figure 5.2).



**Please note: this graph includes postoperative values*

Figure 5.2. All (including sequential and post-operative) Trop T levels plotted against time from arrival at ER (n=39; 95% CI 0-2.3). Trop T values <0.03 are plotted as 0.

The average time from arrival at the ER to theatre for the cardiac injury group with Trop T levels taken was 162 minutes (range 10-720). If the postoperative results are removed and the Trop T readings are plotted against the first 6 hours from arrival in the ER, then a peak is found at 3 hours. Therefore, as in myocardial infarction, it takes time for the enzyme leak to become significant enough to be measured by serum assay.

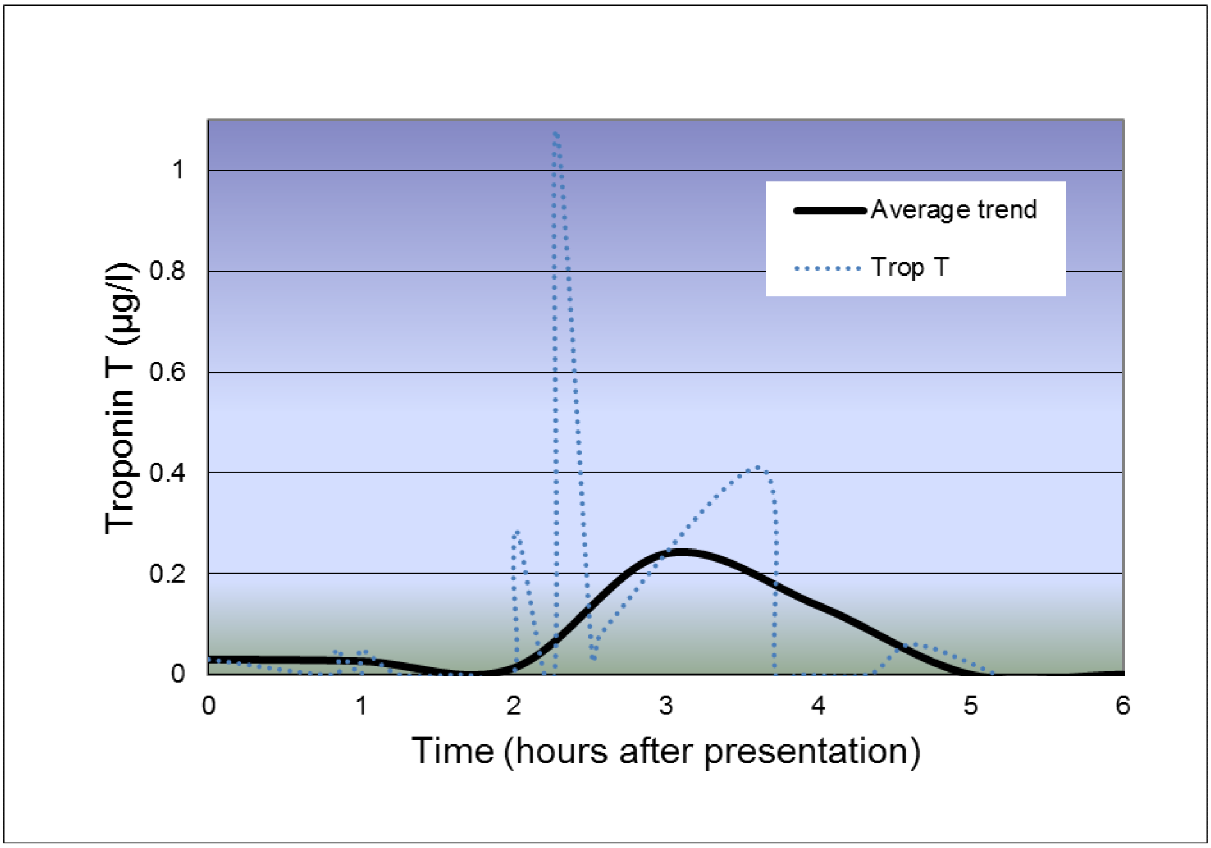


Figure 5.3. Trop T levels (sequential included) plotted against time for the first 6 hours from arrival in the ER (post op results removed) (n=29; 95% CI 0-3.0). All Trop T values <0.03 are plotted as 0.

If the accuracy data are calculated using Trop T blood tests taken hourly up to 6 hours post arrival at the ER with post-operative results removed, then the sensitivity increases to 100%, the specificity remains 94%, the PPV drops to 50% and the NPV is 100% at 6 hours. All of these values are statistically significant (p <0.0001). This indicates that by 6 hours post arrival at the ER, there are no false negative results.

Table 5.2. Accuracy of using hourly Trop T levels in all patients from arrival at ER

	Trop T after arrival at ER						
	> 0hr	> 1hr	> 2hr	> 3hr	> 4hr	> 5hr	> 6hr
N	260	257	254	248	245	243	242
Sens (%)	71.88	72.41	80.77	80.00	88.24	93.33	100.00
Spec (%)	93.86	93.86	93.86	93.86	93.86	93.86	93.86
PPV (%)	62.16	60.00	60.00	53.33	51.72	50.00	50.00
NPV (%)	95.96	96.40	97.72	98.17	99.07	99.53	100.00
p-value	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

Abbreviations: Sens, sensitivity; Spec, specificity

Only 6 patients in the cardiac injury group were treated at a smaller, peripheral institution or a private hospital before being transported to the CMJAH Trauma unit. These patients, therefore, had a significant delay before arrival at the ER. If the Trop T values for these patients are excluded from the data set, then, by 4 hours, the sensitivity is 100%, the specificity remains at 94%, the PPV drops to 46% and the NPV is 100% (p <0.0001). By 6 hours, the false negative rate was 0 (Table 5.3). It is worth noting that this group contained no gunshot injuries (as the only patient to have sustained a cardiac injury from a gunshot with a Trop T level taken was transferred from a private institution and was, therefore, removed).

Table 5.3. Accuracy of using hourly Trop T levels from arrival at ER with patients transferred from other institutions and gunshot injuries excluded.

	> 0hr	> 1hr	> 2hr	> 3hr	> 4hr	> 5hr	> 6hr
N	252	250	247	243	241	240	240
Sens (%)	70.83	72.73	84.21	86.67	92.31	100.00	100.00
Spec (%)	93.86	93.86	93.86	93.86	93.86	93.86	93.86
PPV (%)	54.84	53.33	53.33	48.15	46.15	46.15	46.15
NPV (%)	96.83	97.27	98.62	99.07	99.53	100.00	100.00
P-value	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

Abbreviations: Sens, sensitivity; Spec, specificity

5.3.8 Troponin I

Only 6 patients had a Trop I taken as this test was not available in CMJAH until towards the end of the study. A sensitivity of 100%, specificity of 80%, PPV of 50% and NPV of 100%. These results were not statistically significant.

5.3.9 Subxiphoid window

Subxiphoid window was performed on only 15 patients with a sensitivity of 100%, specificity of 77.78%, PPV 75% and NPV 100%. This was not statistically significant with a p-value 0.151.

5.3.10 Pericardiocentesis

Although pericardiocentesis was not part of the trauma protocol for penetrating cardiac injury at CMJAH, 1 patient was subjected to this procedure. This proved negative in a patient with a stab right ventricle.

Table 5.4. Accuracy of all special investigations in diagnosing cardiac injury.

	n	Sens (%)	Spec (%)	PPV (%)	NPV (%)	p-value
CXR	782	22.86	97.32	28.57	96.42	**<0.0001**
ECG	165	27.27	90.91	17.65	94.59	0.1604
US	203	41.67	97.38	50.00	96.37	**<0.0001**
TEE	0	NA	NA	NA	NA	NA
TTE	25	100.00	95.00	83.33	100.00	**0.0001**
CT	138	66.67	94.81	22.22	99.22	**0.002**
CK	206	90.00	15.34	15.34	90.00	0.6242
CKMB	175	85.19	29.05	17.97	91.49	0.1936
%	175	74.07	33.11	16.81	87.50	0.6101
Trop T	255	59.26	93.86	53.33	95.11	**<0.0001**
Trop I	6	100.00	80.00	50.00	100.00	0.6985
Subxiphoid window	15	100.00	77.78	75.00	100.00	0.151
Pericardiocentesis	1	0.00	NA	NA	0.00	NA

Abbreviations: Sens, sensitivity; Spec, specificity

CHAPTER 6 – DISCUSSION & CONCLUSION

Although the true impact of missed occult cardiac injury is not known (4), it can have a disastrous and even lethal outcome for the patient (11, 13, 22). How then, is the clinician to approach the haemodynamically stable patient with a penetrating precordial wound? How reliable is clinical assessment and does this patient warrant sophisticated, expensive investigation?

The vast majority of penetrating cardiac injury occurs within the precordial space. Although cardiac injury has been reported outside this area, our series of 1591 patients shows no such case. For this reason, injury outside the precordium was not assessed. The incidence of cardiac injury in penetrating precordial trauma was 6% in this study.

6.1 Clinical evaluation

Due to the retrospective design of this study and the lack of an appropriate area on the resuscitation form to record cardiac tamponade as a diagnosis, the relationship of Beck's triad to tamponade could not be assessed. However, the usefulness of Beck's triad in diagnosing cardiac injury was assessed whether the patients presented with tamponade or not.

An episode of hypotension (systolic blood pressure < 90mmHg) in the resuscitation bay gives a specificity of 92% and NPV of 95.9% ($p < 0.0001$) in predicting penetrating cardiac injury. However, as the cardiac injuries in this study were only confirmed at surgery, this episode may represent haemodynamic instability on presentation or early decompensation rather than diagnostic accuracy. The time to hypotensive event was also not recorded. Intuitively, waiting for haemodynamic decompensation is counterproductive as this converts a controlled pathology into one complicated by late stage shock.

This study showed that neck vein distension could be a useful screening tool for penetrating cardiac injury with a sensitivity 92% and NPV 95.9%, but it should be noted that it may be absent in cases of shock leading to increased false negative rates. Demetriades et al showed that neck vein distension occurred in only 22% of cases of tamponade (20). This is also a subjective clinical sign that may easily be missed (17, 20) if the clinician is not sufficiently vigilant or the ER is particularly busy and disruptive (17, 20).

If hypotension and neck vein distension are combined, then the sensitivity increases to 98.1% and the NPV to 95.7% although the data has the same limitations discussed above.

Review of the literature varies with respect to clinical assessment of cardiac injury with some authors stating that clinical signs are present in as many as 90% of cases (20) and others finding signs in fewer than 50% (9). Clinical assessment of penetrating precordial injury alone is not sufficient to diagnose occult cardiac involvement (otherwise it would not be called “occult”). No statistically significant clinical tests could be found for cardiac injury in our series. However, this does not mean that patients should not be carefully examined, looking for signs of penetrating cardiac involvement, as many clinical signs will result in further workup and diagnosis.

6.2 Special Investigations

Performance of a CXR is mandatory in all patients with a thoracic stab wound as other intrathoracic organs (as well as the heart) must be assessed. It provides a sensitivity of 22.86%, spec of 97.32%, PPV of 28.57% and NPV of 96.42% ($p < 0.0001$). Nagy *et al* found that CXR could not exclude cardiac injury (24) and only 50% of CXR findings were diagnostic of cardiac injury according to another study (5). This data is in conflict with our own which seems to show that CXR, while a poor diagnostic test (poor sensitivity and PPV), may be a possible screening tool (high specificity and NPV). However, the time from presentation at the ER until CXR was not documented in our patients and very non-specific interpretation is employed such as the presence of a straight left heart border, globular cardiac shadow, widened superior mediastinum or pneumomediastinum. This makes interpretation of our data difficult for several reasons:

1) CXR performed early in the disease process may miss a cardiac injury that would become apparent after 5, 10, 20 etc minutes.
2) Any positive sign would have resulted in further testing with subsequent review of the CXR in the ER and alteration of the results recorded on the resuscitation form.

With the above in mind, our data must be interpreted with caution as the immediate interpretation and response to the CXR findings are not recorded on the resuscitation form (multiple CXR also not noted). Therefore, no significant conclusion can be drawn about the usefulness or not of CXR in the diagnosis of penetrating cardiac injury as the data may be incorrect.

All ultrasound done in this study was performed by a radiologist on a portable machine. Surprisingly, the sensitivity is 41.67% and PPV 50%. However, it has a specificity of 97.38% and NPV of 96.37%. Most guidelines currently promote surgeon performed ultrasound which is not assessed in this study. According to this data, if a radiologist is willing and able to perform a cardiac ultrasound, a substantial number of pericardial fluid collections will be missed.

Our data suggest that TTE is an excellent test with a sensitivity of 100%, spec 95%, PPV 83.3% and NPV 100% ($p = 0.0001$). In this series, due to difficulty of obtaining this test, only 25 (3%) patients underwent TTE. This represents a logistical problem with this test as no equipment for surgeon performed TTE exists at CMJAH and cardiology services for this are not available after hours. As TEE requires specialist equipment and expertise, it is not readily available. This alone decreases the practical relevance of TEE for diagnosis of cardiac injury at CMJAH.

ECG and raised cardiac enzyme assays are not statistically significant in this study.

CT chest was able to rule out many cardiac injuries with a specificity of 94.81% and NPV of 99.22% ($p = 0.002$). This fell out of the highly significant cutoff for this study ($p<0.001$) but would be considered by most to be significant (<0.05). These findings have been identified previously in the literature (8) and CT has the added benefit of assessing injury to other thoracic organs. CT angiography was not distinguished from routine CT chest with intravenous contrast.

Troponin T taken on admission to the ER gives a sensitivity of 59.26%, spec of 93.86%, PPV of 53.33% and NPV of 95.11% ($p < 0.0001$). If consecutive pre-operative results are assessed for patients that arrive directly from scene (patients transferred from other institution excluded) and calculated hourly from arrival up to 6 hours, then the sensitivity and NPV increase to 100%, the spec to 93.86% and PPV decreases to 46.15% ($p < 0.0001$) at 4 hours. This appears to indicate that Trop T takes time to rise and delay or repetition of testing will increase accuracy. Since the false negative rate drops to 0 after 6 hours, Trop T is a valuable screening tool for cardiac injury.

It is worthwhile to note that Trop T is not part of the protocol for management of these patients and, therefore, no regulated sampling occurred. Therefore, all Trop T samples were taken and repeated at the discretion of the attending clinician.

Trop I was only assessed in 6 patients as it was only introduced to our institution late in the study. However, given the promising results obtained with Trop T, Trop I may prove to be valuable in the future.

Subxiphoid window was only assessed in 15 patients. This is a useful test for the patient with multiple injuries (eg thoracoabdominal stab/multiple stabs) who requires theatre for their other injuries and subxiphoid window is a small but useful addition to their procedure

6.3 Conclusion

Given the above results, a rational approach to the diagnosis of an occult cardiac injury in resource and skills limited South Africa can be formulated. Clinical examination is important to rule out other injuries. It is equally important to diagnose cardiac tamponade which needs to be addressed before a patient decompensates from a haemodynamic perspective.

Special investigations each suffer from problems of availability, expertise and usefulness. A CXR is mandatory in the patient with a penetrating thoracic injury but is not an accurate diagnostic test. US was not a useful diagnostic test in this study. ECG, CE and pericardiocentesis should not be used in the diagnosis of an occult cardiac injury. Subxiphoid window remains the gold standard of investigation although echocardiography is fast being accepted as its replacement. TTE is a good diagnostic test but is limited by availability of equipment and expertise and subxiphoid window is an invasive procedure for the haemodynamically normal patient who may harbor an occult cardiac injury. CT has been shown to be useful in the haemodynamically stable patient but also requires specialised equipment and training. It is also an expansive test but has the benefit of picking up other injuries as well.

Given the usefulness of Trop T as a screening tool and the easy availability of point of care testing in the ER, we suggest an algorithm for the management of haemodynamically stable patients with a penetrating precordial injury.

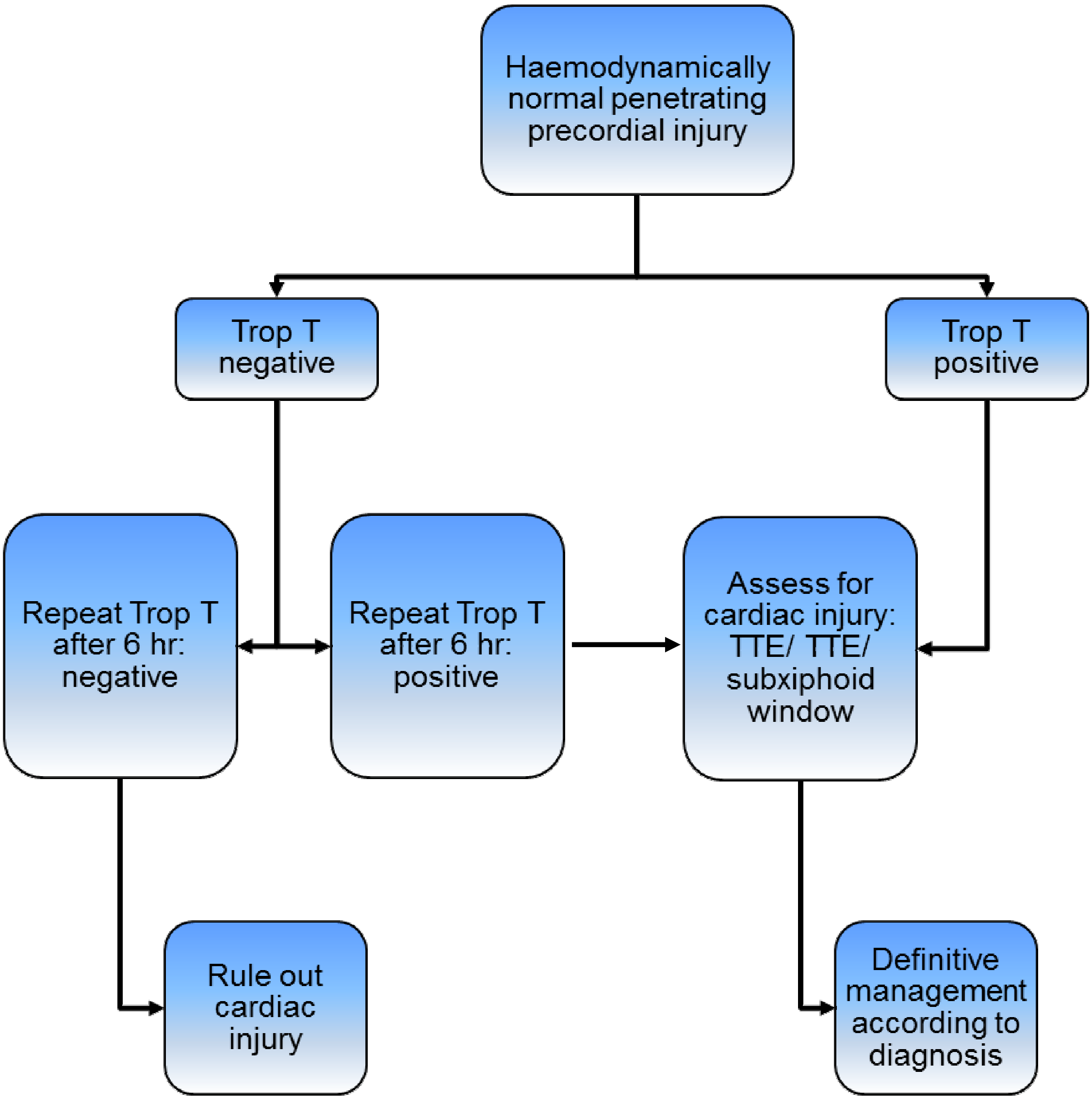


Figure 6.1. Suggested algorithm for assessment of occult cardiac injury.

Trop T is now available as point of care testing at CMJAH. Although this test modality was not present in casualty during this study, it may result in an even better screening of these patients.

CHAPTER 7 – STUDY LIMITATIONS

1) This was a retrospective study with certain data sets missing. The data was collected, collated and analysed by the same person to avoid confusion and errors.
2) Although there were several occult cardiac injuries included in the trial (those that presented late but ultimately required theatre), most of the test group consisted of patients who were diagnosed rapidly and taken to theatre.
3) Diagnosis of cardiac injury was only made at surgery. Therefore, there is the possibility that several occult injuries were missed as no long term follow up was conducted on the patients.
4) The data from all cardiac injury was extrapolated to haemodynamically stable patients.
5) No differentiation was made for high and low energy trauma in analysis (except for Trop T).

A prospective, cohort study has been started at the trauma unit of CMJAH to address these issues.

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APPENDICES

Appendix A: Resuscitation form

MediBank Resus Form www.medibank.co.za

DEMOGRAPHICS / STICKER

Insert New Patient

Arrival date:	**Arrival time:**	**Gender:** Male / Female
First name:	Surname:	**Age:**
Race: Asian / Black / Coloured / White	Residential Suburb:	Hospital number:

Incident

INCIDENT

Case Priority: P1 / P2 / P3

Incident Date:	Blunt / Penetrating / Burn / Medical / Other:
Incident Time:	**How it happened:** Stab / GSW – handgun, rifle, shotgun, AK47 / Hijacking / Assault / MVC – Taxi – driver, passenger, restrained, unrestrained / PVC / MBC / Bicycle / Train / Fall – from height ______ m, same level / Burn – fire, water, steam, petrol / Animal / Other:
Where it happened (suburb):	
Accidental / Self-harm / Assault: stranger / acquaintance / family member / intimate partner / child abuse / elder abuse / rape	
Site: road / home / work / recreational facility / tavern / educational facility / other:	

Pre Hospital

PRE-HOSPITAL

Ambulance number:	EMS Service:	**Crew:** BLS / ILS / ALS	**Doctor present on scene:** YES / NO
Helicopter used: YES / NO	Airlifted from:	Dispatch time:	Scene arrival time:
Scene departure time:	Hospital arrival time:	**Initial** pulse:	**Initial** Sats:
Patient's **own initial** respiration rate:	Assisted ventilation rate:	**Initial** BP:	**Initial** GCS:
GCS Eyes: 1 2 3 4	GCS Verbal: 1 2 3 4 5	GCS Motor: 1 2 3 4 5 6	**Airway intervention:** Nil / O2 mask ________ % / Oral airway / Bag-valve-mask / Oral ETT ________(size) / Nasal ETT ______ (size) / Cric / Other:
IV Access: Peripheral / Jugular / Femoral / Central 1 line / 2 lines	**Fluids infused:** Ringers __________ mls Colloid __________ mls Other:	**Drugs:** Nil / Tramal ____ mg / Morphine _____ mg / Dormicum _____ mg / Scoline _____ mg / Pavulon _____ mg / Other:	**Other:** Nil / Collar / Spine Board / Scoop / TRAC 3 / Splint – arm / Splint – leg / CPR / Defib / Other:
Scene complications:			

Referring Hospital

TRANSFERRING HOSPITAL

Transferred: YES / NO	Transferring Hospital:		**Arranged:** YES / NO
Vitals on **arrival** at Transferring Hospital:			
Pulse:	Patient's **own** respiration rate:	Assisted Ventilation rate:	BP:
GCS at other hospital:	GCS Eyes: 1 2 3 4	GCS Verbal: 1 2 3 4 5	GCS Motor: 1 2 3 4 5 6
Transferring Hospital intervention:		Drugs:	Diagnostic Procedures:
Airway:	IV:		

Emergency Unit Admission

EMERGENCY UNIT TIMES AND RESUS TEAM

Highest level of Scene care: Ambulance BLS / ILS / ALS / Helicopter / Police / Private Vehicle / Walk / Other:	**Arrived from:** Scene / Transferring Hospital / Home / Upgraded	If upgraded to P1 **TIME UPGRADED:**	Resus Team **ACTIVATION** time:
	Primary Survey complete:	**Secondary Survey complete:**	**Resus complete time:**
Consultant Surgeon:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Registrar:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Doctor 1:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Doctor 2:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Sister 1:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Sister 2:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Neurosurgeon:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Orthopaedic Surgeon:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Paediatric Surgeon:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**
Other:		Time present:	**Telephonic / OT / Other Resus / Absent / Not called**

Time to Radiology:	**Time back from Radiology:**	**Time bloods called:**	**Time blood Taken:**	**Time blood Results in**

Emergency Unit Data

EMERGENCY UNIT DATA

Initial Emergency Unit Vitals, Assessment and IV Fluids / Drugs administered:

Initial Pulse:	Patient's **own** initial *respiration* rate:	Assisted *ventilation* rate:	Initial BP:
Temperature:	GCS Eyes: 1 2 3 4	GCS Verbal: 1 2 3 4 5	GCS Motor: 1 2 3 4 5 6
Comorbidity: Asthmatic / Diabetic / Epileptic / Pregnant / Anticoagulants / Hypothermic / Hypertensive / Other:			
Allergies:	Meds:	PMS Hx:	Last Meal:
Alcohol suspected: **YES / NO**	Highest level of **Airway Management:** O2 Mask % / Oral Airway / Bag-Valve-Mask / Oral ETT, size / Nasal ETT, size / Cric / Nil		
Patient's **Total IV Access:** Peripheral 14G / 14G / 16G / 16G / 8.5 / 8.5 / Other: Subclavian L / R / Femoral L / R / Jugular L / R		**Estimated blood loss:** IC Drain **L** ml, **R** ml	**Other estimated blood loss:** ml
RESUS **Crystalloid** type: _________ml infused at end 2' survey	RESUS **Colloid** type: _________ml infused at end 2' survey	**Blood** units ordered:	**FFP** units ordered:
		Blood units given:	**FFP** units given:
Analgesia: Nil / Morphine / Other:	**Sedation:** Nil / Dormicum / Hypnomidate / Other:	**Paralysis:** Nil / Scoline / Pavulon / Other:	Antibiotics:
Cardiac drugs: Nil / Adrenaline / Atropine / Anti-arrythmics / **Other**:			Anti Tetanus:

Procedures Performed *in* the Emergency Unit:

Endotracheal Intubation: YES / NO	**Cric / Trachy:** YES / NO	**ICD L:** YES / NO; X1 / X2	**ICD R:** YES / NO; X1 / X2
Peripheral line inserted: **L** X	Peripheral line inserted: **R** X	Subclavian / Femoral line: **L** X	Subclavian / Femoral line: **R** X
Dressing: YES / NO	**Fasciotomy:** YES / NO	**E R Thorocotomy:** YES / NO	**Bronchoscopy:** YES / NO
Suturing: YES / NO	**Urinary catheter:** YES / NO	**NG / OG tube:** YES / NO	**Eye care:** YES / NO
Other: Nil / **CPR** / Defib / TRAC 3 / Pelvic Sheet / Bair Hugger / Other:			

Diagnostic Investigations and *Findings*:

X-Ray C-Spine:			**IVP:**	YES / NO	Negative / Positive
X-Ray Chest:			**CT Head:**	YES / NO	Negative / Positive
X-Ray Pelvis:			**CT Spine:**	YES / NO	Negative / Positive
X-Ray:			**CT Chest:**	YES / NO	Negative / Positive
X Ray:			**CT Abdo:**	YES / NO	Negative / Positive
X Ray:			**Dopplers:**	LEGS / ARMS	L = R =
Ultrasound Abdo:	YES / NO	Negative / Positive	**ECG:**	YES / NO	Normal / Abnormal
DPL Open / Closed:	YES / NO	Negative / Positive	**Trop T levels:**	YES / NO	Normal / Elevated
Angiogram:	YES / NO	Negative / Positive	**Other:**		Negative / Positive

Complications:

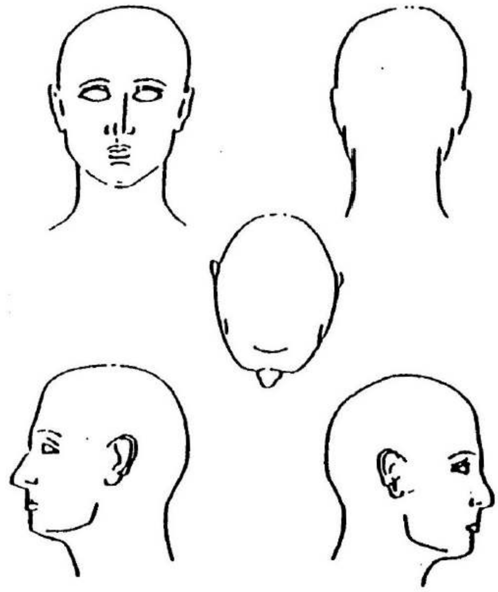
ISS Diagnosis

INJURY LIST / DIAGNOSIS:

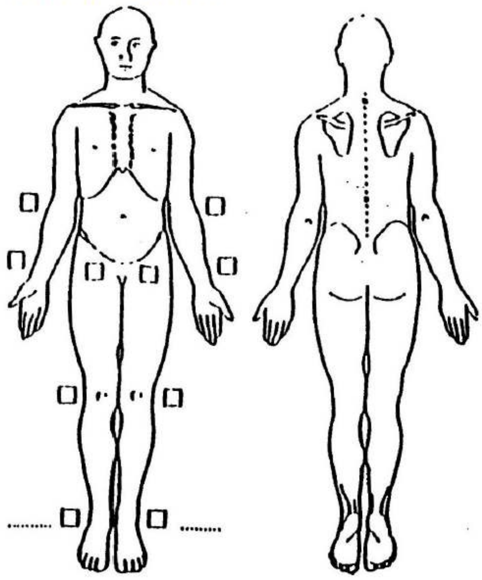
HEAD:	**FACE:** Pupils equal: YES / NO SIZE L (draw) , reactive: YES / NO; Contact lenses: YES / NO SIZE R (draw) , reactive: YES / NO
NECK: **Trachea central:** YES / NO; **Neck veins distended:** YES / NO; Carotid Pulses present: **L** Y / N; **R** Y / N	**THORAX:** **Flail:** **YES / NO**; **Open chest:** **YES / NO**; **Pneumothorax:** YES / NO **L / R**; **Haemothorax:** YES / NO **L / R**; **Haemo-pneumo:** YES / NO **L / R**
ABDO & PELVIC CONTENTS: **Rectal:** **Tone** normal / flaccid, **Blood** YES / NO, **Prostate:**	**SPINE:**
UPPER EXTREMITIES: **Pulses present?** Brachial: **L** YES / NO; **R** YES / NO; Radial: **L** YES / NO; **R** YES / NO	**LOWER EXTREMITIES:** **Pulses present?** Femoral: **L** YES / NO; **R** YES / NO; Popliteal: **L** YES / NO; **R** YES / NO; Pedal: **L** YES / NO; **R** YES / NO
EXTERNAL: Abrasions / Contusions / Lacerations / Burns	

INJURY DIAGRAMS

HEAD AND NECK INJURIES:



SYSTEMIC INJURIES:



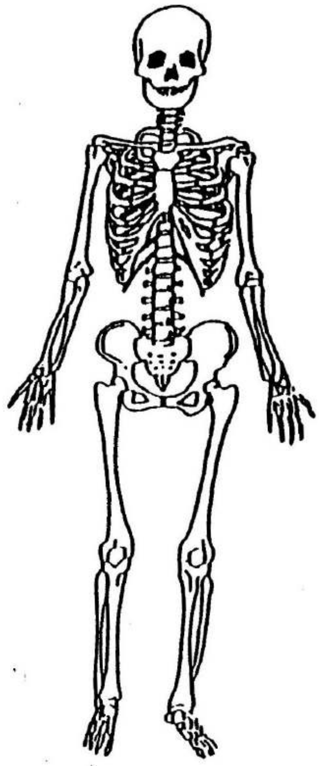
Pulses present? **Record Doppler pressures**

CENTRAL NERVOUS SYSTEM:

PUPILS:			**REFLEXES:**
Size	**L:**	**R:**	Biceps:
React to light	**L:**	**R:**	Triceps:
Consensual:	YES / NO		Patella:
Fundi:			Ankle:
POWER:	**L**	**R**	Babinski:
Arm flexion:			**OTHER INVESTIGATIONS:**
Arm extension:			Blood sugar:
Leg flexion:			Urine dipstick:
Leg extension:			Clearview preg test:
SENSATION:			NG Aspirate:
			CVP:
			Blood sent: FBC / U&E / COMPAT / PI/PTT / GLUC / TOX / HIV / CE / Other:

BLOOD GAS:	**ABG:**	**ABG:**	**VenousBG:**
Time:			
Temp:			
pH:			
PO2:			
PCO2:			
Saturation:			
SBE:			
HCO3:			
Hct:			
Hb:			
Na:			
K:			
Lactate:			

SKELETAL INJURIES:



EMERGENCY UNIT DISCHARGE:

Emergency Unit discharge date:		**Emergency Unit discharge time:**	
Emergency Unit disposition: Ward / Theatre – Ward / ICU / Theatre – ICU / DOT / DIC / Home / Transferred to: Hospital			
Admitting discipline: Trauma / Neuro / Ortho / MaxFac / Plastics / Paeds / Other:			**Admitting Ward:**
Emergency Unit Doctor Name:	**ATLS trained:** YES / NO Signature:	**Nurse team leader** Name:	Form checked by:

NURSING OBSERVATIONS

Time:																	
		240															
		230															
	X	220															
	'	210															
BLOOD	'	200															
PRESSURE	'	190															
	'	180															
	'	170															
	'	160															
	'	150															
	'	140															
	'	130															
	X	120															
		110															
		100															
		90															
PULSE	•	80															
		70															
		60															
		50															
Admission		40															
Temperature:		30															
		20															
A	Patent		Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N
	Tube cm at teeth																
	Cuff checked		Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N	Y/N
B	**SATS**																
	Patient's **Respiration** rate:																
	O2 Mask ________ %, l/min																
	Air entry **L / R**																
	Grunting / Stridor / Distress?																
	Ventilator rate																
	SIMV / CPAP																
	FIO2																
	Minute volume																
	Peak Airway Pressure																
	PEEP																
	Capnograph																
C	**Pulse** rate																
	Rhythm																
	CVP																
	Mucous Membrane colour																
	Chest pain ? / 10																
D	Pupil size **LEFT**																
	Pupil size **RIGHT**																
	Pupil Reaction **LEFT**																
	Pupil Reaction **RIGHT**																
	G Eye Opening																
	C Verbal Response																
	S Motor Response																
	TOTAL GCS																

INTAKE

IV FLUIDS:					BLOOD PRODUCTS:							
SITE	IV FLUID	VOL.	TIME UP	TIME DOWN	SITE	BLOOD / UNIT NUMBER	GROUP	VOL.	TIME UP	TIME DOWN	EXPIREY DATE	SIGNATURE

MEDICATION

SEDATION /ANALGESIA	DOSE	ROUTE	SITE	TIME	SIGN & QUAL	OTHER MEDICATION	DOSE	ROUTE	SITE	TIME	SIGN & QUAL

OUTPUT

TIME	URINE	BLOOD LOSS	UNDER WATER DRAIN	GASTRIC LOSS	OTHER	**TOTAL**

Appendix B: Study Data Collection Sheet

Date	Traumabank number	Hospital number	sex	age	Stab/GSW	Trachea central	Muffled heart sounds	Distended neck veins	hypotension	Beck's	Beck's without muffled heart sounds	CVP	Pulsus paradoxus	Cardiac exam	Chest exam	CXR +	ECG +	US +	TEE +	TTE +	CT +	CK	CKMB	%	TNT	Subxiphoid window	Pericardiocentesis	Stab precordium	Cardiac injury	CXR	US	CT	Emergency room Thoracotomy	Theatre	Procedure	Cardiac injury	D/C/morta
Jan07.	4337	GT00449909	F	26	S	Y		N	N		N			NAD	NAD	N	N	N							<0.03			Y	N	NAD			N	N			D/C
	4343	GT00450014	M	31	G	Y		N	N		N			NAD	reduced air	N												N	N	R haemo			N	Y	thoracotomy		D/C
	4348	GT00450349	M	23	S	Y		N	N		N			NAD	NAD	R pneumo					N							N	N	R pneumo		NAD	N	Y	laparotomy + neck explo		D/C
	4349	GT00450470	M	30	S				Y					NAD	NAD	N					N							N	N	NAD		NAD	N	N			D/C
	4352	GT00450534	M	32	S	Y		N	N		N			NAD	sucking ches	N		N										N	N	R pneumo	NAD		N	N			D/C
	4353	GT00450555	M	27	G	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	N			D/C
	4354	GT00450566	M	40	S	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	Y	laparotomy		D/C
	4357	GT00450729	M	37	S	Y			N					NAD	NAD	N												Y	N	NAD			N	N			D/C
	4363	GT00450981	M	43	G	Y		N	N		N			NAD	NAD	N		N			N							Y	N	L HPT	NAD	NAD	N	Y	repair brachial artery		D/C
	4366	GT00451180	M	27	S	Y		N	N		N			NAD	reduced air	N												Y	N	L pneumo			N	N			D/C
	4374	GT00451896	M	39	G	Y		N	N		N			NAD	reduced air	N												N	N	NAD			N	N			D/C
	4379	GT00452135	M	27	S	Y		N	N		N	7		NAD	NAD	N	N			N					<0.03			N	N	L haemo			N	N			D/C
	4383	GT00452179	M	33	S											N												N	N	R HPT			N	N			D/C
	4388	GT00452226	M	22	S	Y		N	N		N			NAD	reduced air	N												N	N	L pneumo			N	N			D/C
	4403	GT00453282	M	22	S	Y		N	N		N			NAD	reduced air	N					N							N	N	L rib 3 # + L HPT		L rib 3	N	N			D/C
	4405	GT00453520	M	46	S	Y			N					NAD	NAD	N												Y	N	NAD	NAD		N	N			D/C
	4406	GT00453535	M	28	S	Y		N	N		N			NAD	NAD	N												Y	N	R pneumo			N	N			D/C
	4411	GT***********	M	25	G	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4412	GT00453658	M	31	S	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	N			D/C
	4413	GT00453668	M	54	S	Y		N	N		N			NAD	reduced air	N												N	N	NAD			N	N			D/C
	4422	GT00454246	M	32	G				N					NAD	reduced air	N												Y	N	R ICD			N	N			D/C
	4427	GT00454601	M	21	G	Y		N	N		N			NAD	NAD	N		N										Y	N	NAD	NAD		N	N			D/C
	4428	GT00454790	M	20	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4430	GT00454797	M	27	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4432	GT00454988	M	24	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4439	GT00455060	M	19	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4443	GT00455121	M	21	S	Y		N	N		N			NAD	reduced air	N												N	N	R pneumo			N	N			D/C
	4444	GT00455144	M	38	G	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	N			D/C
	4445	GT00455147	M	23	S	Y		N	N		N			NAD	reduced air	N												N	N	L pneumo			N	N			D/C
	4446	GT00455148	M	32	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4449	GT00455161	M	24	S	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	N			D/C
	4450	GT00455162	M	29	S	Y		N	N		N			NAD	NAD	N												N	N	L HPT			N	N			D/C
	4451	GT00455173	M	32	S	Y			N					NAD	NAD	N		N								Neg		N	N	NAD	NAD		N	Y	pericardial window + biila		D/C
	4454	GT00455186	M	24	G	Y		N	N		N			NAD	reduced air	N		N			N							N	N	bilateral H	liver l	HPT b	N	Y	laparotomy + thoracotor		D/C
	4462	GT00455285	F	38	S	Y		N	N		N			NAD	NAD	N												N	N	Haemothorax + surgica			N	N			D/C
Feb07.	4467	GT00456357	M	30	S	Y		N	Y		N			NAD	reduced air	N												N	N	L haemo			N	Y	thoracotomy		D/C
	4571	GT09157413	M	36	S	Y		Y	N		N			NAD	NAD	N	N					##	58	#	<0.03			Y	N	NAD			N	Y	sternotomy		D/C
	4478	GT00456374	M	24	S	Y		N	N		N			NAD	reduced air	N		N										Y	N	L pneumo	NAD		N	N			D/C
	4481	GT00456605	M		S	Y		N	N		N			NAD	NAD	N												Y	N	L pneumo			N	N			D/C
	4482	GT09391104	M	22	S	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	N			D/C
	4483	GT00456635	M	33	S	Y		N	N		N			NAD	NAD	N												Y	N	L haemo			N	N			D/C
	4484	GT00456641	M	30	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4488	GT00456711	M	34	G	Y		N	N		N			NAD	reduced air	N												N	N	NAD			N	N			D/C
	4495	GT00455202	F	26	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4496	GT00456828	M	24	S	Y		N	N		N			NAD	reduced air	N												Y	N	R pneumo			N	N			D/C
	4498	GT00456843	M	32	S	Y		N	N		N			NAD	NAD	N					N							N	N	NAD		NAD	N	N			D/C
	4501	GT00456869	M	27	G	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4512	GT00457700	M	27	S	Y		N	N		N			NAD	NAD	N	N					##	95	#	<0.03			N	N	L pneumo			N	N			D/C
	4517	GT00458145	M	29	G	Y		N	N		N			NAD	reduced air	N					N							Y	N	R HPT			N	N			D/C
	4519	GT00458189	M	30	S	Y		N	N		N			NAD	NAD	N	N								<0.03			Y	N	NAD			N	N			D/C
	4522	GT00458214	M	23	S	Y		N	N		N			NAD	reduced air	N		N										N	N	NAD	NAD		N	N			D/C
	4524	GT00458225	M	37	S				N					NAD	reduced air	N												N	N	L HPT			N	N			D/C
	4531	GT00458365	M	22	S	Y		N	N		N			NAD	reduced air	N												Y	N	R pneumo			N	N			D/C
	4532	GT00458407	F	27	S	Y		N	N		N			NAD	reduced air	N									<0.03			N	N	R HPT			N	N			D/C
	4543	GT00459012	M	25	S	Y		N	N		N			NAD	NAD	N		N										Y	N	NAD	NAD		N	N			D/C
	4550	GT00459581	M	29	G	Y		N	N		N			NAD	NAD	N					N							N	N	NAD		Liver	N	N			D/C
	4552	GT00459860	M	25	S											N												N	N	R HPT			N	N			D/C
	4557	GT00410169	F	25	S				N					NAD	NAD	N												N	N	NAD			N	N			D/C
	4563	GT00460016	M	23	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4571	GT00468968	M	27	S	Y		N	N		N			NAD	reduced air	N		N										N	N	NAD	NAD		N	N			D/C
	4572	GT00460108	M	24	S	Y			N					NAD	NAD	N												N	N	NAD			N	N			D/C
	4573	GT00460120	M	42	G	Y		N	Y		N			NAD	reduced air	N												N	N	NAD			N	Y	laparotomy		D/C
	4576	GT00460382	M	31	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4577	GT00460396	M	35	S	Y		Y	Y		Y			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4578	GT00413810	M	27	G	Y		N	Y		N			NAD	reduced air	N												Y	N	R haemo			Y	N	bilat thoracotomy + clam		DiC
	4580	GT00460664	M	25	G	Y		N	N		N			NAD	reduced air	N												Y	N	NAD			N	Y	laparotomy		D/C
	4592	GT00461243	M	13	S	Y		N	N		N	5		NAD	reduced air	N	N	N										Y	N	L HPT	NAD		N	N			D/C
	4593	GT00461245	M	26	G	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	Y	laparotomy		D/C
	4596	GT00461487	M	20	S	Y		N	N		N			NAD	reduced air	N						##	37	#	<0.03			Y	N	NAD			N	N			D/C
	4599	GT00461496	M	18	S	Y		N	N		N			NAD	reduced air	N												N	N	L HPT			N	N			D/C
	4600	GT00461510	M	55	G	Y		N	N		N			NAD	NAD	N												Y	N	free air under diaphrag			N	Y	laparotomy		D/C
	4603	GT00461603	M	18	S	Y		N	N		N			NAD	sucking ches	N		N										N	N	L pneumo	NAD		N	N			D/C
	4604	GT00461620	M	31	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4607	GT00461652	M	26	S	Y		N	N		N			NAD	reduced air	N												N	N	L pneumo + free air un			N	Y	laparotomy		D/C
	4610	GT00461668	M	23	S	Y		N	N		N			NAD	NAD	N												Y	N	L pneumo			N	N			D/C
	4612	GT00461897	F	35	S	Y		N	N		N			NAD	NAD	N												N	N	L pneumoo			N	N			D/C
	4614	GT00462236	M	15	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
Mar07	4617	GP00008203	M	46	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4626	GT00463881	M	22	S	Y		N	N		N			NAD	reduced air	N												N	N	R pneumo			N	N			D/C
	4628	GT00463905	M	24	S	Y		N	N		N			NAD	reduced air	N		N										Y	N	L pneumo			N	N			D/C
	4632	GT25495020	M	23	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4636	GT***********	M	30	G	Y		N	N		N			NAD	reduced air	N												Y	N	L haemo			N	Y	Thoracotomy + cross clai		DiC
	4645	GT00464221	M	23	S	Y		N	N		N			NAD	sucking ches	N												Y	N	R HPT			N	N			D/C
	4648	GT00464539	M	35	G	Y		N	N		N			NAD	NAD	N					N	58			<0.03			N	N	R HPT		NAD	N	N			D/C
	4655	GT00465413	M	32	S	Y		Y	N		N	15		NAD	emphysema	N	N								<0.03			Y	N	L pneumo			N	N			D/C
	4657	GT00465426	M	28	S	Y		N	N		N			NAD	NAD	N		N										N	N	NAD	NAD		N	N			D/C
	4662	GT00169153	M	28	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C
	4665	GT00465702	M	31	G	Y		N	N		N	6		NAD	NAD	N	N	N			N	##	75	6	<0.03			Y	N	NAD	NAD	Liver l	N	N			D/C
	4666	GT00465703	M	20	S				N					NAD	reduced air	N		N			N							Y	N	R HPT	NAD	NAD	N	N			D/C
	4668	GT00465790	M	40	S	Y			N					NAD	reduced air	N												N	N	NAD			N	N			D/C
	4670	GT00360853	M	31	S	Y		N	N		N			NAD	NAD	N		N										Y	N	NAD	NAD		N	N			D/C
	4674	GT00465902	F	21	S	Y		N	N		N			NAD	reduced air	N												N	N	L pneumo			N	N			D/C
	4679	GT00466188	M	28	S	Y		N	N		N			NAD	reduced air	N		N										N	N	NAD	NAD		N	N			D/C
	4687	GT00466759	M	20	G	Y		N	N		N			NAD	NAD	N												N	N	NAD			N	N			D/C
	4695	GT00467309	M	23	S	Y		N	N		N			NAD	reduced air	N												Y	N	R HPT			N	N			D/C
	4700	GT00467337	M	27	S	Y		N	N		N			NAD	NAD	N												Y	N	NAD			N	N			D/C

Appendix C: Human Research Ethics Committee (HREC) Clearance Certificate

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Dr Daniel Surridge

CLEARANCE CERTIFICATE **Protocol M090122**

PROJECT Surgery
Precordial Stab Wounds in the Developing World: A Retrospective Analysis

INVESTIGATORS Dr Daniel Surridge.

DEPARTMENT Surgery

DATE CONSIDERED 09.01.30

DECISION OF THE COMMITTEE* Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE 09.02.02 **CHAIRPERSON** ....
(Professor P E Cleaton Jones)

*Guidelines for written 'informed consent' attached where applicable

cc: Supervisor : Dr G Candy

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10004, 10th Floor, Senate House, University.
I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES...

Appendix D: Poster for International Congress