A DISSERTATION ON

"TRANEXAMIC ACID USE IN SEVERELY INJURED PATIENTS WITH SIGNIFICANT HAEMORRHAGE - A PROSPECTIVE COHORT STUDY"

Submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In partial fulfillment for the award of the degree of

M.S (GENERAL SURGERY)

DEPARTMENT OF GENERAL SURGERY

GOVERNMENT STANLEY MEDICAL COLLEGE

CHENNAI

CERTIFICATE

This is to certify that the dissertation titled **"TRANEXAMIC ACID USE IN SEVERELY INJURED PATIENTS WITH SIGNIFICANT HAEMORRHAGE- A PROSPECTIVE COHORT STUDY"** submitted by **DR.S.PRAVEEN KUMAR** in partial fulfilment for the award of the degree of M.S(GENERAL SURGERY) by The Tamilnadu DR.M.G.R Medical University, Chennai is an original work done by him in the Department of General Surgery, Stanley Medical College, Chennai during the academic year 2017-2020.

Prof.Dr.R.Shanthi Malar M.D, D.A
Dean
Stanley Medical College,
Chennai- 600 001

Prof.Dr.T.Sivakumar M.S,
Head of the Department,
Department of General Surgery,
Stanley Medical College,
Chennai- 600 001

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation titled **"TRANEXAMIC ACID USE IN SEVERELY INJURED PATIENTS WITH SIGNIFICANT HAEMORRHAGE- A PROSPECTIVE COHORT STUDY"** submitted by **DR.S.PRAVEEN KUMAR** in partial fulfilment for the award of the degree of M.S(GENERAL SURGERY) by The Tamilnadu DR.M.G.R Medical University, Chennai is a Bonafide work done by him in the Department of General Surgery, Stanley Medical College, Chennai under my guidance and supervision during the academic year 2017-2020.

Prof.Dr.G.Venkatesh M.S,
Department of General Surgery,
Stanley Medical College,
Chennai- 600 001

Date:

Place: Chennai

DECLARATION

I, DR.S. PRAVEEN KUMAR solemnly declare that the dissertation **"TRANEXAMIC ACID USE IN SEVERELY INJURED PATIENTS WITH SIGNIFICANT HAEMORRHAGE- A PROSPECTIVE COHORT STUDY"** is a bonafide work done by me in the Department of General Surgery, Stanley Medical College, Chennai under the guidance of Professor Dr.G.Venkatesh M.S, Department of General Surgery, Stanley Medical College, Chennai and submitted to The Tamilnadu DR.M.G.R. Medical University, Guindy, Chennai-32, in partial fulfilment for the requirements for the award of the degree of M.S (General Surgery) examinations to be held in MAY 2020. I have not submitted this dissertation previously to any university for award of degree or diploma.

Place: Chennai. **Dr.S.Praveen Kumar**

Date:

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Date:

Place: Chennai.

DR.S. PRAVEEN KUMAR M.B.B.S

Postgraduate in General Surgery

Stanley Medical College,

Chennai.

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01

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DESIGNATION : PG IN MS GENERAL SURGERY
DEPARTMENT : DEPARTMENT OF GENERAL SURGERY, GOVT. STANLEY MEDICAL COLLEGE.

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INTRODUCTION

Injuries following trauma are a major cause of morbidity and mortality worldwide. It is predicted that injuries may contribute to 20% of all causes by the year 2020[i]

Trauma remains a major cause of death in individuals younger than the age of 45 years throughout the world. These individuals suffer from life threatening complications due to severe haemorrhage which further aggravates the immune system response leading to more tissue damage[ii]

Haemorrhage due to traumatic injuries contributes one third of trauma deaths and also cause death by multiorgan failure[iii]. The haemostatic system maintains blood circulation following vascular injury in trauma. In response to surgery or trauma fibrinolysis occurs and may become pathologically hyper fibrinolytic. This poses a challenge to the circulatory system following severe blood loss[iv]

Tranexamic acid is a synthetic derivative of the amino acid lysine that inhibits fibrinolysis by blocking the lysine binding sites on plasminogen[v]

Tranexamic Acid (TXA) is used as an anti-fibrinolytic agent to reduce surgical bleeding if administered prior to or during surgery, and to improve survival in trauma if given early after trauma[vi]

A large, randomized control trial CRASH 2 across 274 hospitals in 40 countries studied the use of tranexamic acid in bleeding trauma patients & proposed that early use of tranexamic acid in patients, with or at risk of significant bleeding, resulted in significant reduction in mortality[vii]

Tranexamic acid use has also been found to be beneficial in the military setting in combat trauma[viii].Tranexamic acid has especially found to be beneficial in severely injured patients with shock in a mature civilian trauma setup with improved morbidity and mortality [ix].

There was reduced mortality for patients receiving tranexamic acid in spite of their increased acuity and decreased likelihood of survival. Trauma patients receiving tranexamic acid demonstrate decreased mortality in spite of increased acuity and increased transfusion requirements.[x]

The following study analyses the effect of tranexamic acid on mortality and other clinical outcomes in a severely injured trauma patient with significant haemorrhage in a civilian tertiary trauma care setup.

OBJECTIVES

PRIMARY OBJECTIVE

To determine the relationship between tranexamic acid use and patient outcomes in a severely injured civilian cohort

SECONDARY OBJECTIVE

To determine differential effects between patients who presented with or without shock

REVIEW OF LITERATURE

INTRODUCTION

Trauma is called the ‘neglected disease’ of the modern society. It is also called the ‘unsolved epidemic of the future’. It is the principal cause of death between 15 and 44 years of age, the most productive group of population.

As per the Royal College of Surgeons data, 20% of the deaths are preventable. If the neurosurgical cases are excluded, up to 40% of the deaths are preventable. It is not the lack of hi-tech care, but ordinary surgical care in the form of identifying and managing the internal haemorrhage and treatment of hypoxia. It was not the fractures that killed, but internal haemorrhage according to the American series. It is important to note that some of the patients died while being transported to the CT room. An estimated 5 million people die from injuries worldwide forming the third leading cause of death. The economic impact of trauma is huge and the social cost is still higher.

ADVANCED TRAUMA LIFE SUPPORT

The Advanced Trauma Life Support (ATLS) was initially deviced by James Styner, an orthopedic surgeon in 1970. He was involved in an air crash and found that there is no structured way of trauma management, and hence devised ATLS. This was later on adopted by the American college of Surgeons committee on trauma.

This is a four stage continuous approach:

1. Primary survey.

2. Resuscitation.

3. Secondary survey.

4. Definitive care (+ 5. Tertiary survey).

PRIMARY SURVEY

It is a 60 second head-to-toe examination looking for ABCDE.

A—airway with cervical spine protection.

B—breathing and ventilation.

C—circulation and hemorrhage control.

D—disability and neurological status.

E—exposure/entry with prevention of hypothermia.

Airway

The simplest method of checking the airway is to ask the patient 'what is your name, and what hurts?' A correct answer shows that patient has got a patent airway. In addition, it also shows that the patient has got sufficient cerebral function to process the stimulus and sufficient ventilation to phonate the answer. Complete obstruction will produce aphonia. Partial obstruction will produce snoring/stridor.

There are basic airway techniques and advanced airway techniques. But, however, it should be instituted while protecting the cervical spine. Clear the mouth and airway with a large bore sucker. If foreign bodies are there, finger sweep will be enough. If the GCS is less than 8, consider definitive airway.

The Basic Airway Techniques include:

1. Modified jaw thrust maneuver and

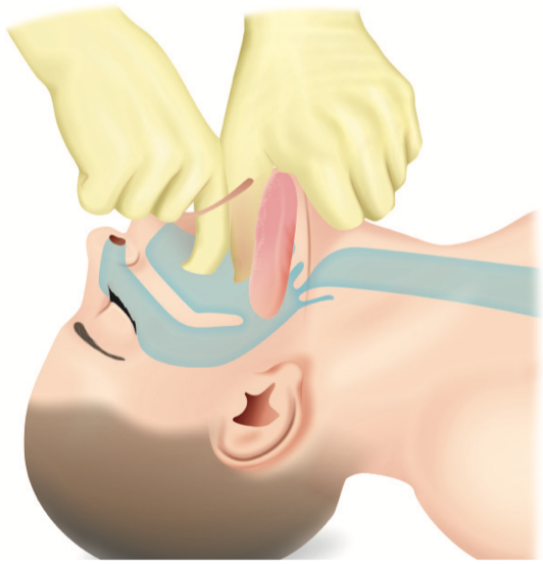
2. Oral/nasopharyngeal airway

The Advanced Airway Techniques Consists of:

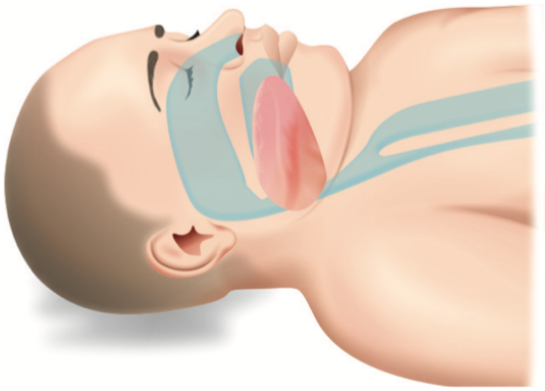
1. Oral/nasal intubation

2. Surgical/needle (13G) cricothyroidotomy.

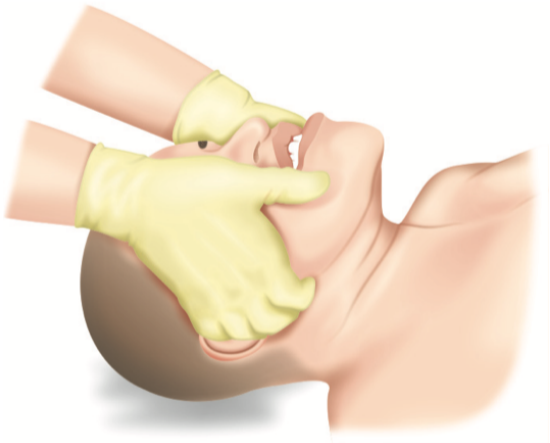
Figure 1. Basic Airway Techniques



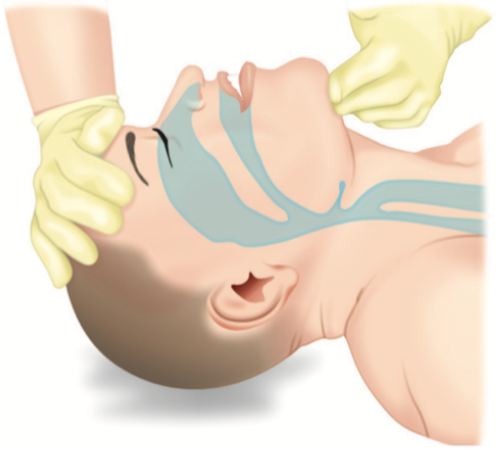
Finger sweep method of clearing the oral cavity



Compromised airway



Jaw thrust maneuver



Chin lift

BREATHING AND VENTILATION

It is important to identify hypoxia, tension pneumothorax, flail chest, hemothorax and other life-threatening injuries. They are not radiological diagnosis but clinical diagnosis by observation or the absence of chest movements and percussion and auscultation findings.

Open Chest Wounds

Open chest wounds are called sucking wounds and they should be managed by occluding it with a three-sided dressing followed by tube thoracostomy through a separate incision.

First aid management of flail chest—It is by turning the patient to the side of paradoxical movement, so that segment will be immobilized.

Tension pneumothorax—It is identified by tracheal shift and mediastinal shift in a patient with acute dyspnea and absent air entry on one side with hyper-resonance on percussion. It is important to treat tension pneumothorax without waiting for X-ray chest by either tube thoracostomy (in safe triangleread tube thoracostomy) or needle thoracostomy (2nd ICS).

Administration of oxygen—100% oxygen must be administered to all trauma patients at a high flow rate.

Indications for Mechanical Ventilation

1. Tachypnea above 40.

2. PaO 2 below 60 mm Hg or less.

3. PaCO 2 above 45 mm Hg.

4. Progressive fall in PaO2 .

5. Extensive pulmonary contusion or diffused infiltrative changes on X-ray.

6. Severe flail chest (>8 U/L or >4 B/L rib fractures).

CIRCULATION

Rough estimation of the BP is possible by palpating the pulse. If the radial pulse is palpable, the BP will be 80 mm Hg. If the radial pulse is not felt, feel the femoral. If femoral alone is palpable, the BP will be 70 mm Hg. If both radial and femoral are not felt, feel the carotids. The carotid pulse is felt with a BP of 60 mm Hg.

The pulse will be rapid and thready in case of hemorrhagic shock. The skin color will be pale, ashen and gray looking in hypovolemia. Assessment of the conscious level is also important.

Look for evidence of internal and external bleeding. ‘Blood on the floor and four more places’—externally, chest, abdomen, retroperitoneum and pelvis, muscle compartment.

Tennis score classification of haemorrhage

Hemor-rhage	*% Loss*	*Volume loss (mL)*	*Pulse rate*	*BP*	*Pulse pressure*	*Respiratory rate*
Class I	15	750	<100	NL	NL/>	14–20
Class II	15–30	750–1,500	>100	NL	Decreased	20–30
Class III	30–40	1,500–2,000	>120	Decreased	Decreased	30–40
Class IV	>40	>2,000	>140	Decreased	Decreased	>40

Table 1. Tennis score classification of haemorrhage

Medical Anti-shock Trousser (MAST) (Pneumatic anti-shock garment)

It was extensively used in Vietnam war. It consists of inflatable sections for each leg and abdomen and it is radiolucent. There is access for perrectal examination and urinary catheter. When it is inflated, it will reduce hemorrhage, reduce the total functioning volume of the vascular compartment and give auto-transfusion effect of 0.5 to 1 liter of blood. In addition, it will splint the lower limb and pelvic fractures.

Indications

1. Splinting and control of pelvic fractures.

2. Abdominal trauma with hypovolemia.

Contraindications

1. Pulmonary edema.

2. Diaphragmatic rupture.

3. Thoracic and upper limb hemorrhage.

Figure 2. Medical Anti Shock Trouser

DISABILITY

Disability is usually measured by two scales

GCS—Glasgow Coma Scale

or **AVPU score** AVPU—A-alert, V-response to vocal stimuli, P-response to painful stimuli, U-unresponsive.

GLASGOW COMA SCALE

Table 2. Glasgow coma scale

Eye opening	Spontaneously To verbal command To painful stimulus Do not open	4 3 2 1
Verbal response	Normal oriented conversation Confused Inappropriate/words only Sounds only No sounds Intubated patient	5 4 3 2 1 T
Motor response	Obeys commands Localizes to pain Withdraws/flexion Abnormal flexion Extension No motor response	6 5 4 3 2 1

RESUSCITATION PHASE

The role of conventional aggressive resuscitation is slowly going out of vogue in favour of a controlled infusion of fluid (graded resuscitation), especially in penetrating injury.

Conventional Aggressive Resuscitation

1. Secure large bore IV, access for shock therapy.
2. Continuous ECG monitoring.
3. Blood samples for CBC, electrolytes, glucose, coagulation studies, ABG and cross-matching.
4. Nasogastric tube is introduced in all multisystem trauma cases.
5. Foleys catheter (if not contraindicated).

Peripheral IV lines central line

It is preferable to put two 14G peripheral IV lines rather than a 16G, 8 inch length central cannula. As per Poiseuille's law, the flow is proportional to the 4th power of radius of the cannula and inversely related to its length. A 14G cannula with 2¼ inch length will give a flow of 200 cc/minute compared to 150 cc for a 16G, 8 inch length central cannula. Therefore, peripheral lines are recommended for resuscitation.

FLUID RESUSCITATION IN TRAUMA

Controlled Infusion of fluid/Graded ResuscitationThe New Trend

Fluid administration before surgical control of hemorrhage may actually worsen bleeding and increase mortality. Therefore, the current aggressive resuscitation is potentially harmful and at best experimental. Permissive hypotension

is recommended in penetrating injury.

Problems of IV Fluids in Aggressive Resuscitation

1. Inhibit platelet aggregation.

2. Dilute clotting factors.

3. Modulate the physical properties of thrombus.

4. Mechanical disruption of clot by increased BP.

Damage Control Resuscitation

The controlled infusion of fluid with permissive hypotension until surgical hemostasis is called damage control resuscitation. Other components are:

1. Minimize crystalloid use.

2. Use 5% hypertonic saline.

3. Use blood products early.

4. Use of r-factor VIIa and factor IX.

5. Avoid hypothermia

BLOOD TRANSFUSION AND HEMOGLOBIN-BASED OXYGEN CARRIERS

Packed Red Cells

It has immunosuppressive potential. A second indication must be present in addition to a decreased hemoglobin concentration. Young trauma patients can tolerate a hemoglobin level of 7 g/dL.

New generation hemoglobin-based oxygen carriers provide volume expansion and oxygen carrying capacity.

Role of Recombinant Activated Factor VII (rFVIIa)

Massively bleeding multitransfused coagulopathic trauma patients benefit from this.

Resuscitation with Whole Blood

'Walking blood bank' concept is there in war situations. However, aggressive use of FFP is recommended nowadays. FFP, PRC and platelets are used in ratio of 1:1:1.

Monitoring Progress and Treatment

This is done by monitoring the following:

1. Urinary output.
2. Pulse rate.
3. Pulse pressure.
4. Temperature.
5. Mental state.
6. Arterial pressure.
7. Central venous pressure/Swan–Ganz catheter.
8. Oxygen saturation.

Avoid Hypothermia

1. All fluids for transfusion must be stored at 39°C in a fluid warmer
2. Packed red cells reconstituted by warm saline.
3. Irrigating fluid should be warm.
4. Use warm blankets.

Hypothermia will lead on to cardiac irritability, coagulopathy and enzyme impairment.

Bloody Vicious Cycle

It is formed by **hypothermia, metabolic acidosis and coagulopathy**.

ADVERSE EFFECTS OF TRANSFUSION

Transmission of disease:

It can be a serious problem. HIV, Hepatitis B virus, Hepatitis C virus and Cytomegalovirus can all be transmitted by transfusion. So each unit of blood or its components given, they must be carefully scrutinized before they are administered.

Febrile reactions:

It occur in approximately 1% of all transfusions

Allergic non hemolytic reactions:

It will manifest as an increase in temperature, pruritus and urticaria. This may be difficult to diagnose in the anesthetized patient. Treatment consists of administration of antihistamines and discontinuation of the transfusion

MICROEMBOLISATION:

Microembolisation can occur from the transfusion of blood or its components. Stored blood forms microaggregates that are too small to be removed by the standard 170-micron blood filters

TRANSFUSION ASSOCIATED CIRCULATORY OVERLOAD

Transfusion associated circulatory overload is simply a volume overload state. It occurs because of the rate of volume infusion of blood products is in excess of what the patient's cardiovascular status can handle.

Large amounts of plasma transfusion that may occur during massive transfusion that may occur during massive transfusion or coagulopathy correction increase the likelihood of Transfusion associated circulatory overload.

TRANSFUSION RELATED ACUTE LUNG INJURY

This occurs within 6 hours of a transfusion. Hypoxia and bilateral non-cardiogenic pulmonary oedema will occur. It is due to neutrophil and/or endothelial activation via multiple mechanisms in the lung, resulting in pulmonary vascular injury and pulmonary oedema.

MECHANISM

2 hit model: First an inflammatory event may be required to activate and upregulate inflammatory cells and vascular endothelium, followed by a second, transfusion event that actually triggers an acute inflammatory response and injury. The primary event may be caused by prolonged storage of blood, recent viral illness, other events such as cardiopulmonary bypass. Transfusion of blood products from donors containing

antibodies against white blood cell antigens can result in direct binding and activation of intravascular polymorphonuclear leukocytes. Activated neutrophils bind to the pulmonary vascular endothelium, they release multiple proinflammatory substances including proteolytic enzymes, O2 free radicals, thromboxane and other inflammatory mediators both locally at the site of vascular injury and systemically. This complex series of events results in damage to endothelial cells, vascular leakage and pulmonary oedema.

TRANSFUSION RELATED ACUTE RESPONSES AND IMMUNOMODULATION

Inflammatory

Transfusion of multiple blood products have the potential for proinflammatory responses including acute hypersensitivity responses and anaphylaxis that may not affect the lung.

Graft Vs Host Disease

Transfusion associated graft vs host disease is a very rare complication of blood transfusion. GvHD can complicate allogenic bone marrow transplants, but in those who are immunocompromised, it can occur after simple blood transfusion. Donor derived immune cells,particularly T Lymphocytes, mount an immune response against host tissue. Clinical features include a maculopapular rash , abdominal pain, diarrohea and abnormal liver function tests.

MASSIVE TRANSFUSION

It is defined as the replacement of the patient's total blood volume in a 24 hour period. This is usually between 8 and 10 units of packed red blood cells. Many trauma cases far exceed this amount and may require other blood components in addition to the red blood cells

COMPLICATIONS

HYPOTHERMIA :

It is an obvious consequence of infusing cold , banked blood

CITRATE TOXICITY:

It is rare. However in the paediatric population citrate toxicity should be considered if in any child who does not respond to rapid volume administration of blood products. Citrate is added to stored blood to bind calcium and therefore prevent clotting. Citrate binds calcium and decreases the patient's ionized calcium level. Hypocalcemia may present as a prolongation of the QT interval which result in decreased cardiac performance.

HYPERKALEMIA:

It can be a rare occurrence in the massively transfused patient. A unit of packed red blood cells contains insignificant amounts of potassium because most plasma is removed. However, in the shock state with hypo perfusion and acidemia, hyperkalemia may become evident.

HYPOKALAEMIA :

It is also a possibility after a massive transfusion. Citrate is metabolised to bicarbonate, resulting in a metabolic alkalosis that can cause hypokalaemia

ACID BASE IMBALANCE:

It is a problem after massive transfusion.

IMPAIRED HAEMOGLOBIN FUNCTION:

It is a theoretic possibility after massive transfusion. The 2,3diphosphoglycerate level is decreased in banked blood. This will shift the oxygen- haemoglobin dissociation curve to the left, and oxygen will be held more tightly by the haemoglobin molecule.

Secondary Survey

A detailed head to toe examination is done by 'look, listen and feel technique'. Get three high yield X-rays [the three most important X-rays in multi-system trauma:

1. Cervical lateral (swimmer's view),
2. Upright chest,
3. Pelvis.

Rule out intra-abdominal bleeding in all cases of multi system trauma by Focused Assessment Sonography for Trauma (FAST), Ultrasonography (USG) and CT scan (never send unstable patient for CT) and serial haematocrit and repeated physical examination. In the history, 'AMPLE' is important. (A-allergy, M-medication, P-past medical history and pregnancy, L-last meal, E-events of the incident). Assume

cervical spine injury until proven otherwise. Four people are required for transfer of a trauma victim. One for spinal in-line traction (anaesthesiologist), one for the torso, one for pelvis and one for lower limbs. The turning of the patient, if required, is by the spinal log roll.

Dangerous Mechanisms of Injury

1. Fall from height of 20 feet or more.
2. Crash greater than 20 miles per hour.
3. 20 inch impingement on the passenger compartment.
4. Ejection of the passenger.
5. Roll over of the vehicle.
6. Death of another person.

Definitive Care

Coordinate consultations and all planned operations in definitive care.

Tertiary Survey (When the Dust is Settled)

Another detailed examination is conducted for identification of missed injuries. Missed injuries are called ‘the nemesis of the trauma surgeon’. Fifteen-percent incidence of clinically significant injuries are diagnosed after initial resuscitation. The tertiary survey is by a physical examination and review of results. Early detection of all clinically significant injuries is important to save the life of the patient.

MECHANISM OF BLOOD CLOTTING:

Blood Clotting is one of three mechanisms that reduce the loss of blood from broken blood vessels.

The three Mechanisms are:

i**. Vascular Spasm:**

The smooth muscle in blood vessel walls contracts immediately the blood vessel is broken. This response reduces blood loss for some time, while the other haemostatic mechanisms become active.

ii. Platelet Plug Formation:

When blood platelets encounter a damaged blood vessel they form a "platelet plug" to help to close the gap in the broken blood vessel. (The key stages of this process are called platelet adhesion, platelet release reaction, and platelet aggregation)

iii. Coagulation:

Following damage to a blood vessel, vascular spasm occurs to reduce blood loss while other mechanisms also take effect. Blood platelets congregate at the site of damage and amass to form a platelet plug. This is the beginning of the process of the blood "breaking down" from its usual liquid form in such a way that its constituents play their own parts in processes to minimize blood loss.

Blood normally remains in its liquid state while it is within the blood vessels but when it leaves them the blood may thicken and form a gel (coagulation). Blood clotting (technically “blood coagulation”) is the process by which (liquid) blood is transformed into a solid state.

This blood clotting is a complex process involving many clotting factors (incl. calcium ions, enzymes, platelets, damaged tissues) activating each other.

STAGES OF BLOOD CLOTTING:

1. Formation of Prothrombinase:

Prothrombinase can be formed in two ways, depending of which of two “systems” or “pathways” apply.

These are:

i. Intrinsic System:

This is initiated by liquid blood making contact with a foreign surface, i. e. something that is not part of the body

ii. Extrinsic System:

This is initiated by liquid blood making contact with damage tissue.

Both the intrinsic and the extrinsic systems involve interactions between coagulation factors. These coagulation factors have individual names but are often referred to by a standardised set of Roman Numerals, e.g. Factor VIII (anti-haemophilic factor), Factor IX (Christmas factor).

2. Prothrombin Converted Into the Enzyme Thrombin:

Prothrombinase (formed in stage 1.) converts prothrombin, which is a plasma protein that is formed in the liver, into the enzyme thrombin.

3. Fibrinogen (Soluble) Converted to Fibrin (Insoluble):

In turn, thrombin converts fibrinogen (which is also a plasma protein synthesized in the liver) into fibrin.

Fibrin is insoluble and forms the threads that bind the clot.

There are two pathways that lead to the conversion of prothrombin to thrombin:

(1) The intrinsic pathway and

(2) The extrinsic pathway.

(1) Intrinsic Pathway:

The intrinsic pathway, which is triggered by elements that lie within the blood inself (intrinsic to the blood), occurs in the flowing way. Damage to the vessel wall stimulates the activation of a cascade of clotting factors (for the sake of simplicity we will not consider the individual factors). This cascade results in the activation of factor X.

Activated factor X is an enzyme that converts prothrombin to thrombin. Thrombin converts fibrinogen to fibrin monomers, which then polymerize

in fibrin fibers. Fibirin fibers form a losse meshwork that is stabilized by crosslinks created by factor XIII. The stabilized meshwork of fibrin fibers ins now a clot that traps red blood cells and platelets and thus stops the flow of blood.

(2) Extrinsic Pathway:

The extrinsic pathway is triggered by tissue damage outside of the blood vessel. This pathway acts to clot blood that has escaped from the vessel into the tissues. Damage to tissue stimulates the activation of tissue thromboplastin, an enzyme that catalyzed the activation of factor X. At this point the intrinsic and extrinsic pathways converge and the subsequent steps are the same as those described above.

CLOTTING FACTORS

FACTOR	**NAME**
I	Fibrinogen
II	Prothrombin
III	Tissue factor or thromboplastin
IV	Calcium
V	Proaccelerin (Labile factor)
VII	Proconvertin (Stable factor)
VIII	Antihaemophilic factor A, Antihaemophilic globulin
IX	Antihaemophilic factor B, Plasma thromboplastin component, Christmas factor
X	Stuart-Prower factor

XI Plasma thromboplastin antecedent, Haemophilia C, Rosenthal syndrome

XII Hageman factor

XIII Fibrin stabilising factor, Laki-Lorand factor

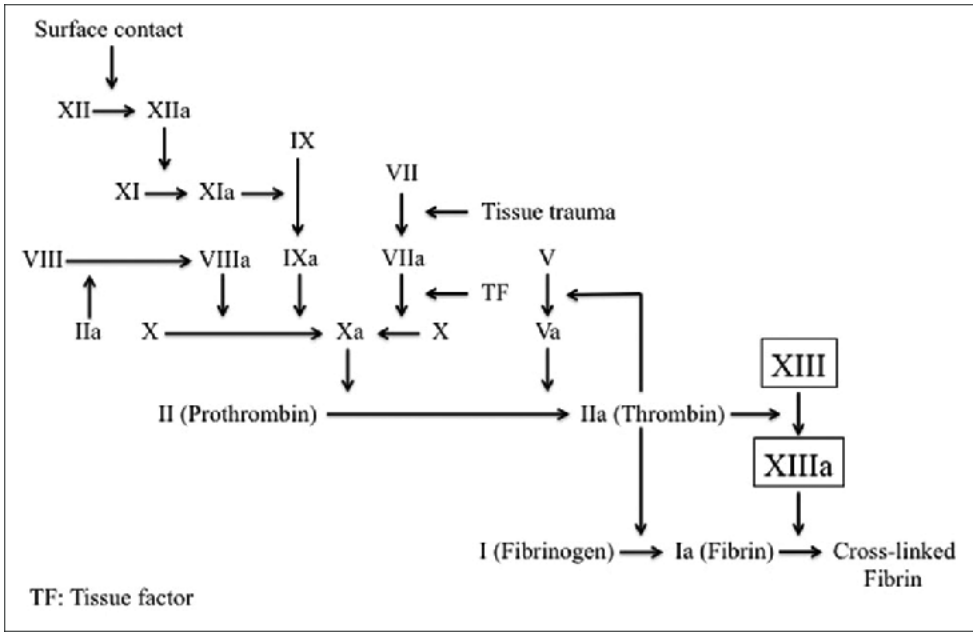


Figure 3. COAGULATION CASCADE

BLOOD CLOTTING FACTORS

FIBRINOGEN

Fibrinogen is a plasma globulin formed in the liver. It is a dimer having two sets (A&B) of three linked polypeptide chains. Clot is formed by the conversion of fibrinogen into fibrin by the action of thrombin. Only fluids containing fibrinogen can clot. Hence plasma can clot but not serum.

PROTHROMBIN

Prothrombin is a plasma globulin formed in the liver and requires vitamin K. It is a monomer and is a proenzyme and a precursor of thrombin. Its plasma concentration is about 15 mg /100 ml and its half-life is about 3-4 days. It is absent in the serum.

THROMBIN

Thrombin is not normally present in the plasma. It is formed from prothrombin by the action of prothrombin activators in the presence of Ca ++ ions .It is a protein with a molecular weight of 7 about 34000. It enzymatically converts fibrinogen to fibrin. It can also activate factor V & VII, and promotes platelet aggregation and activation.

TISSUE THROMBOPLASTIN

Tissue thromboplastin is released by injured tissues and is composed of tissue factor (a proteolytic enzyme) and tissue phospholipid. It activates factor VII.
Thromboplastin is liberated from all tissues, but the most potent thromboplastin are found in extracts of brain , lung and placenta.

CALCIUM

Ionic calcium is essential for clotting of blood .Decalcified blood does not clot .
Calcium ions are necessary for the activation of factors IX and X. Formation of prothrombin activators , and for activation of factor XIII.

FACTOR V (Labile factor, Accelerator globulin)

It is a heat labile high molecular weight protein formed in the liver. It is activated by factor X and thrombin , and is a co factor in the formation of prothrombin activator . It is used up during clotting and is absent in the serum. Its plasma half-life is about 36 hours.

FACTOR VII (Stable factor , or Serum Prothrombin Conversion Accelerator SPCA)

It is a protein formed in the liver and requires Vitamin K for synthesis. It is activated by tissue thromboplastin , and is a proenzyme for the activation of factor X. Its plasma half-life is 4 – 6 hours.

FACTOR VIII (Antihaemophilic globulin AHG, or AH factor A)

It is a high molecular weight globulin formed in the liver. It is transported in the blood combined with another protein von willebrand Factor (vWF) which is formed in the vascular endothelium (and also in the megakaryocytes). Factor VIII is activated on separation from vWF by thrombin and is a cofactor in the intrinsic pathway. It is used up during clotting and is absent in the serum. Its plasma life is about 12hours.Its deficiency causes Hemophilia A (vWF can also favor platelet adhesion)

FACTOR IX (Christmas factor, AH factor Plasma thromboplastin component PTC)

It is a protein formed in the liver and requires Vitamin K .It is activated by factor XI a and is a pro enzyme in the intrinsic pathway. Its Plasma half-life is about 24 hours. It is not used up in clotting and is present in serum. It is called Christmas factor , because this factor was first found to be deficient in a patient named Christmas. Deficiency of this factor causes Christmas disease or Hemophilia B.

FACTOR X (Stuart prower factor)

It is a two chain protein formed in the liver and requires Vitamin.K.

It is a pro enzyme for the formation of prothrombin activators in both intrinsic and extrinsic coagulation pathways. It is activated by active factors VIII and IX in the intrinsic pathway and by factor VII a in the extrinsic pathway . Its plasma half-life is about 24 to 40 hours. It is present in the serum.

FACTOR XI (Plasma thromboplastin antecedent PTA or AH factorC)

It is a pro enzyme in the intrinsic pathway, which activates factor IX and is activated by factor XII and by HMW kininogen. This protein is formed in the liver. Its plasma half-life is 2 to 3 days, and is present in the serum.

FACTOR XII (Hageman factor, Glass factor)

It is a protein formed in the liver. Its plasma half-life is about 2 -3 days and it is present in the serum. It is activated by high molecular weight kininogen and prekallikrein , and by contact with sub endothelial collagen in the injured vessel. (Contact with negatively charged glass surface also activates factor 12).Activated XII converts pre kallikrein to kallikrein, which further activates factor XII . Activated factor XII is an intact pro enzyme which activates factor XI along with HMWK.Activation of factor XII is the first reaction that initiates the process of coagulation, as observed in test tube. However factor XII deficiency does not result in a clinical bleeding disorder, whereas factor XI deficiency does. Hence Factor XI activation must be considered to be an important initial reaction and there is apparently some mechanism in addition to factor XII, involved in activation of factor XI.

FACTOR XIII (Fibrin stabilizing factor Laki- Lorand factor)

It is a protein with 2 alpha and 2 beta chains with a plasma halflife 5 -7days. It is formed in the liver and probably megakaryocytes. It is activated by thrombin in the presence of Ca 2++ by forming cross linkage between the fibrin monomers and polymers.

PREKALLIKREIN (Pre-K, Fletcher factor)

It is a globulin and is a part of the kinin system. It activates factor XII which in turn activates prekallikrein to kallikrein .This pro enzyme is also involved in the activation of factor XI

HIGH MOLECULAR WEIGHT KININOGEN: (Fitzgerald factor)

It is an alpha globulin and is a cofactor in the activation of factor XI and XII.

PLATELET PHOSPHOLIPID

Injury to a blood vessel causes platelet aggregation as well as release from damaged platelets , platelet phospholipids which is a cofactor in the conversion of prothrombin to thrombin.

TRAUMA SCORING SYSTEMS

Purpose of scoring systems

- Appropriate triage and classification of trauma patients
- Predict outcomes for patient and family counseling
- Quality assurance
- Research
- Extremely useful for the study of outcomes
- Reimbursement purposes

Classifications

Physiologic

- Revised Trauma Score (RTS)
- Acute Physiology and Chronic Health Evaluation (APACHE)
- Sequential Organ Failure Assessment Score (SOFA)
- Systemic Inflammatory Response Syndrome Score (SIRS)
- Emergency Trauma Score

Anatomic

- Abbreviated injury score (ais)
- Injury severity score (iss)
- New injury severity score (niss)
- Anatomic profile (ap)
- Penetrating abdominal trauma index (pati)
- Icd-based injury severity score (iciss)
- Trauma mortality prediction model (tmpm-icd9)
- Combined
- Trauma score – injury severity score (triss)
- A Severity Characterization of Trauma (ASCOT)
- International Classification of Diseases Injury Severity Score (ICISS)

REVISED TRAUMA SCORE (RTS)

Introduction

most widely used prehospital field triage tool

Variables

Glasgow Coma Scale (GCS) score

4: 13-15

3: 9-12

2: 6-8

1: 4-5

0: 3

Systolic blood pressure score

4: >90

3: 76-89

2: 50-75

1: 1-49

0: 0

Respiratory rate score

4: 10-29

3: >30

2: 6-9

1: 1-5

0: 0

Calculation

Glasgow coma scale score + systolic blood pressure score + respiratory rate score

Interpretation

Lower score indicates higher severity

RTS <4 proposed for transfer to trauma center

Pros

useful during triage to determine which patients need to be transported to a trauma center

Cons

can underestimate injury severity in patients injured in one system

INJURY SEVERITY SCALE (ISS)

- first scoring system to be based on anatomic criteria
- defines injury severity for comparative purposes

Variables

Based on scores of 9 anatomic regions

1. Head
2. Face
3. Neck
4. Thorax

5. Abdominal and pelvic contents
6. Spine
7. Upper extremity
8. Lower extremity
9. External

Calculation

Abbreviated Injury Scale (AIS) grades

0 – no injury

1 – minor

2 – moderate

3 – severe (not life-threatening)

4 – severe (life-threatening, survival probable)

5 – severe (critical, survival uncertain)

6 – maximal, possibly fatal

ISS = sum of squares for the highest AIS grades in the three most severely injured ISS body regions

ISS = A2 + B2 + C2

where A, B, C are the AIS scores of the three most severely injured ISS body regions

scores range from 1 to 75

single score of 6 on any AIS region results in automatic score of 75

Interpretation

ISS > 15 associated with mortality of 10%

Pros

integrates anatomic areas of injury in formulating a prediction of outcomes

Cons

difficult to calculate during initial evaluation and resuscitation in emergency room

difficult to predict outcomes for patients with severe single body area injury

New Injury Severity Score (NISS) overcomes this deficit.

New Injury Severity Score (NISS)

- Takes three highest scores regardless of anatomic area
- More predictive of complications and mortality than ISS

Modifications

Modified Injury Severity Score (MISS)

- Similar to ISS but for pediatric trauma
- categorizes body into 5 areas, instead of 9
- sum of the squares for the highest injury score grades in the three most severely injured body regions

Mangled Extremity Severity Score (MESS)

Introduction

Used to predict necessity of amputation after lower extremity trauma

Variables

- Skeletal and soft tissue injury (graded 1-4)
- Limb ischemia (graded 1-3)
- Shock (graded 0-2)
- Age (graded 0-2)

Calculation

Score determined by adding scores of components in four categories

Interpretation

Score of >7 is predictive of amputation

Pros

High specificity for predicting amputation

Cons

Low sensitivity for predicting amputation

Systemic Inflammatory Response Syndrome (SIRS)

Introduction

a generalized response to trauma characterized by

- an increase in cytokines

- an increase in complement
- an increase in hormones
- a marker for an individual's generalized response to trauma that likely has a genetic predisposition

associated with conditions such as

- disseminated intravascular coagulopathy (DIC)
- acute respiratory distress syndrome (ARDS)
- renal failure
- multisystem organ failure
- shock

Variables

- heart rate > 90 beats/min
- WBC count <4000cells/mm^3 OR >12,000 cells/mm^3
- respiratory rate > 20 or $PaCO_2$ < 32mm (4.3kPa)
- temperature less than 36 degrees or greater than 38 degrees

Calculation

each component (heart rate, WBC count, respiratory rate, temperature) is given 1 point if it meets the above criteria

Interpretation

score of 2 or more meets criteria for SIRS

ACUTE TRAUMATIC COAGULOPATHY

not simply a ‘dilutional coagulopathy’ or ‘consumptive coagulopathy’!

Pathophysiology

- Acute traumatic coagulopathy was conventionally construed simply as depletion, dysfunction or dilution of procoagulant factors
- Actually an imbalance of the dynamic equilibrium between procoagulant factors, anticoagulant factors, platelets, endothelium and fibrinolysis
- Characterized by isolated factor V inhibition, dysfibrinogenemia, systemic anticoagulation, impaired platelet function and hyperfibrinolysis
- Exacerbated by hypothermia, acidosis (together with coagulopathy they form **‘the lethal triad’**) and resuscitation with hypocoagulable fluids.

TRANEXAMIC ACID

FORMULATION:

Chemical Name: trans-4-(aminomethyl) cyclohexanecarboxylic acid.

STRUCTURAL FORMULA:

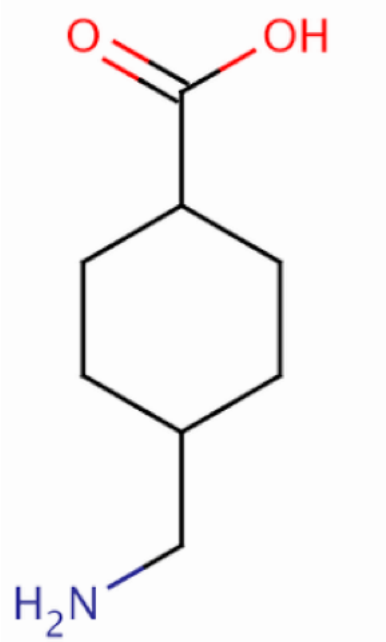


Figure 4. Tranexamic acid molecular structure

Empirical Formula: C8 H15 N02 . Molecular Weight: 157.2 Tranexamic acid is a white crystalline powder. The aqueous solution for injection has a pH of 6.5 to 8.0.

Tranexamic Acid is a drug administered for a variety of reasons. In Japan, women can buy it over the counter for heavy menstrual bleeding. It is used for prophylaxis before some knee surgeries to prevent bleeding. In the military, it is used as an anti-fibrinolytic in trauma patients and has been in use for several years at the

Role III hospitals in Afghanistan. In 2011, it was added to the Tactical Combat Casualty Care (TCCC) guidelines by the Committee on TCCC for the use for casualties that are expected to receive a blood transfusion.

MECHANISM OF ACTION

Tranexamic Acid is a synthetic derivative of the amino acid lysine, it inhibits fibrinolysis by blocking the lysine binding sites on plasminogen. In normal clotting cascade and fibrinolysis , plasminogen is cleaved to plasmin by tissue plasminogen activator (tPA). In turn, plasmin then breaks down fibrin, the insoluble substance that holds clots together. Therefore, by blocking this conversion of plasminogen to plasmin, the formed clot will be maintained.

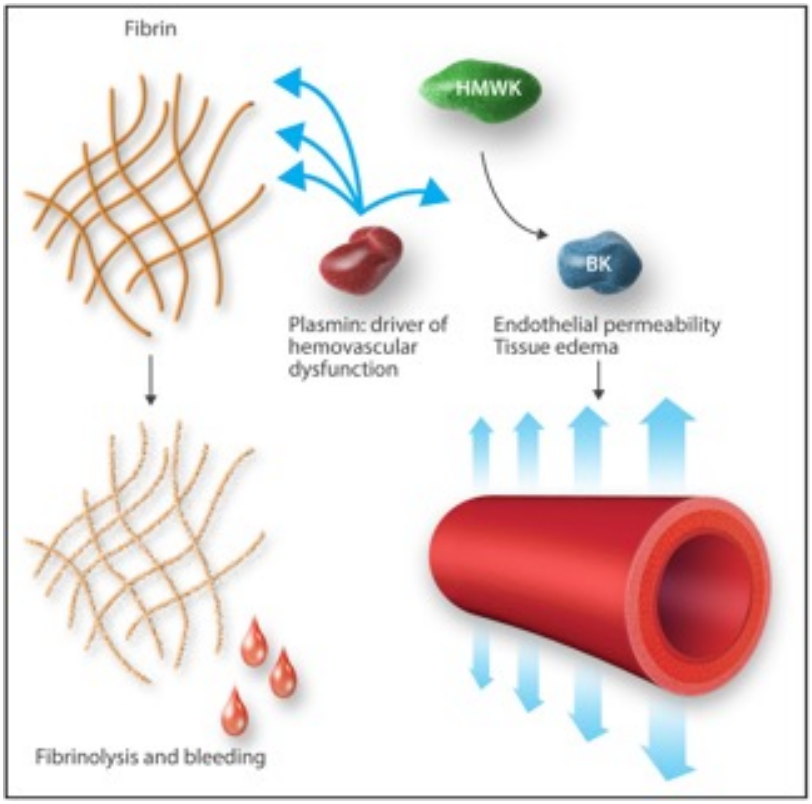


Figure 5. Clotting cascade and fibrinolysis

Due to trauma induced coagulopathy (TIC) and acute traumatic coagulopathy (ATC), there can be increased fibrinolysis in severe trauma . Both ATC and TIC are

convoluted and still not completely understood.

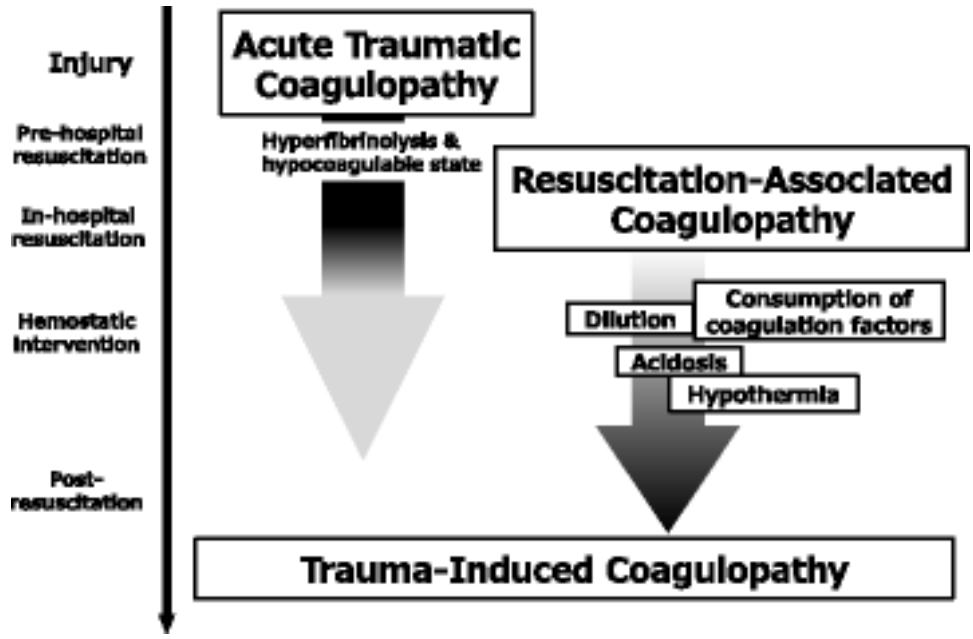


Figure 6. Trauma Induced Coagulopathy

There are several factors involved with Acute Traumatic Coagulopathy and Trauma Induced Coagulopathy. It is known that fibrinogen is being consumed rapidly and if there is no resuscitation, prothrombin drops up to 20%, and thrombin is increased 1.5 times over first 4 hours, and finally, plasmin rises 2.5 times. Some studies support the theory that activated protein C is the primary driver.

The current indications for the use of Tranexamic acid in Trauma are as follows:

"If a casualty is anticipated to need significant blood transfusion (for example: presents with hemorrhagic shock, one or more major amputations, penetrating torso trauma, or evidence of severe bleeding): Administer 1 gm of tranexamic acid in 100 ml Normal Saline or Lactated Ringer's as soon as possible but

NOT later than 3 hours after injury. When given, TXA should be administered over 10 minutes by IV infusion. Begin the second infusion of 1 gm TXA after initial fluid resuscitation has been completed."

The reasoning behind the 10 minute infusion, is due the possibility of hypotension if given too rapidly. This was observed and noted during the CRASH-2 study. Some argue that it can be pushed slowly in a 10 mL syringe over a few minutes. TXA should not be administered in the same line as blood or blood products or in a line used for rFVIIa or Penicillin. It should be stored between 15-30 C°or 56-86 F°. TXA is supplied as 1 gram in a 10 mL ampule or vial.

Side effects include:

Ocular – color vision change, vision loss

Seizure – probably related to neuronal GABA inhibition

Renal Impairment

Ureteral Obstruction – upper tract obstruction may lead to bleeding

The contraindications for TXA:

Acquired defective color vision

SAH

Active intravascular clotting

Hypersensitivity to TXA

METHODOLOGY

a) SOURCE OF DATA:

Patients admitted in the emergency trauma centre of Stanley Medical College hospital,Chennai.

b) METHOD OF COLLECTION OF DATA (including sampling procedure, if any):

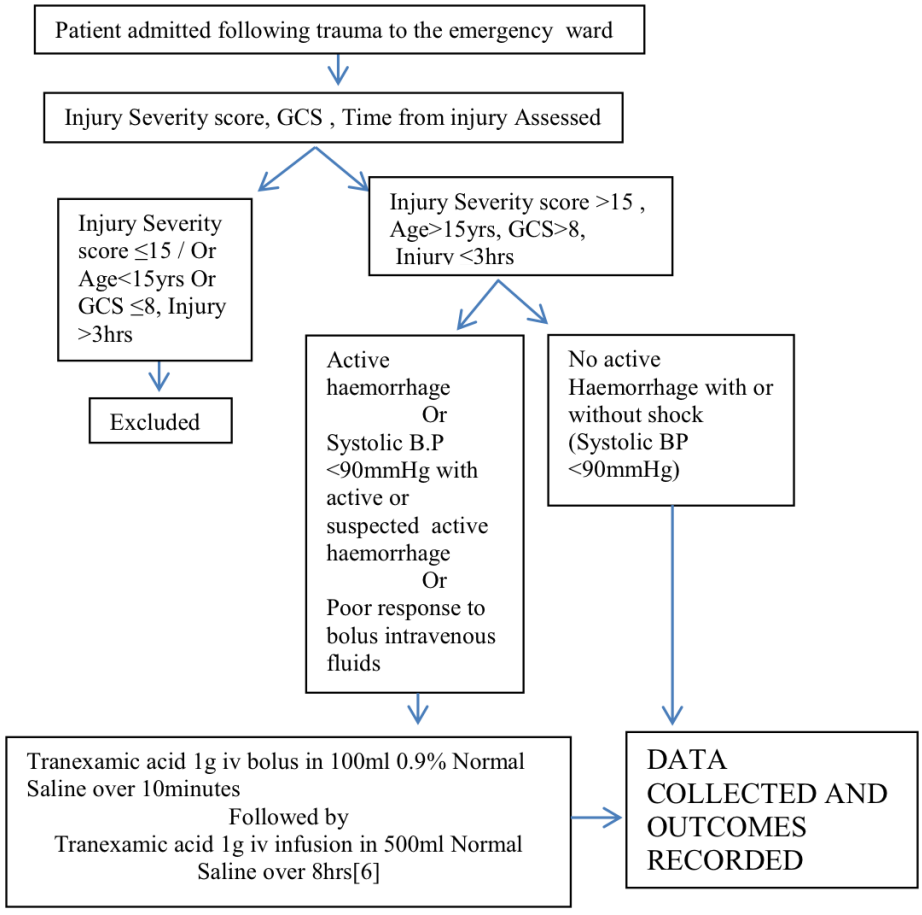


Figure 7. Methodology

Data Collected:

1. Patient demographics

2. Mode of Injury

3. Mechanism of Injury(Blunt/Penetrating)

4. Injury Severity Score

5. Abbreviated Injury Score

6. Glasgow Coma Score

7. Systolic BP

8. Details of transfusions in first 24 hrs from injury

9.Post transfusion Haemoglobin after 24 hrs

10. Fluid resuscitation details

Outcomes to be recorded:

1.Mortality <48hrs

3.Cause of Mortality

4.Critical Care length of stay

c.)INCLUSION CRITERIA:

1. Age >15yrs

2. Injury Severity Score >15 with or without active haemorrhage

EXCLUSION CRITERIA:

1. Age <15 years

2. Injury Severity Score ≤15

3. Duration from time of injury >3hrs

4. GCS Score ≤8

PERIOD OF STUDY: One year. June 2018 to June 2019

SAMPLE SIZE CALCULATION

Reference Study

Tranexamic Acid Use in Severely injured civilian patients and the effects on Outcomes Elaine Cole MSc,Ross Davenport Phd, Keith Willet FRCS and Karim Brohi FRCS,FRCA Ann Surg 2015:261:390-394

Sample size of reference Study = 385

No.of severely injured individuals who received Tranexamic acid= 162

Therefore, percentage who received Tranexamic acid= (162/385)*100 = 42.1%

Sample size **$N=4pq/ d^2$**

N = sample size

p = percentage of population who received intervention in previous study

q = 100-p

d = 20 % of p (considering margin of error at 20%)

Calculation: N= [4* 42.1* (100-42.1)] / (20*42.1/100)

N= 137.5

On rounding off **Sample size (N) = 138**

RESULTS

The study was conducted for a period of one year in the emergency department of Government Stanley Medical College, Chennai. All variables and outcomes were recorded according to the methodology.

A total of 138 patients were included in the study with Injury Severity scale more than 15 and trauma within 3 hours. Of the 138 patient cohorts with severe injury 56 patients (40.6%)received tranexamic acid and 82 patients (59.4%) were categorized into the non tranexamic acid group.

DATA ANALYSIS

Data was collected and analysed using Microsoft Excel 2016 Professional edition and IBM SPSS v25 and Graphpad Prism softwares.

Data was compiled with Microsoft Excel 2016 professional edition. Descriptive analysis of all variables was conducted using IBM SPSS v25 software. Unpaired t test was used to compare and analyse the variables and outcomes between the two cohort groups.

I.)DISTRIBUTION OF TRANEXAMIC AND NONTRANEXAMIC COHORTS

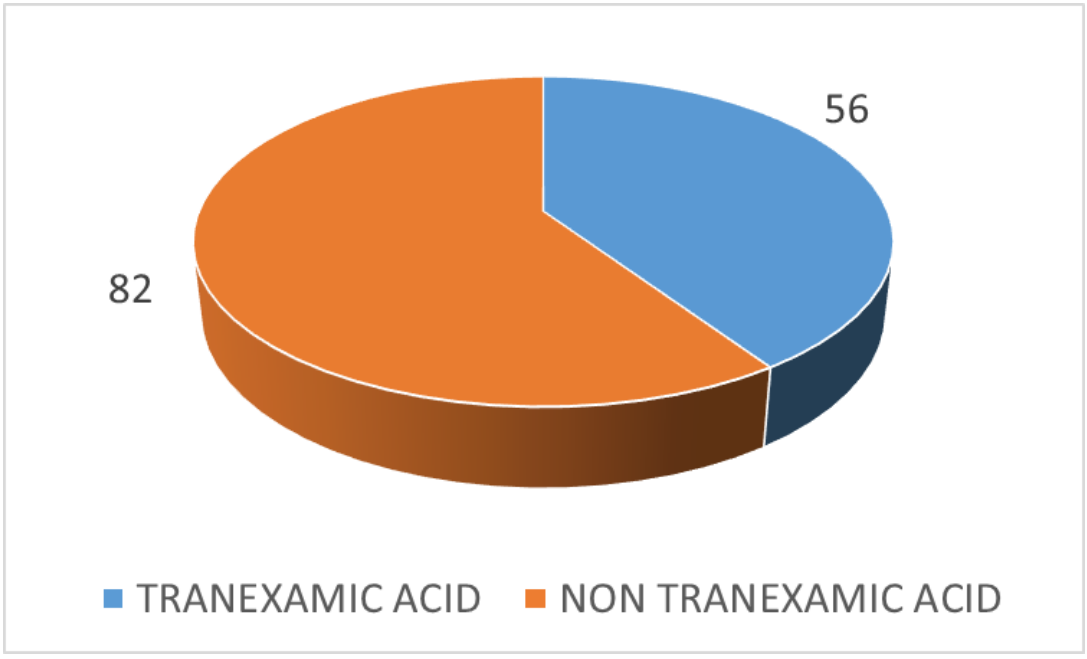


Figure 8

Distribution of tranexamic and nontranexamic cohorts

Total participants of the study were 138 satisfying the inclusion criteria. Among these patients 56 patients received Tranexamic acid (40.6%)and 82 patients (59.4%) were grouped into the non tranexamic acid group.

II.) SEXWISE DISTRIBUTION

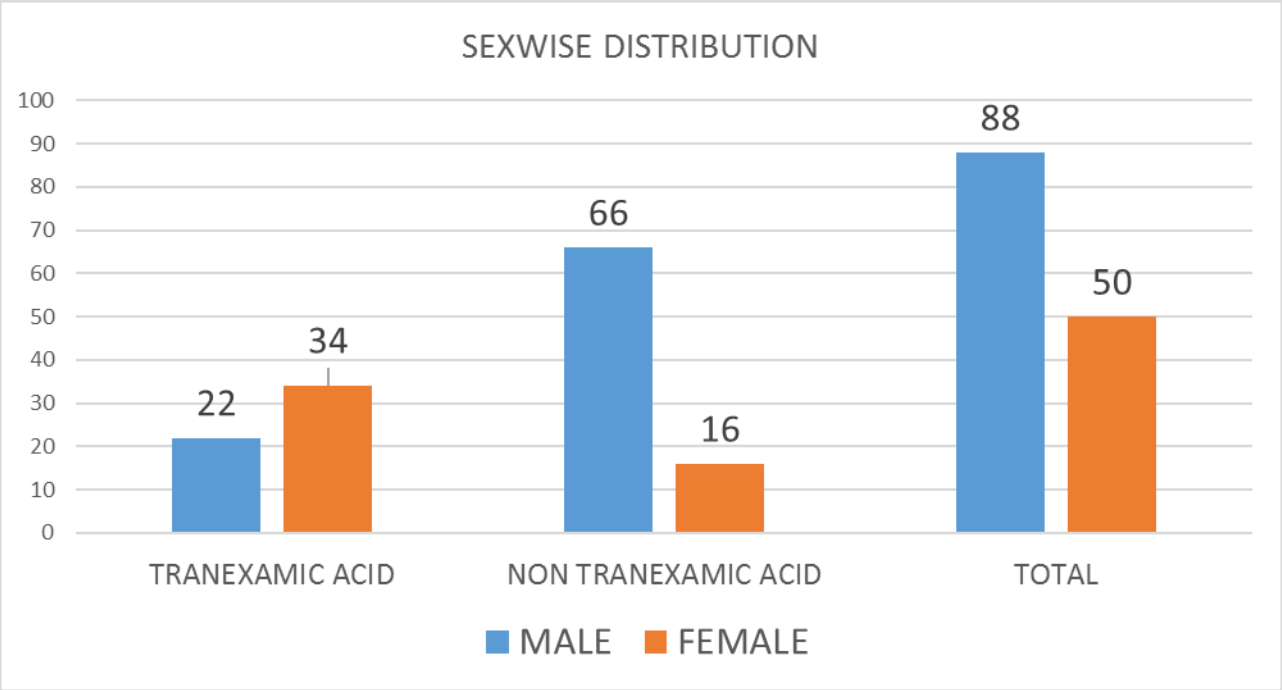


Figure 9. Sexwise distribution

Of the total 138 patients included in the study there were 88 males (63.8%) and 50 females (36.2%). 56 patients (22 males and 34 females - 40.5%) received tranexamic acid and 82 patients(66 males and 16 females – 49.5%)were categorised into the non tranexamic acid group.

Tranexamic requirement was found to be more in females.

III.)MODE OF INJURY

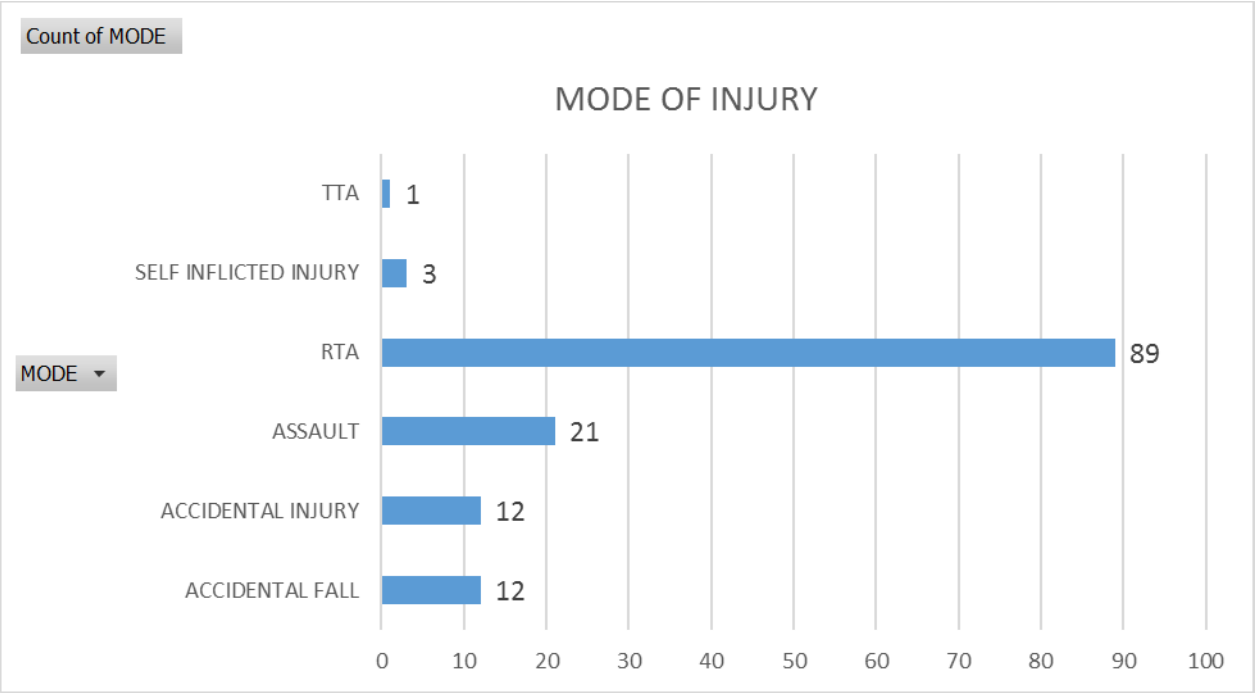


Figure10.Mode of Injury

The most common mode of injury was Road Traffic Accidents(RTA) contributing to 89 cases(64.5%) , followed by assault with 21 cases(15.2%). There were 12 cases(8.7%) due to accidental injury and 12 cases(8.7%) due to accidental fall. 3 cases were due to self inflicted injury (2.2%) and 1 case due to Train Traffic Accident(0.7%)

III A.) MODE OF INJURY IN TRANEXAMIC ACID GROUP.

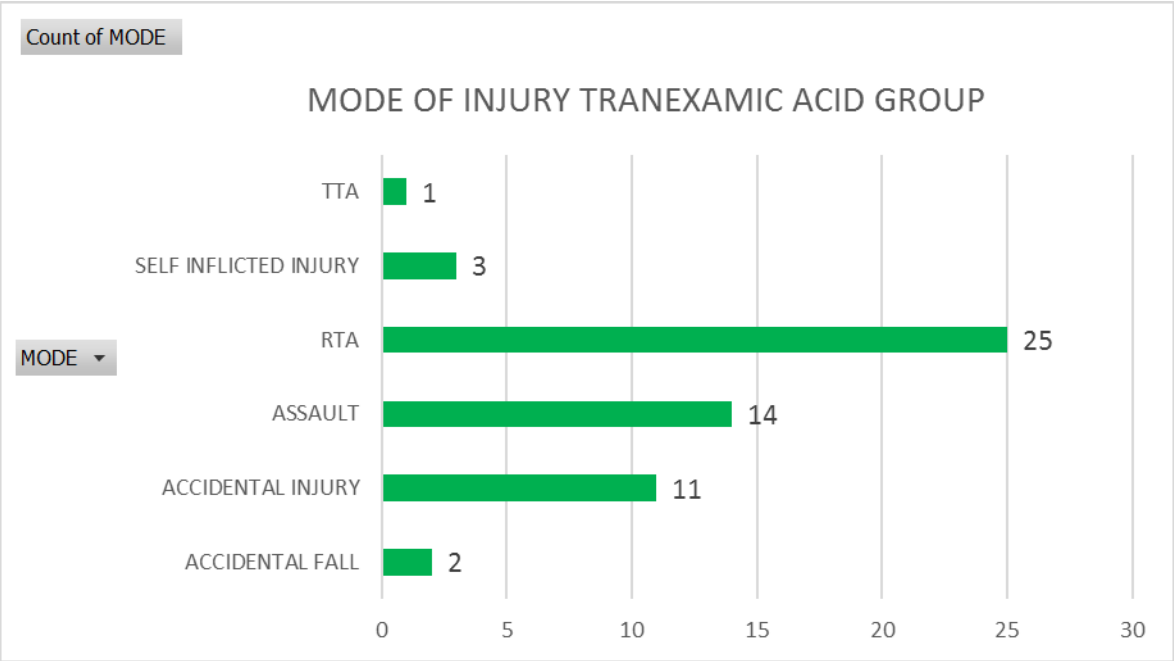


Figure 11. Mode of Injury in Tranexamic acid group

The most common mode of injury in the Tranexamic acid group was Road Traffic Accidents(RTA) contributing to 25 cases(44.6%) , followed by assault with 14 cases(25%). There were 11 cases(19.6%)due to accidental injury and 2 cases(3.6%) due to accidental fall. 3 cases were due to self inflicted injury (5.3%) and 1 case due to Train Traffic Accident(1.8%).

III B).MODE OF INJURY IN NON-TRANEXAMIC ACID GROUP

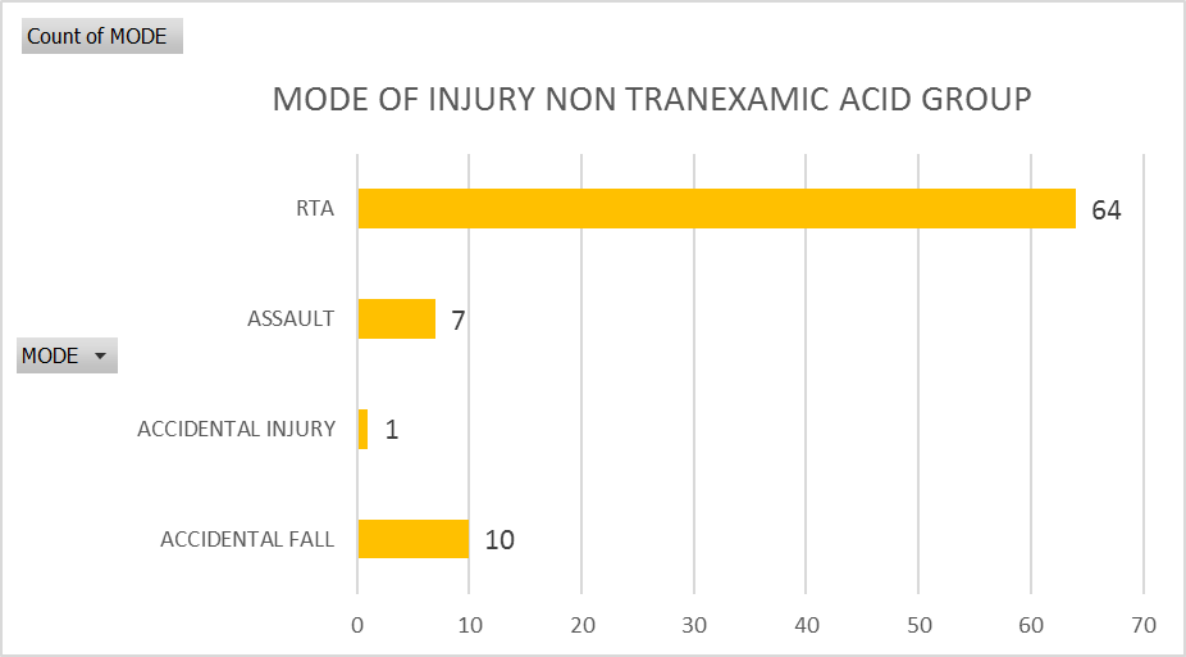


Figure 12. Mode of Injury in Non Tranexamic acid group

The most common mode of injury in the Non Tranexamic acid group was Road Traffic Accidents(RTA) contributing to 64 cases(78%) , followed by assault with 14 cases(8.5%). There was 1 cases(1.2%)due to accidental injury and 10 cases(12.2%) due to accidental fall.

IV.) TYPE OF INJURY

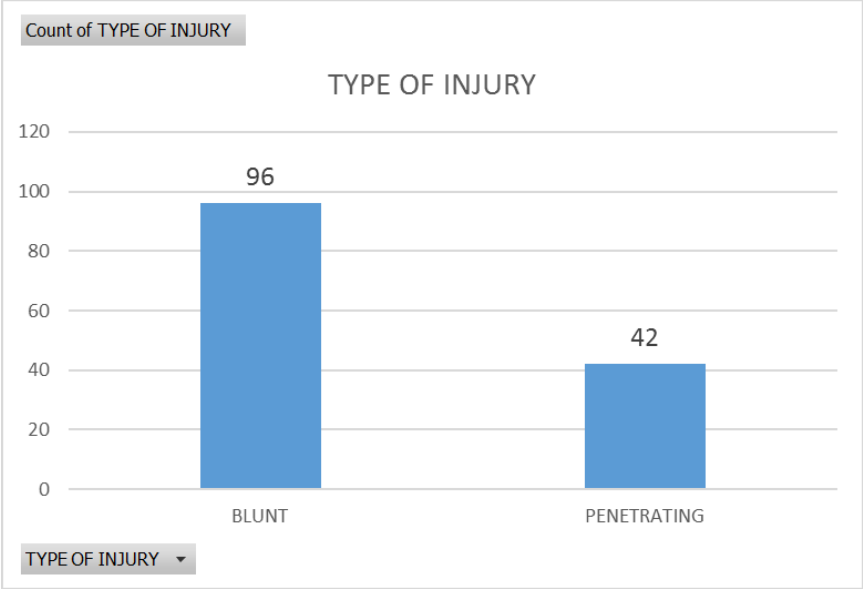


Figure 13. Type of injury

The most overall type of injury was due to blunt trauma with 96 cases (69.5%)and penetrating trauma with 42 cases(30.5%).

IV A.) TYPE OF INJURY IN TRANEXAMIC ACID GROUP

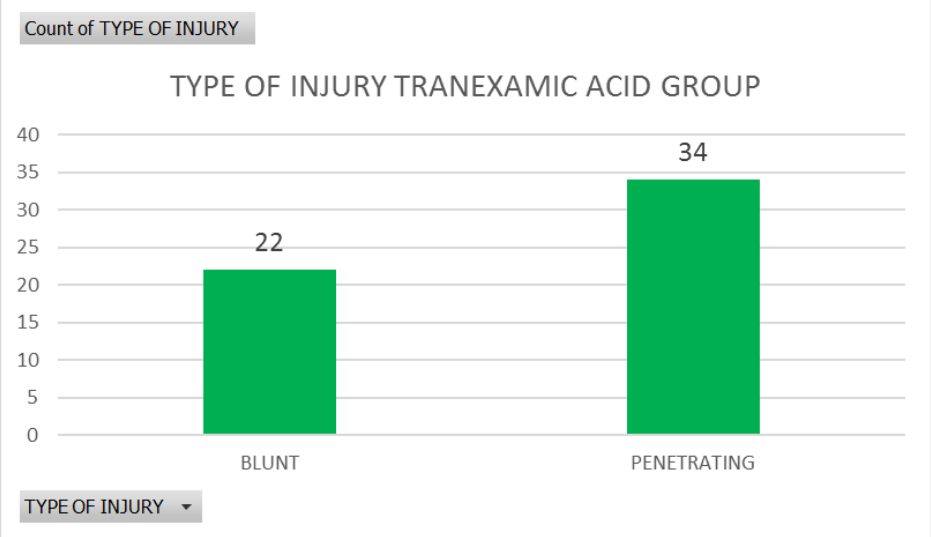


Figure 14. Type of injury in Tranexamic acid group.

The most common mode of injury was due to penetrating trauma with 34 cases(60.7%) followed by blunt trauma with 22 cases (39.3%).

IV B.) TYPE OF INJURY IN TRANEXAMIC ACID GROUP

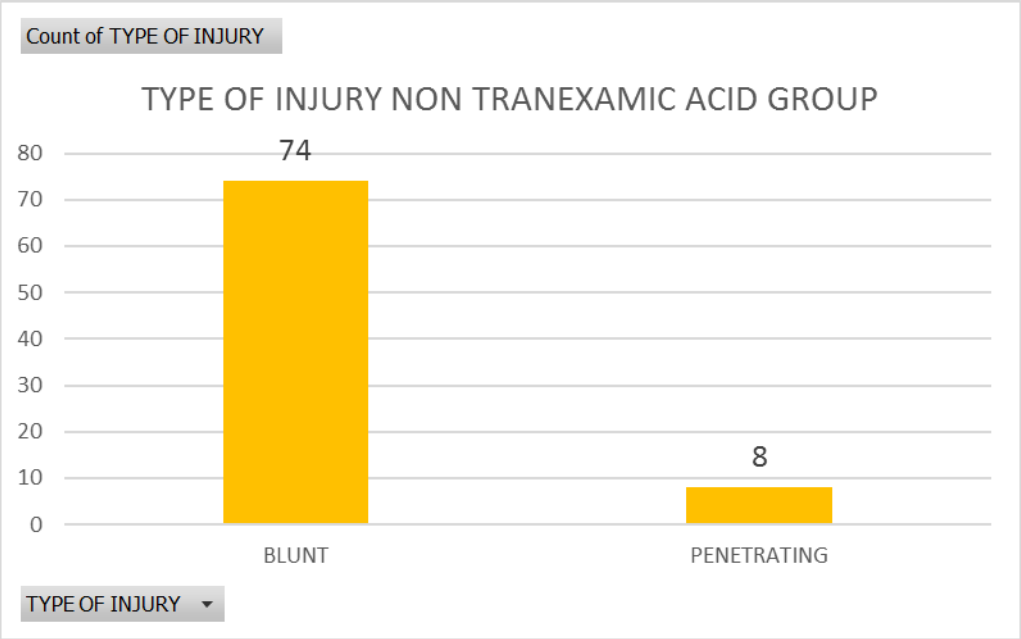


Figure 15. Type of injury in non tranexamic acid group

The most common mode of injury was due to blunt trauma with 74 cases(90.2%) followed by blunt trauma with 8 cases (9.8%).

V.) DURATION FROM TIME OF INJURY

The average duration from time of injury to administration of loading dose tranexamic acid was **124 minutes**.

VI.) AGE DISTRIBUTION

Statistics

AGE

N	Valid	138
	Missing	0
Mean		38.49
Median		36.50
Std. Deviation		12.713

Table 3. Age distribution

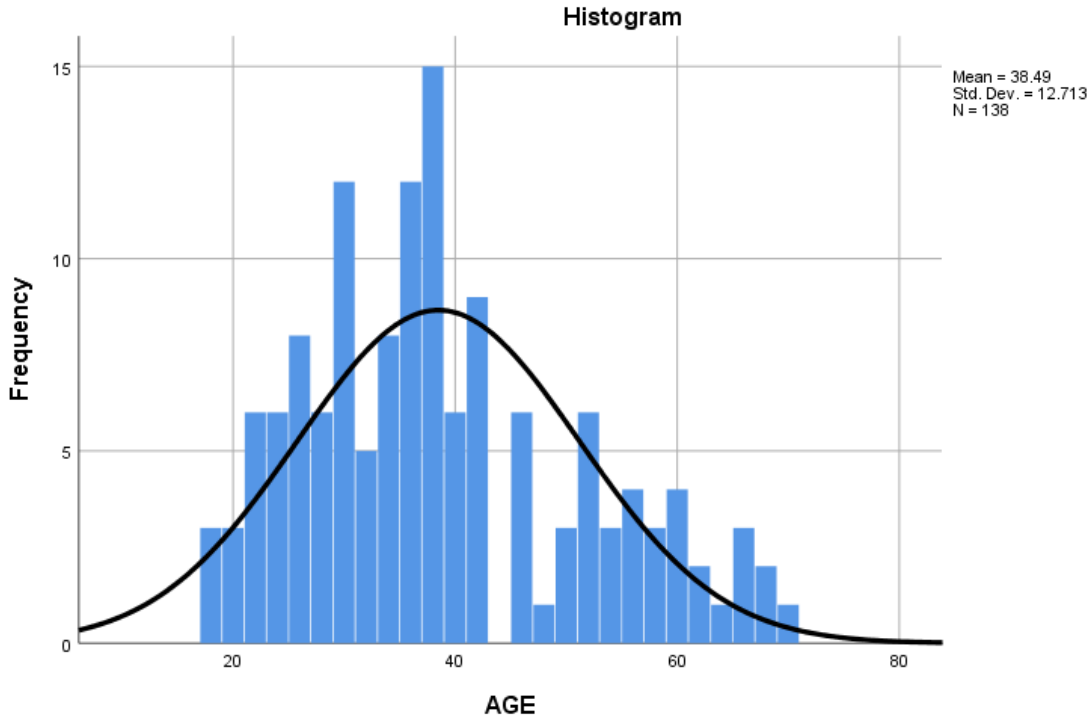


Figure 16. Histogram of Age distribution

The Mean Age was **38.49 years.** The Median Age was **36.50 years** and the Standard Deviation was **12.71 years**

Trauma was found to be most commonly affecting the middle aged group.

VI.A) AGE DISTRIBUTION TRANEXAMIC ACID GROUP

Statistics

AGE

N	Valid	56
	Missing	0
Mean		36.23
Median		34.00
Mode		34
Std. Deviation		13.090

Table 4. Age distribution in Tranexamic acid group

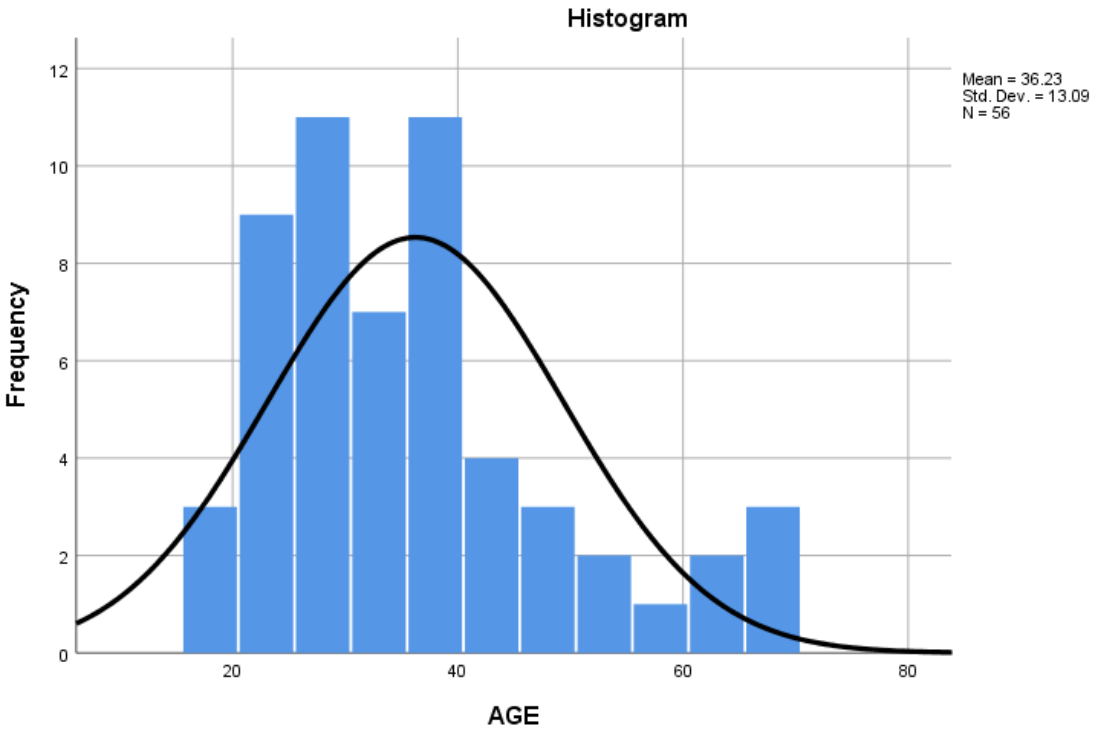


Figure 17. Histogram of Age distribution in Tranexamic acid group

The Mean Age was **36.23years.** The Median Age was**34.00 years** and the Standard Deviation was **13.09years**

Trauma was found to be most commonly affecting the middle aged group in the tranexamic acid group.

VI B). AGE DISTRIBUTION NON TRANEXAMIC ACID GROUP

Statistics

AGE

N	Valid	82
	Missing	0
Mean		40.02
Median		37.00
Std. Deviation		12.292

Table 5. Age distribution in non tranexamic acid group

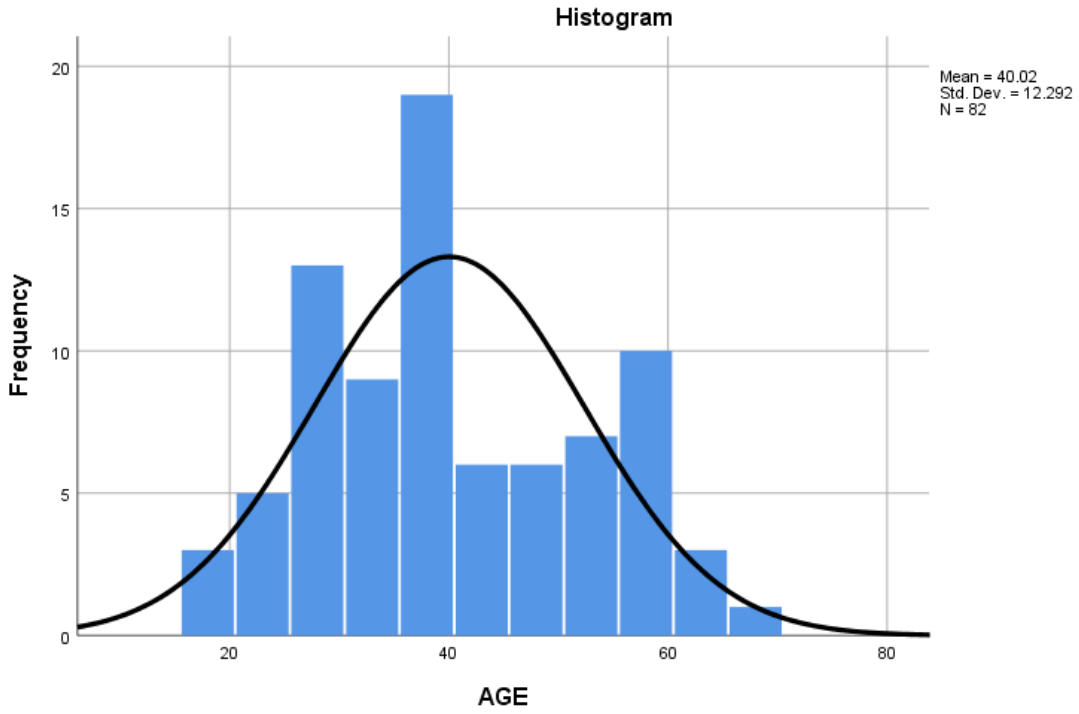


Figure 18. Histogram of Age distribution in non tranexamic acid group.

The Mean Age was **40.02years.** The Median Age was **37.00 years** and the Standard Deviation was **12.292years**

Trauma was found to be most commonly affecting the middle aged group in the non tranexamic acid group.

VII) GLASGOW COMA SCORE

Statistics

GCS

N	Valid	138
	Missing	0
Mean		13.58
Median		14.00
Std. Deviation		1.513
Percentiles	25	13.00
	50	14.00
	75	15.00

Table 6. Glasgow Coma Score

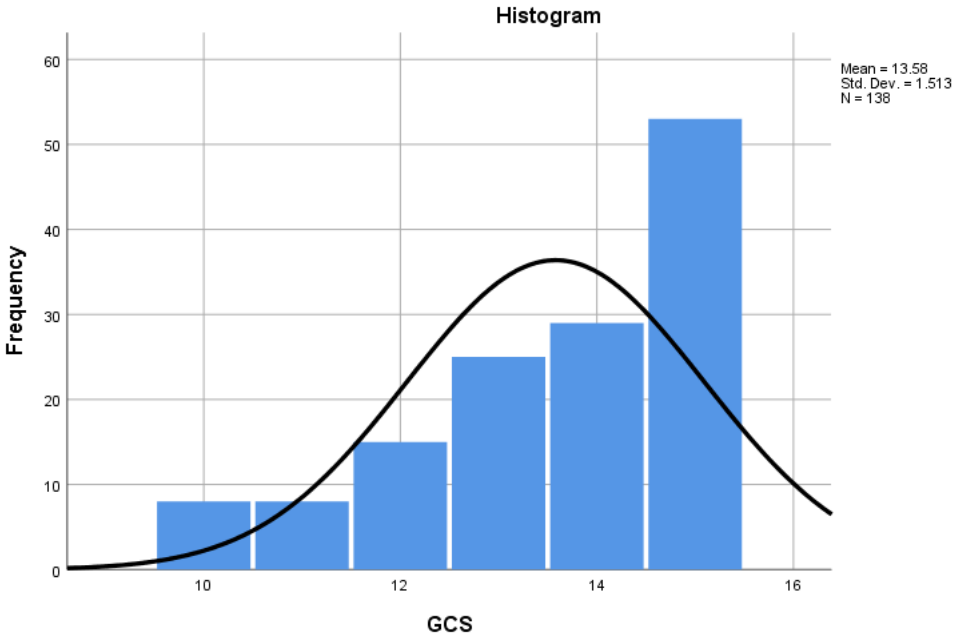


Figure 19. Histogram of Glasgow Coma Score

The Mean Glasgow coma score was **13.58.** The Median score was **14.00** and the standard deviation was **1.513.**

VIIA) GLASGOW COMA SCORE IN TRANEXAMIC ACID GROUP

Statistics

GCS

N	Valid	56
	Missing	0
Mean		13.77
Median		14.00
Std. Deviation		1.561
Percentiles	25	13.00
	50	14.00
	75	15.00

Table 7. Glasgow Coma Score in Tranexamic acid group

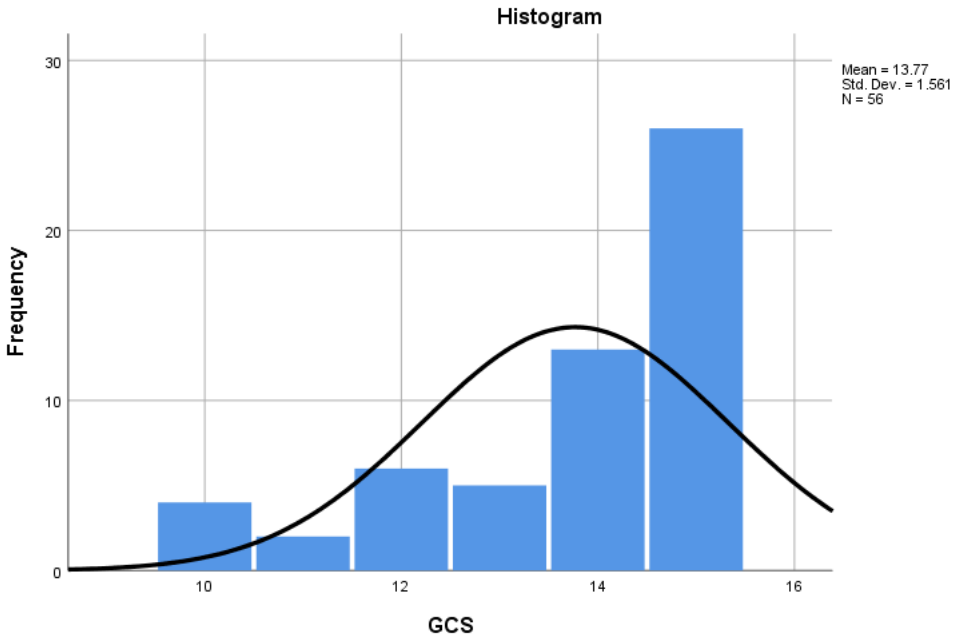


Figure 20. Histogram of Glasgow Coma Score in Tranexamic acid group

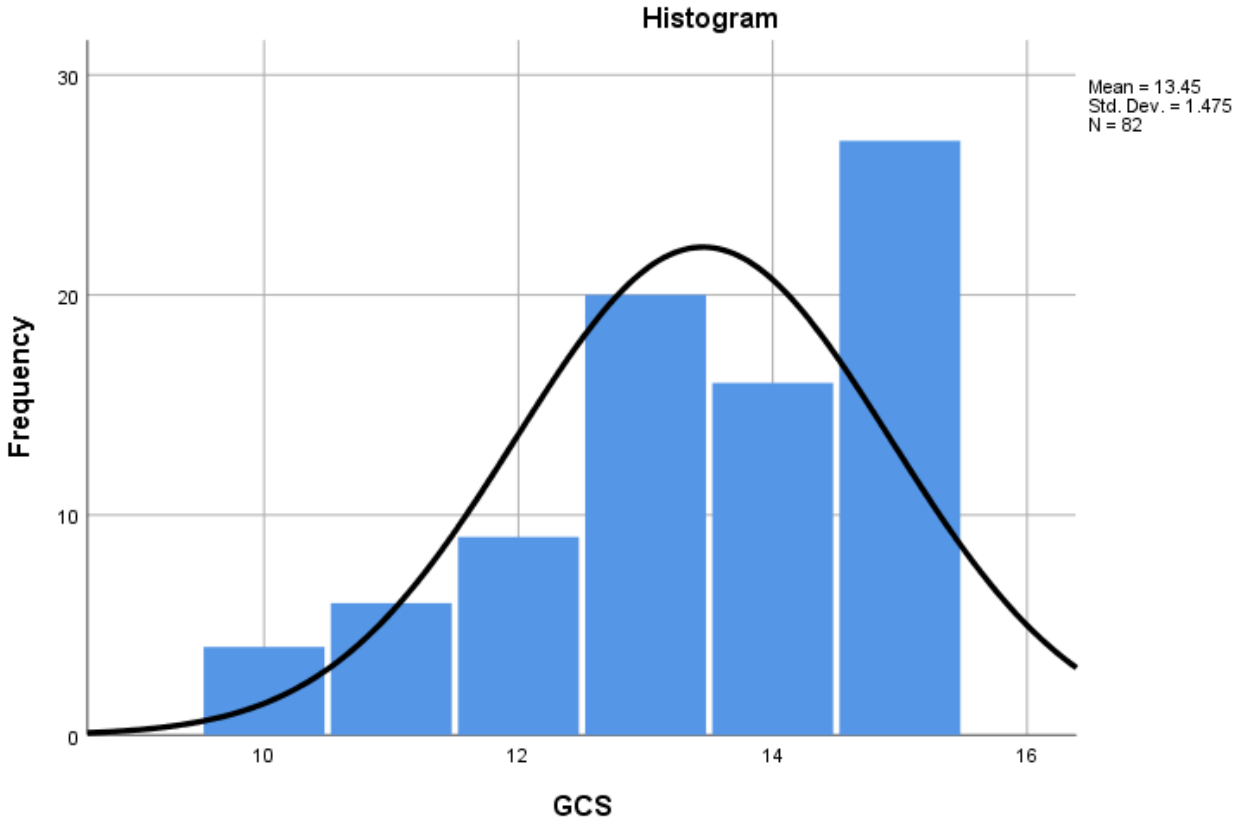
The Mean Glasgow coma score was **13.77.** The Median score was **14.00** and the standard deviation was **1.561**

VII B.) GLASGOW COMA SCORE NON TRANEXAMIC ACID GROUP

Statistics

GCS

N	Valid	82
	Missing	0
Mean		13.45
Median		14.00
Std. Deviation		1.475



The mean score was 13.45. The Median was 14.00

VII C.) GLASGOW COMA SCORE ANALYSIS -UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
GLASGOW COMA SCORE	TRANEXAMIC ACID	56	13.7679	1.56078	.20857
	NONTRANEXAMIC ACID	82	13.4512	1.47533	.16292

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
GLASGOW COMA SCORE	Equal variances assumed	.015	.904	1.209	136	.229	.31664	.26185	-.20118	.83446
	Equal variances not assumed			1.196	113.823	.234	.31664	.26466	-.20766	.84093

There was no significance observed between the Glasgow Coma Score between the Tranexamic acid and non tranexamic acid group (**p>0.05**)

VIII.) INJURY SEVERITY SCALE (ISS)

Statistics

ISS

N	Valid	138
	Missing	0
Mean		17.49
Median		17.00
Std. Deviation		2.186

Table 11. Injury Severity Scale

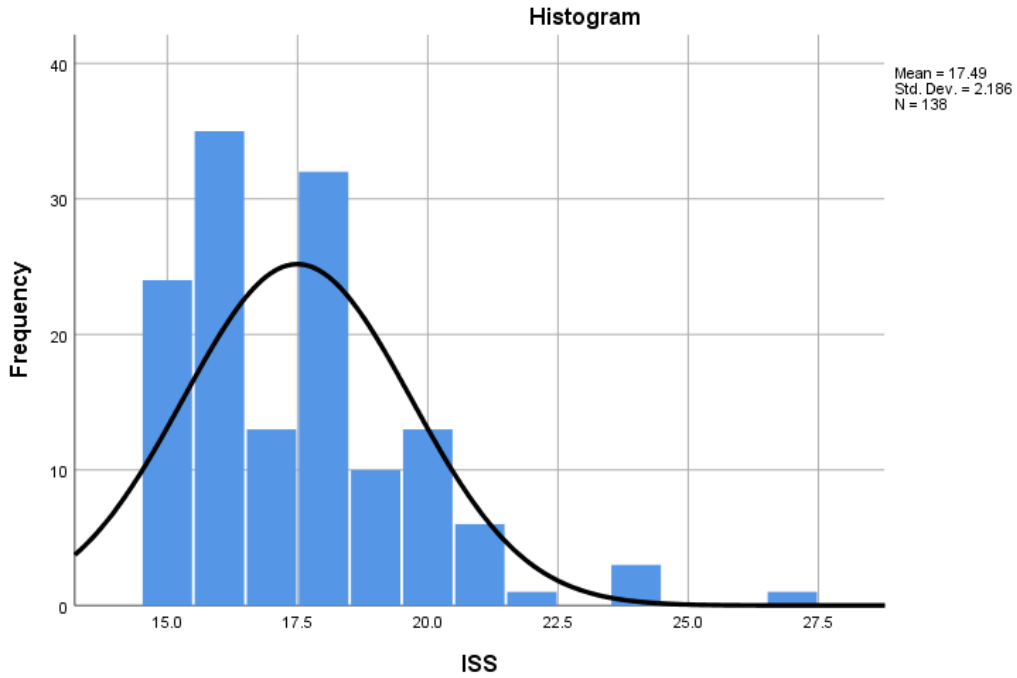


Figure 22. Histogram of Injury severity scale

The mean injury severity scale score was 17.49

The Median injury severity scale score was 17. The Standard deviation was 2.18

VIII A.) INJURY SEVERITY SCALE IN TRANEXAMIC ACID GROUP

Statistics

ISS

N	Valid	56
	Missing	0
Mean		18.88
Median		19.00
Mode		20
Std. Deviation		2.501

Table 12. Injury Severity scale in Tranexamic acid group

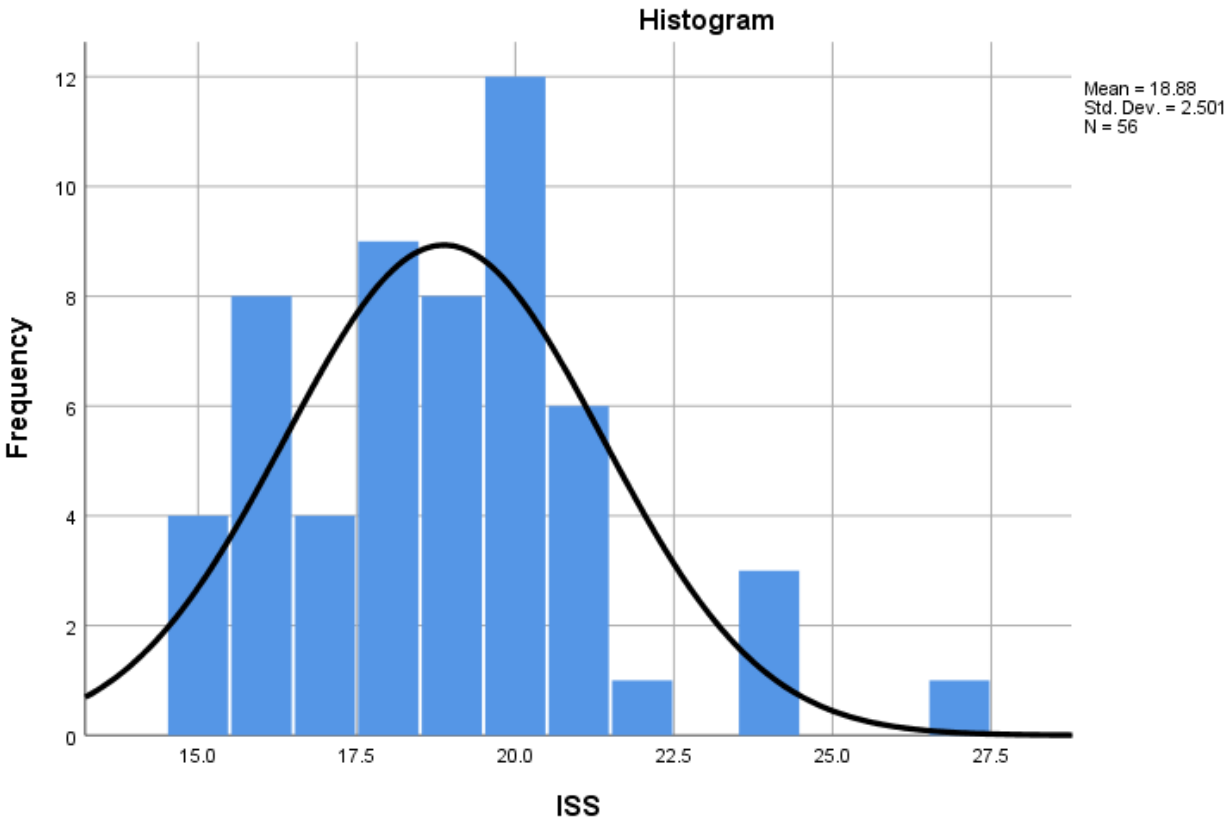


Figure 23. Histogram of Injury Severity scale in Tranexamic acid group

The mean injury severity scale score in Tranexamic acid group was 18.88

The Median injury severity scale score was 19. The Standard deviation was 2.50

VIII B). INJURY SEVERITY SCALE IN NON TRANEXAMIC ACID GROUP

Statistics

ISS

N	Valid	82
	Missing	0
Mean		16.55
Median		16.00
Std. Deviation		1.268

Table 13. Injury Severity Scale in non tranexamic acid group

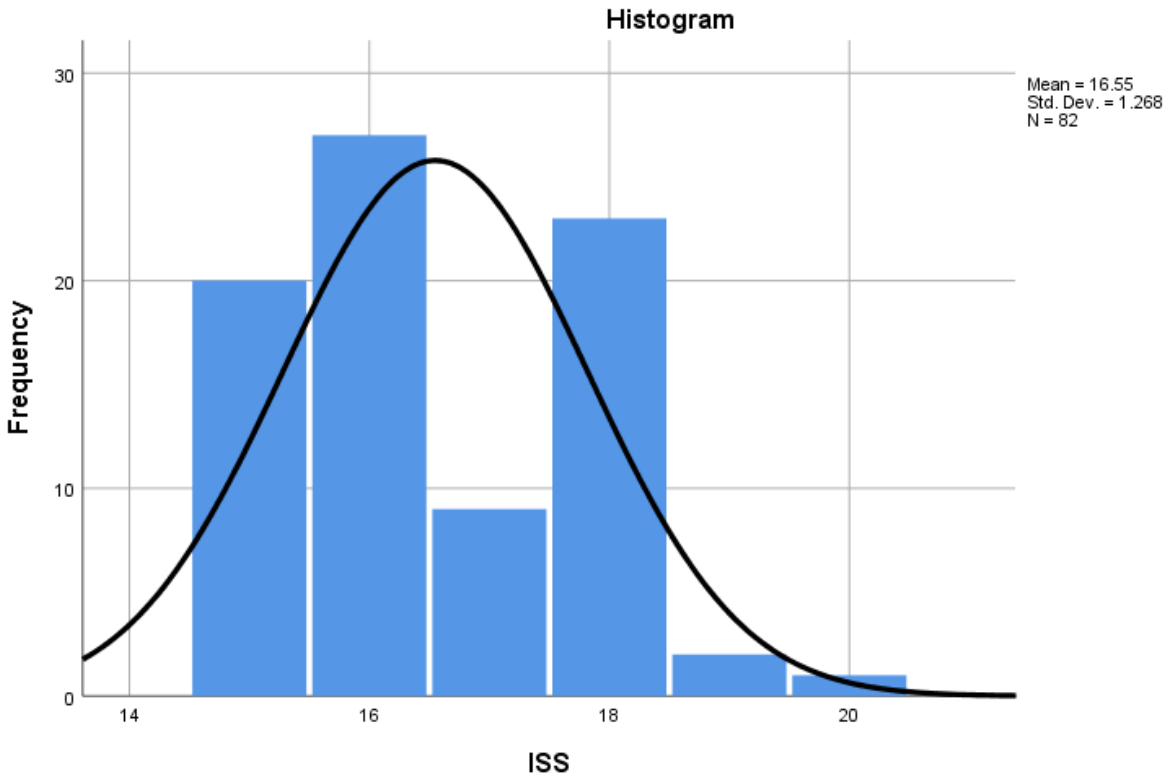


Figure 24. Histogram of Injury Severity Scale in non tranexamic acid group

The mean injury severity scale score in Tranexamic acid group was 16.55

The Median injury severity scale score was 16. The Standard deviation was 1.268

VIII C.)ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
ISS	TRANEXAMIC ACID	56	18.8750	2.50136	.33426
	NONTRANEXAMIC ACID	82	16.5488	1.26835	.14007

Table 14. Descriptive statistics of Injury Severity Scale between the cohort groups

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
ISS	Equal variances assumed	17.891	**.000**	7.184	136	.000	2.32622	.32378	1.68592	2.96652
	Equal variances not assumed			6.419	74.452	.000	2.32622	.36242	1.60416	3.04828

Table 15. Independent Samples test of Injury Severity Scale between the cohort groups

On unpaired t test, Tranexamic acid group cases had more significant severity of injury (**$p<0.05$**) when compared to the non tranexamic acid group

IX.)SYSTOLIC BLOOD PRESSURE

Statistics

SYSTOLIC BP

N	Valid	138
	Missing	0
Mean		104.04
Median		104.00
Std. Deviation		9.778

Table 16. Systolic blood pressure

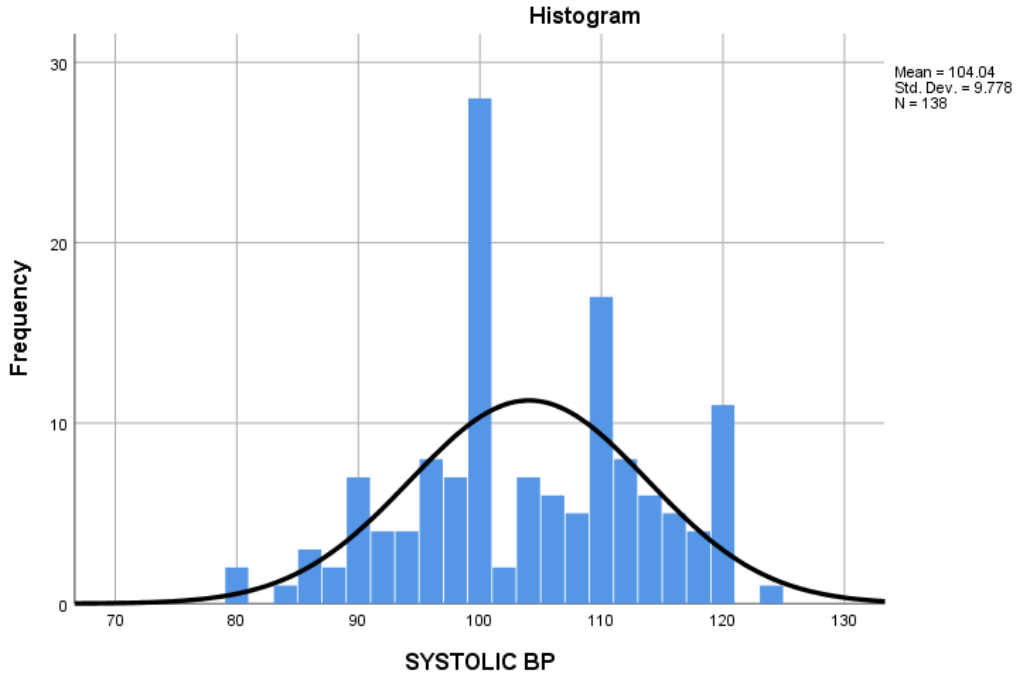


Figure 25. Histogram of systolic blood pressure

The mean systolic blood pressure was 104.04mm Hg. The Median blood pressure was 104mm Hg and Standard deviation was 9.77mm Hg

IX A.) SYSTOLIC BLOOD PRESSURE IN TRANEXAMIC ACID GROUP

Statistics

SYSTOLIC BP

N	Valid	56
	Missing	0
Mean		96.70
Median		98.00
Mode		100
Std. Deviation		7.442

Table 17. Systolic BP in Tranexamic acid group

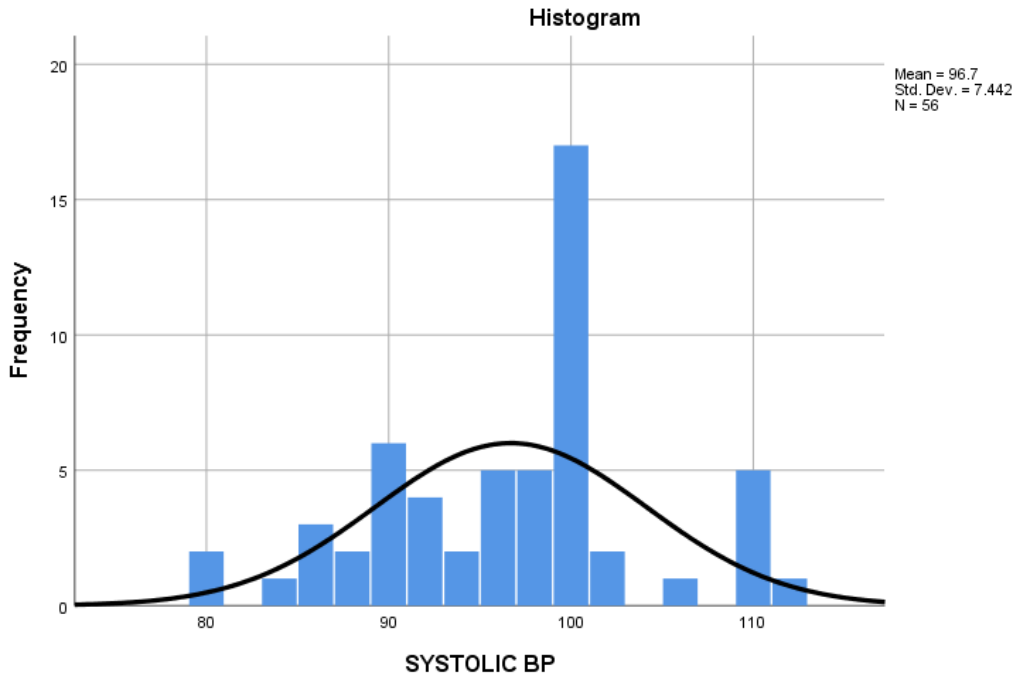


Figure 26. Histogram of Systolic BP in Tranexamic acid group

The mean systolic blood pressure was 96.7mm Hg. The Median blood pressure was 98mm Hg and Standard deviation was 7.44mm Hg

IX B). SYSTOLIC BLOOD PRESSURE IN NON TRANEXAMIC ACID GROUP

Statistics

SYSTOLIC BP

N	Valid	82
	Missing	0
Mean		109.05
Median		110.00
Std. Deviation		7.838

Table 18. Systolic blood pressure in non tranexamic acid group

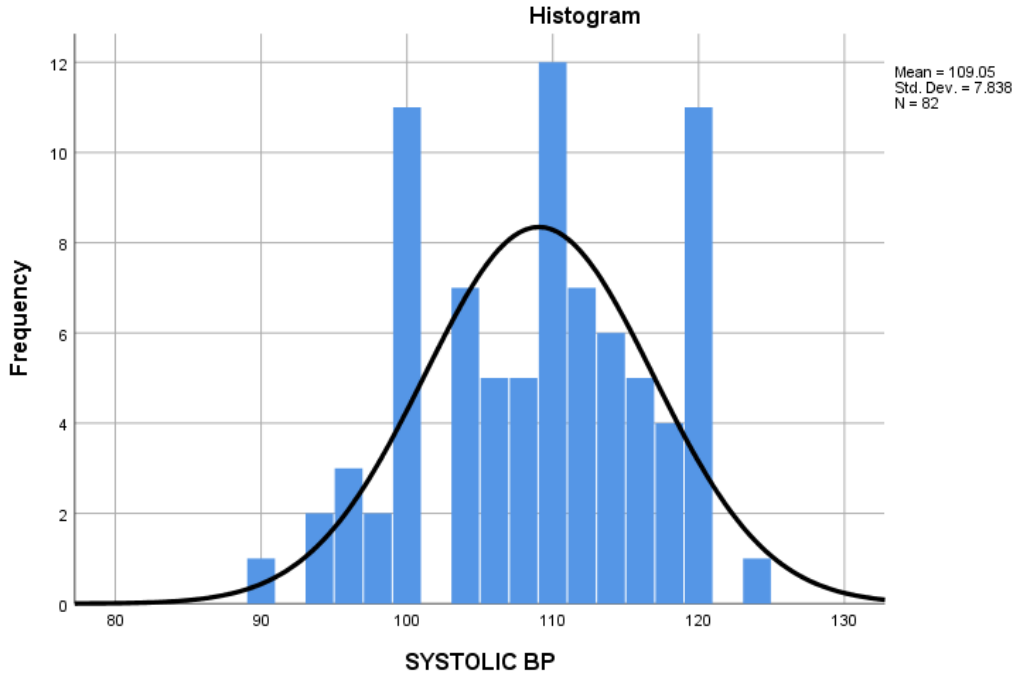


Figure 27. Histogram of Systolic blood pressure in non tranexamic acid group

The mean systolic blood pressure was 109.05mm Hg. The Median blood pressure was 110mm Hg and Standard deviation was 7.84mm Hg

IX C.) SYSTOLIC BLOOD PRESSURE ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
SYSTOLIC BP	TRANEXAMIC ACID	56	96.6964	7.44170	.99444
	NONTRANEXAMIC ACID	82	109.0488	7.83771	.86553

Table 19. Descriptive statistics of Injury Severity Scale between the cohort groups

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
SYSTOLIC BP	Equal variances assumed	.637	.426	-9.278	136	.000	-12.35235	1.33138	-14.98523	-9.71947
	Equal variances not assumed			-9.370	122.254	.000	-12.35235	1.31835	-14.96211	-9.74260

Table 20. Independent Samples test of Systolic blood pressure between the cohort groups

The was no significant difference between the systolic blood pressure between both the Tranexamic acid and non tranexamic acid group ($p>0.05$). Though both the cohort groups presented with a lower than average systolic BP due to the increased acuity of injuries.

X.) PULSE RATE

Statistics

PULSE RATE

N	Valid	138
	Missing	0
Mean		97.93
Median		96.00
Std. Deviation		10.777

Table 21. Pulse rate

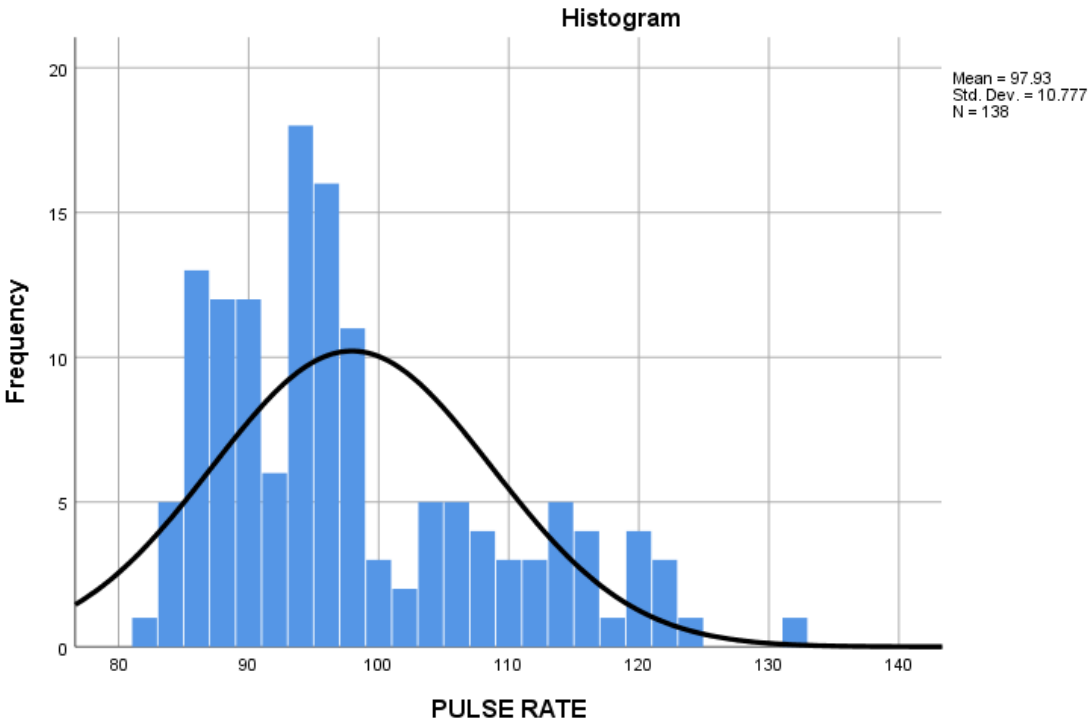


Figure 28. Histogram of Pulse rate

The mean pulse rate was 98 per minute. The Median pulse rate was 96 per minute.

The standard deviation was 10.77 per minute

X A.) PULSE RATE IN TRANEXAMIC ACID GROUP

Statistics

PULSE RATE

N	Valid	56
	Missing	0
Mean		105.20
Median		104.00
Mode		96
Std. Deviation		10.471

Table 22. Pulse rate in Tranexamic acid group

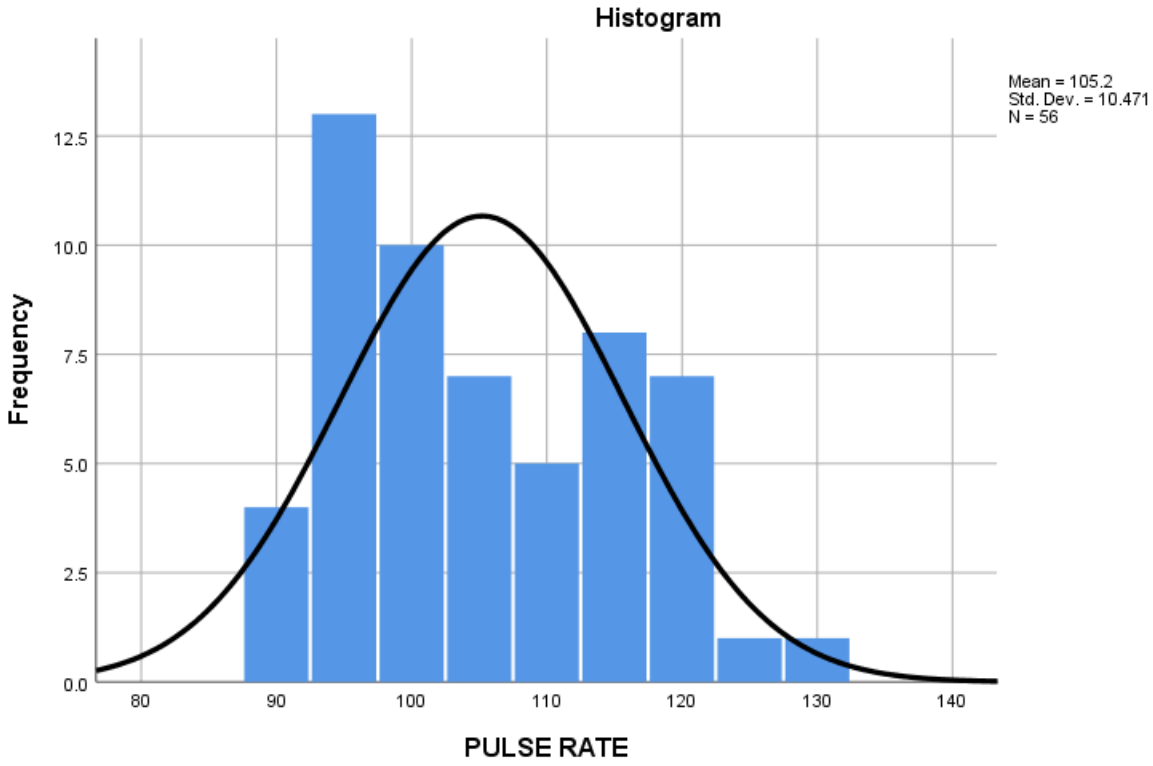


Figure 29. Histogram of Pulse rate in Tranexamic acid group

The mean pulse rate was 105 per minute. The Median pulse rate was 104 per minute.

The standard deviation was 10.47 per minute.

X B). PULSE RATE IN NON TRANEXAMIC ACID GROUP

Statistics

PULSE RATE

N	Valid	82
	Missing	0
Mean		92.98
Median		91.00
Std. Deviation		7.787

Table 23. Pulse rate in non tranexamic acid group

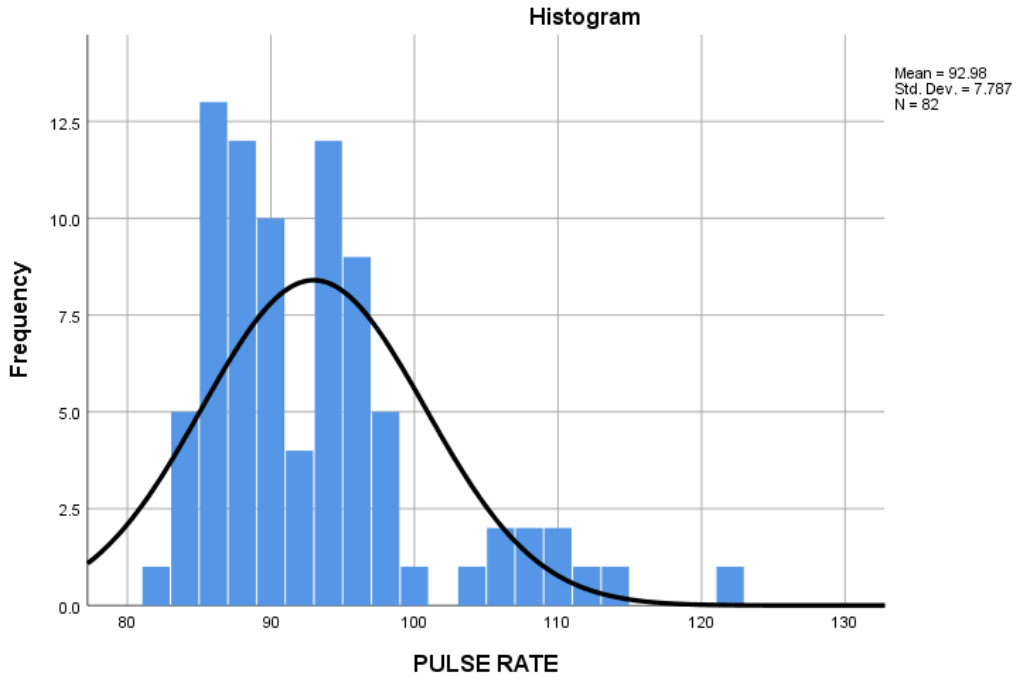


Figure 30. Histogram of Pulse rate in non tranexamic acid group

The mean pulse rate was 92 per minute. The Median pulse rate was 91 per minute.

The standard deviation was 7.87per minute.

X C.) PULSE RATE ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
PULSE RATE	TRANEXAMIC ACID	56	105.1964	10.47147	1.39931
	NONTRANEXAMIC ACID	82	92.9756	7.78726	.85996

Table 24. Descriptive statistics of Pulse rate between the cohort groups

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
PULSE RATE	Equal variances assumed	12.804	.000	7.859	136	.000	12.22082	1.55501	9.14569	15.29595
	Equal variances not assumed			7.441	95.172	.000	12.22082	1.64244	8.96025	15.48139

Table 25. Independent Samples test of Pulse rate between the cohort groups

The Tranexamic acid group patients had significantly tachycardia ($p<0.05$) owing to the volume low due to active bleeding and increased acuity of injury.

XI.)RESPIRATORY RATE

Statistics

		RESPIRATORY RATE
N	Valid	138
	Missing	0
Mean		20.88
Median		20.00
Std. Deviation		2.239

Table 26. Respiratory rate

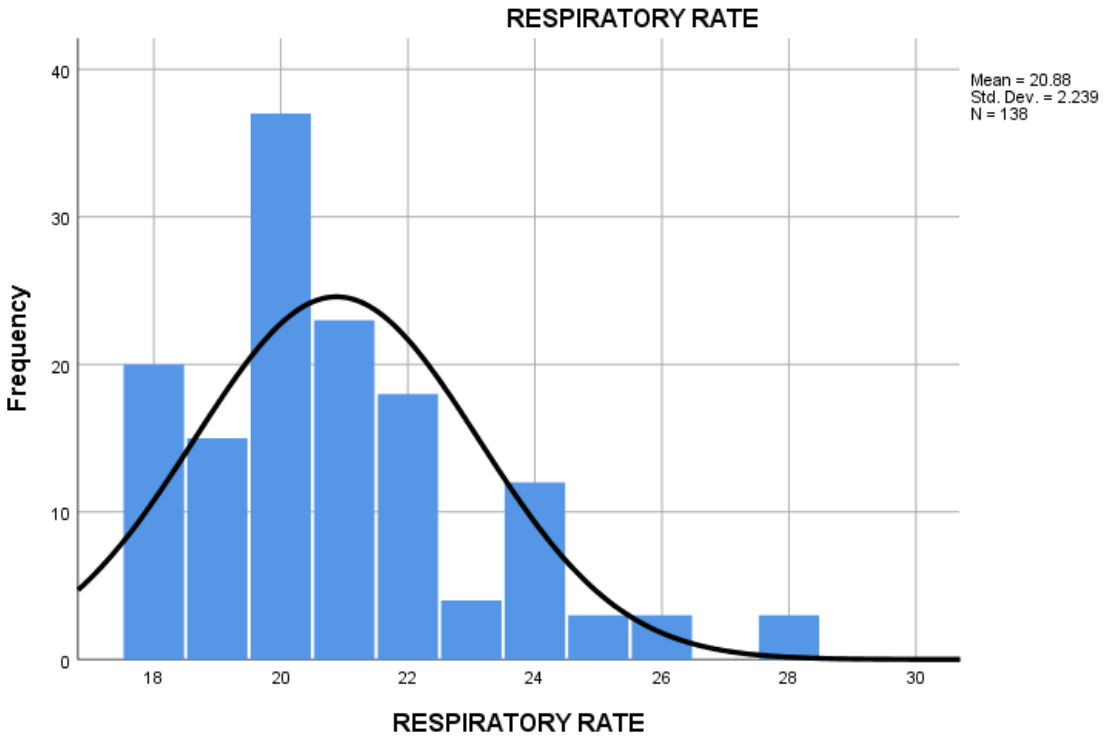


Figure 31. Histogram of Respiratory rate

The mean respiratory rate was 20.88 per minute. The Median respiratory rate was 20 per minute and the standard deviation was 2.239 per minute

XI A). RESPIRATORY RATE IN TRANEXAMIC ACID GROUP

Statistics

RESPIRATORY RATE

N	Valid	56
	Missing	0
Mean		22.23
Median		22.00
Mode		24
Std. Deviation		2.601

Table 27. Respiratory rate in Tranexamic acid group

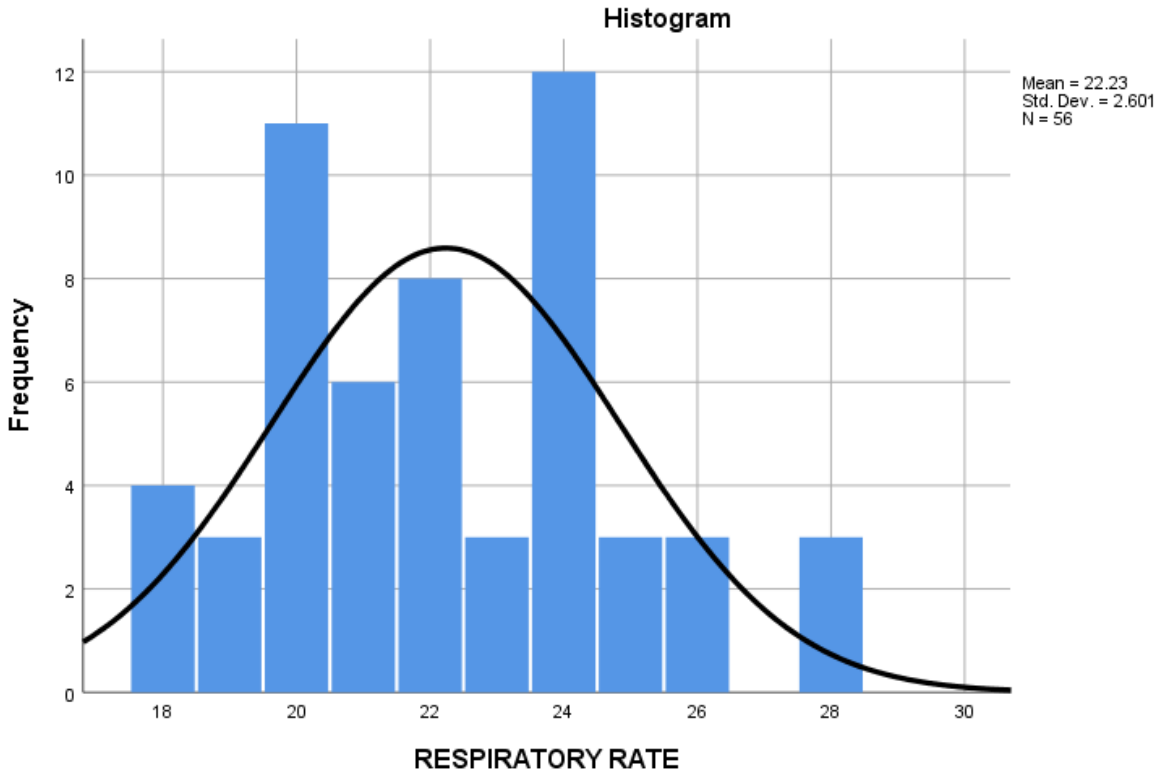


Figure 32. Histogram of Respiratory rate in Tranexamic acid group

The mean respiratory rate was 22.23 per minute. The Median respiratory rate was 22 per minute and the standard deviation was 2.601 per minute

XI B). RESPIRATORY RATE IN NON TRANEXAMIC ACID GROUP

Statistics

RESPIRATORY RATE

N	Valid	82
	Missing	0
Mean		19.95
Median		20.00
Std. Deviation		1.323

Table 28. Respiratory rate in non tranexamic acid group

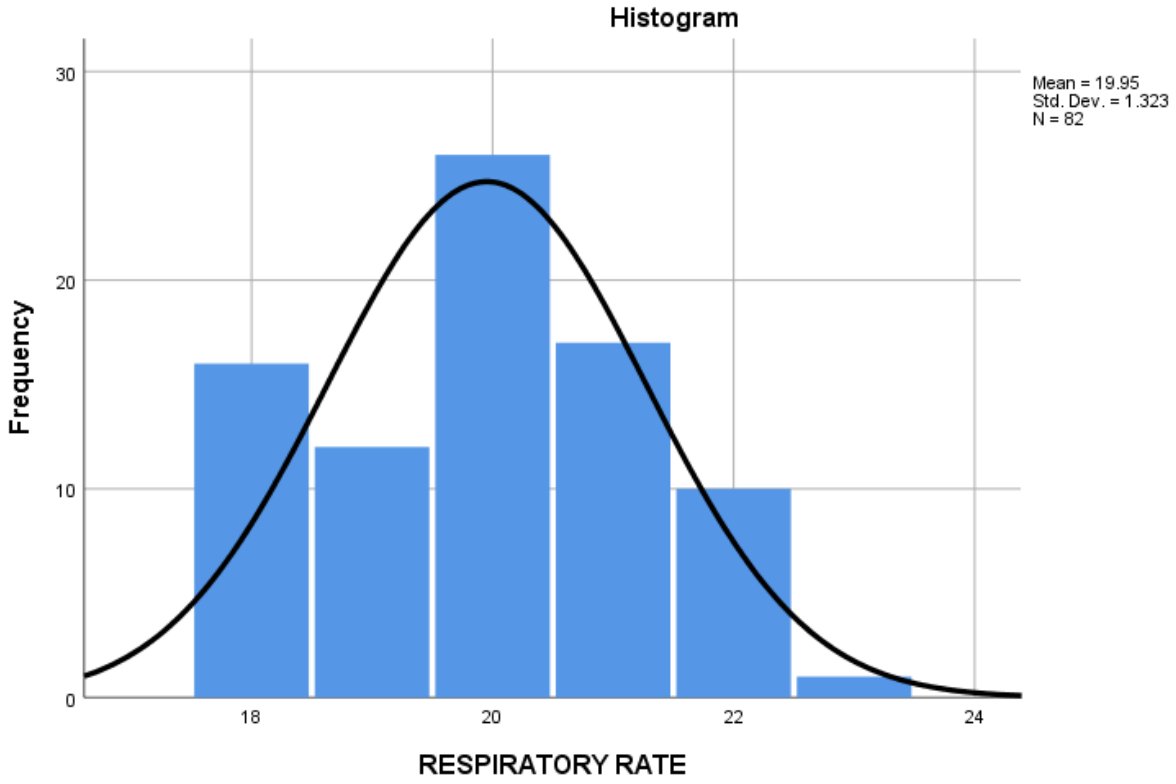


Figure 33. Histogram of Respiratory rate in non tranexamic acid group

The mean respiratory rate was 19.95 per minute. The Median respiratory rate was 20 per minute and the standard deviation was 1.323 per minute

XI C.) RESPIRATORY RATE ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
RESPIRATORY RATE	TRANEXAMIC ACID	56	22.2321	2.60064	.34752
	NONTRANEXAMIC ACID	82	19.9512	1.32313	.14612

Table 29. Descriptive statistics of Respiratory rate between the cohort groups

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
RESPIRATORY RATE	Equal variances assumed	34.448	.000	6.769	136	.000	2.28092	.33695	1.61459	2.94726
	Equal variances not assumed			6.050	74.581	.000	2.28092	.37699	1.52985	3.03200

Table 30. Independent Samples test of Respiratory rate between the cohort groups

The Tranexamic acid group was found to be significantly tachypneic during admission when compared to the non tranexamic acid group (**p< 0.05)**

XII) OXYGEN SATURATION

Statistics

		O2 SATURATION
N	Valid	138
	Missing	0
Mean		95.82
Median		96.00
Std. Deviation		2.065

Table 30. Oxygen saturation

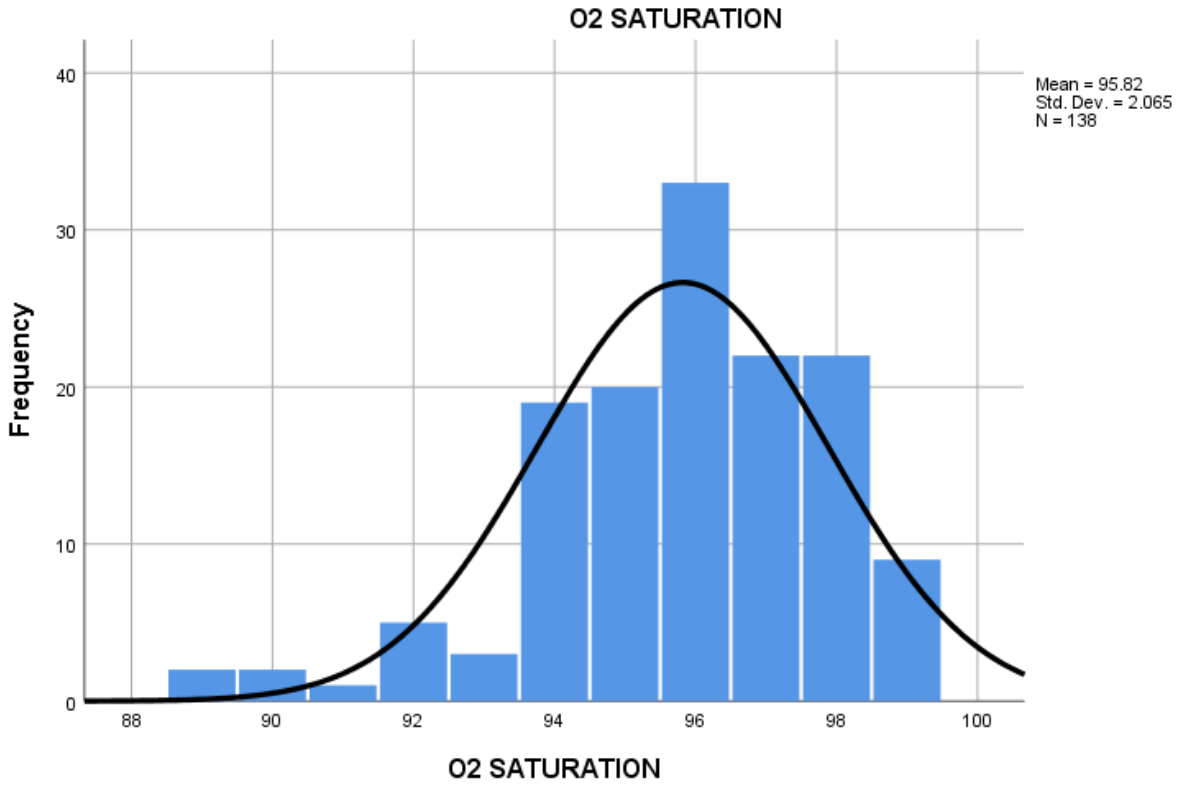


Figure 34. Histogram of Oxygen saturation

The mean Oxygen saturation was 95.82%. The Median saturation was 96%. The standard deviation was 2.06%.

XII A.)TRANEXAMIC ACID GROUP

Statistics

OXYGEN SATURATION

N	Valid	56
	Missing	0
Mean		94.84
Median		95.00
Mode		94
Std. Deviation		2.492

Table 31. Oxygen saturation in Tranexamic acid group

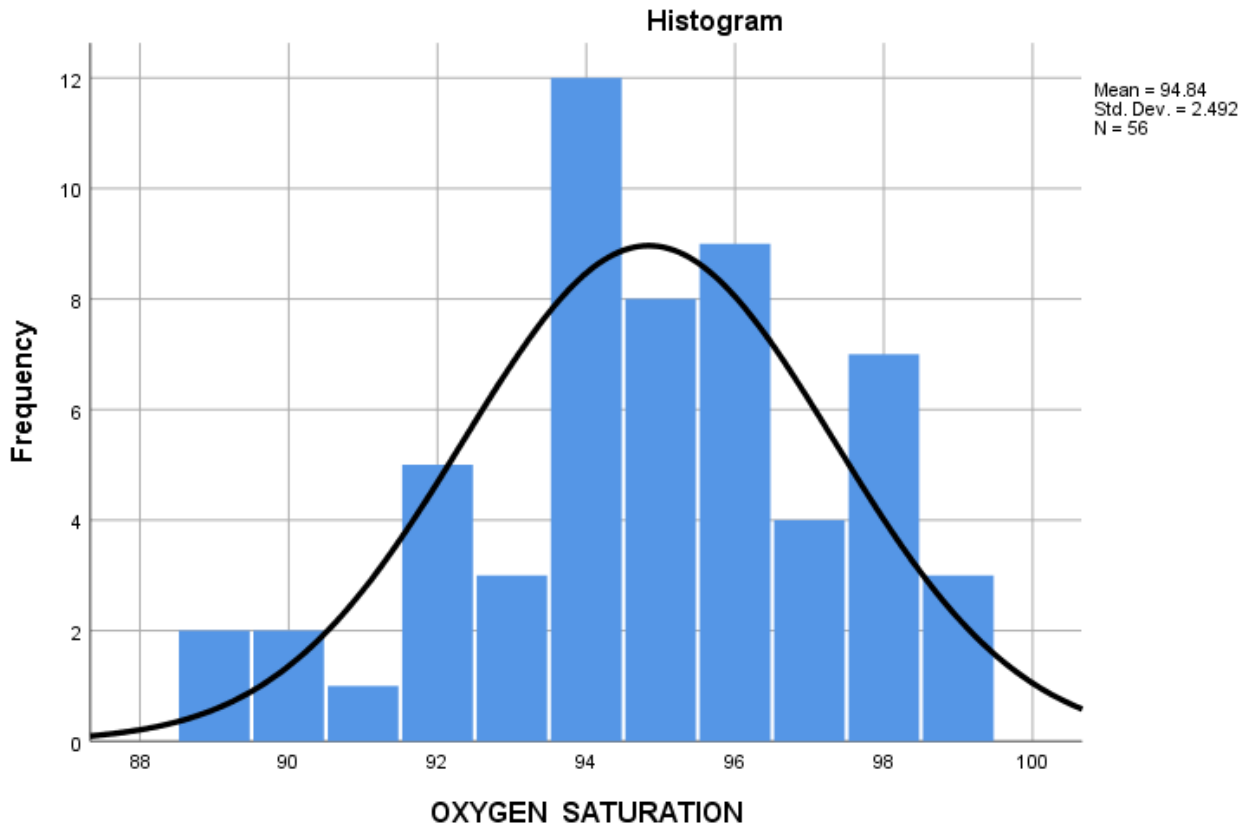


Figure 35. Histogram of Oxygen saturation in tranexamic acid group

The mean Oxygen saturation was 94.84%. The Median saturation was 95%. The standard deviation was 2.49%.

XII B.) OXYGEN SATURATION IN NON TRANEXAMIC ACID GROUP

Statistics

OXYGEN SATURATION

N	Valid	82
	Missing	0
Mean		96.49
Median		96.00
Std. Deviation		1.372

Table 32. Oxygen saturation in non tranexamic acid group

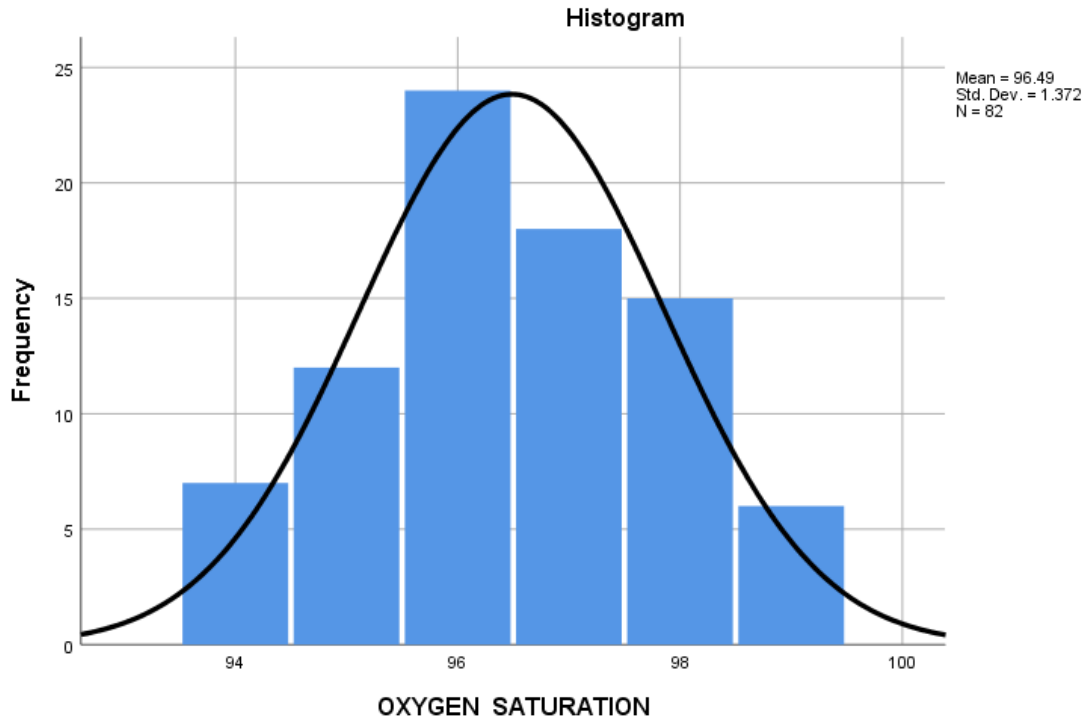


Figure 36. Histogram of Oxygen saturation in non tranexamic acid group

The mean Oxygen saturation was 96.49%. The Median saturation was 96%. The standard deviation was 1.37%.

XII. C) OXYGEN SATURATION ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
OXYGEN SATURATION	TRANEXAMIC ACID	56	94.8393	2.49200	.33301
	NONTRANEXAMIC ACID	82	96.4878	1.37207	.15152

Table 33. Descriptive statistics of Oxygen saturation between the cohort group

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
OXYGEN SATURATION	Equal variances assumed	17.759	.000	-4.989	136	.000	-1.64852	.33041	-2.30192	-.99512
	Equal variances not assumed			-4.506	77.864	.000	-1.64852	.36586	-2.37691	-.92013

Table 34. Independent Samples test of Oxygen saturation between cohort groups

The Tranexamic acid group was found to be slightly hypoxic when compared to the non tranexamic acid group which was found to be statistically significant ($p < 0.05$)

XIII.) INTRAVENOUS FLUDS

Statistics

		INTRAVENOUS FLUIDS
N	Valid	138
	Missing	0
Mean		963.84
Median		950.00
Std. Deviation		253.358

Table 35. Intravenous fluids infused

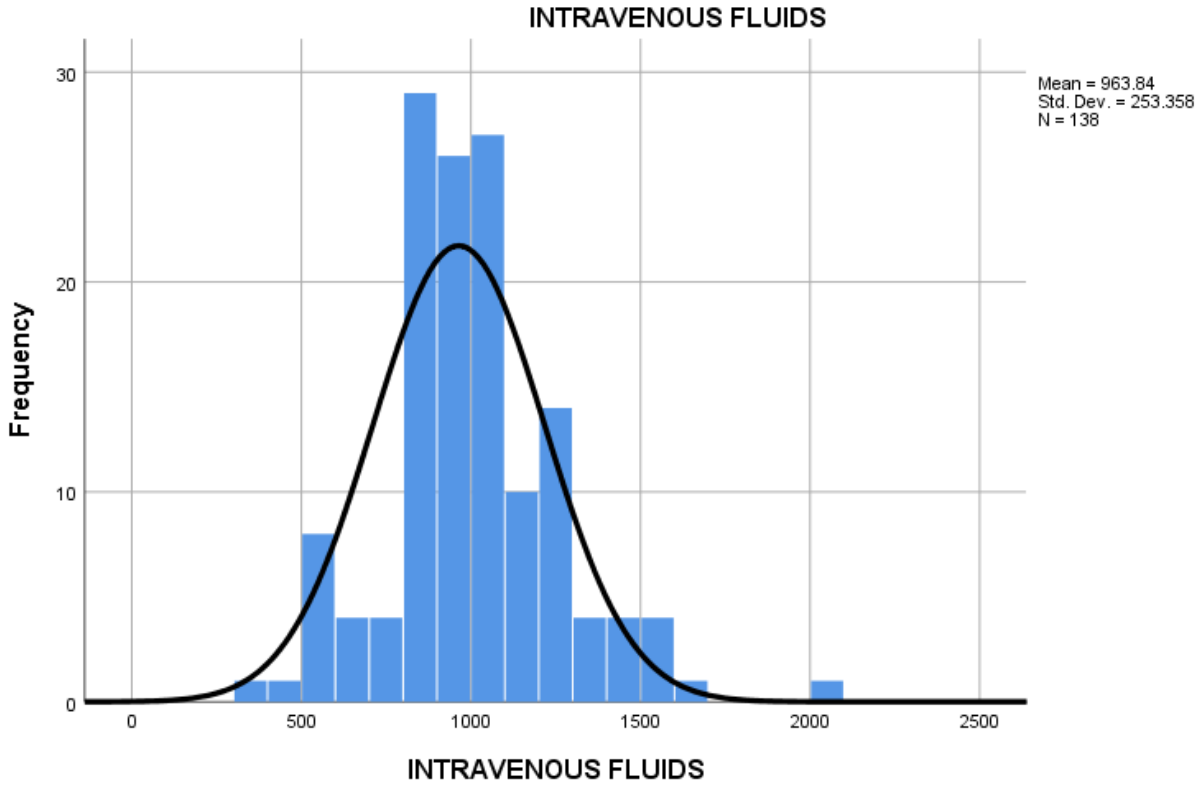


Figure 37. Histogram of intravenous fluids infused

The mean intravenous fluids infused was 963.84 ml. The Median was 950 ml and the standard deviation was 253.36ml

XIII A.) INTRAVENOUS FLUIDS INFUSED IN TRANEXAMIC ACID GROUP

Statistics

INTRAVENOUS FLUIDS

N	Valid	56
	Missing	0
Mean		1089.29
Median		1050.00
Mode		1000
Std. Deviation		288.840

Table 36. Intravenous fluids infused in tranexamic acid group

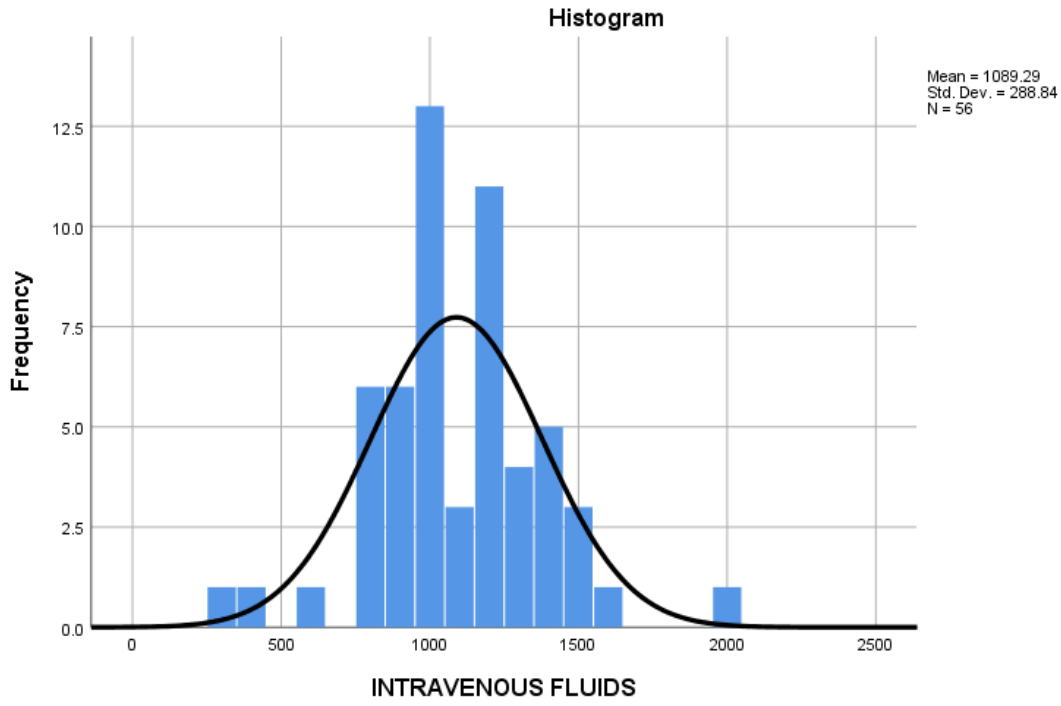


Figure 38. Histogram of intravenous fluids infused in tranexamic acid group

The mean intravenous fluids infused was 1089.29ml The Median was 1050 ml and the standard deviation was 288.84ml.

XIII B.) INTRAVENOUS FLUIDS INFUSED IN NON TRANEXAMIC ACID GROUP

Statistics

INTRAVENOUS FLUIDS

N	Valid	82
	Missing	0
Mean		878.17
Median		900.00
Std. Deviation		183.331

Table 37. Intravenous fluids infused in non tranexamic acid group

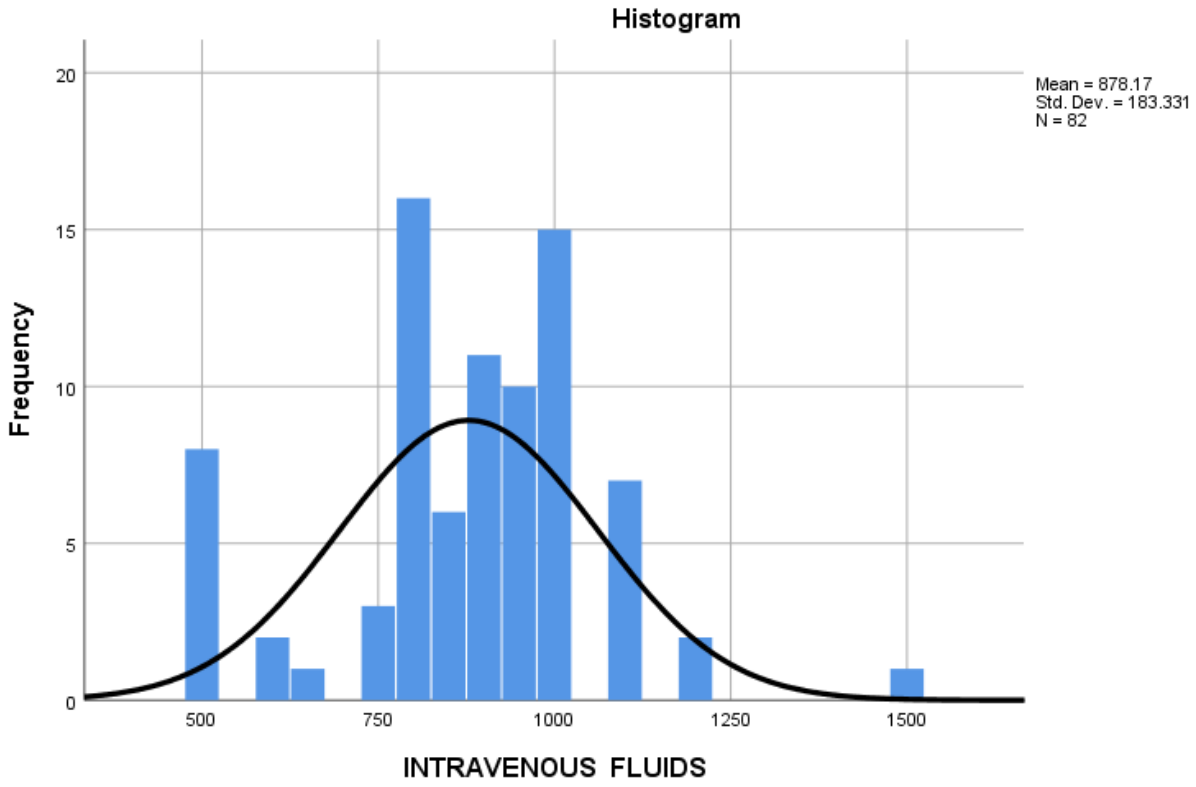


Figure 39. Histogram of intravenous fluids infused in non tranexamic acid group

The mean intravenous fluids infused was 878.17ml The Median was 900 ml and the standard deviation was 183.33ml.

XIII C.) INTRAVENOUS FLUIDS INFUSED ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
INTRAVENOUS FLUIDS	TRANEXAMIC ACID	56	1089.2857	288.84004	38.59787
	NONTRANEXAMIC ACID	82	878.1707	183.33083	20.24550

Table 38. Descriptive statistics of intravenous fluids infused. between the cohort group

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
INTRAVENOUS FLUIDS	Equal variances assumed	10.672	.001	5.252	136	.000	211.11498	40.19365	131.62960	290.60037
	Equal variances not assumed			4.844	85.055	.000	211.11498	43.58528	124.45659	297.77337

Table. 39. Independent Samples test of Oxygen saturation between cohort groups

The Tranexamic acid group was found to have higher intravenous fluids requirement when compared to the non tranexamic acid group(**p <0.05)**

XIV) TOTAL TRANSFUSIONS

Statistics

		TOTAL TRANSFUSION
N	Valid	138
	Missing	0
Mean		.80
Median		1.00
Std. Deviation		.945

Table 40. Total transfusions

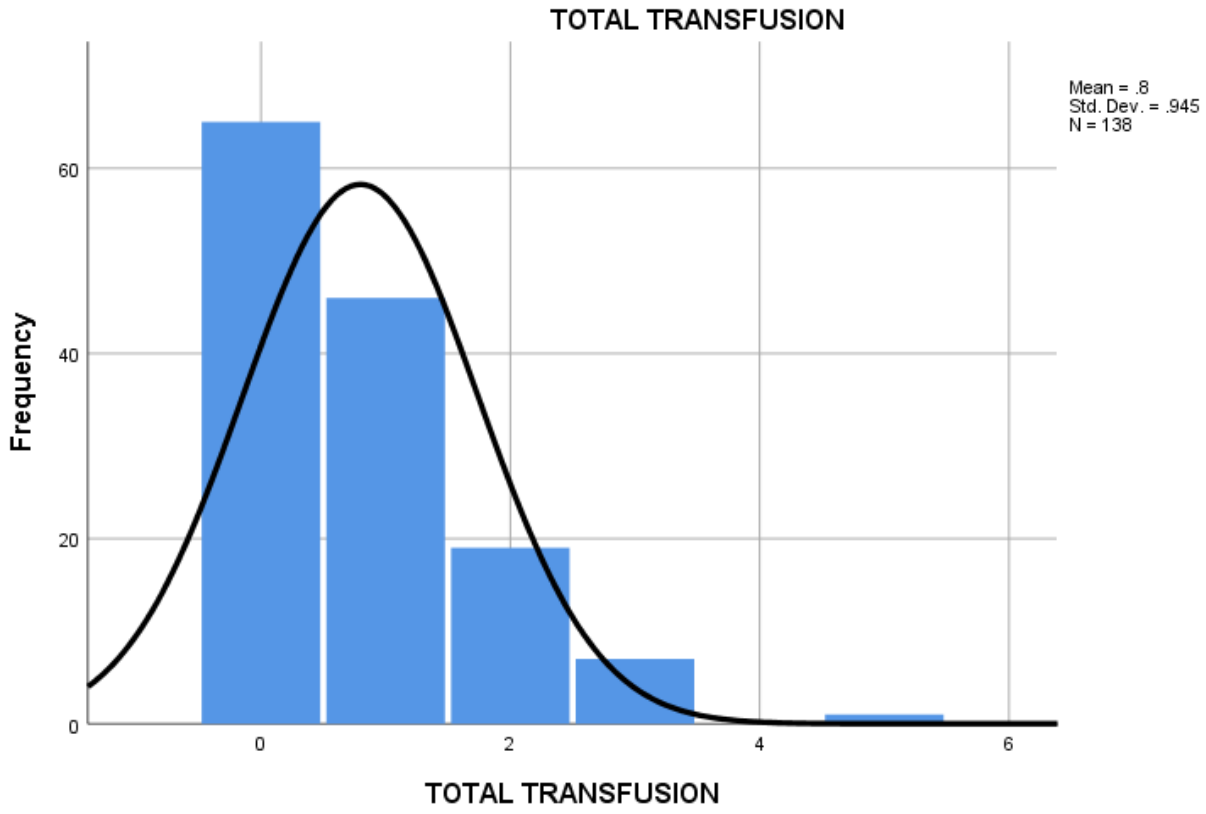


Figure 40. Histogram of total transfusions

The mean number of Transfusion was 0.8. The Median transfusions was 1 and the standard deviation was 0.945

XIV A.) TRANSFUSIONS IN TRANEXAMIC ACID GROUP

Statistics

TOTAL TRANSFUSION

N	Valid	56
	Missing	0
Mean		1.55
Median		1.00
Mode		1
Std. Deviation		.807

Table 41.Transfusions in tranexamic acid group

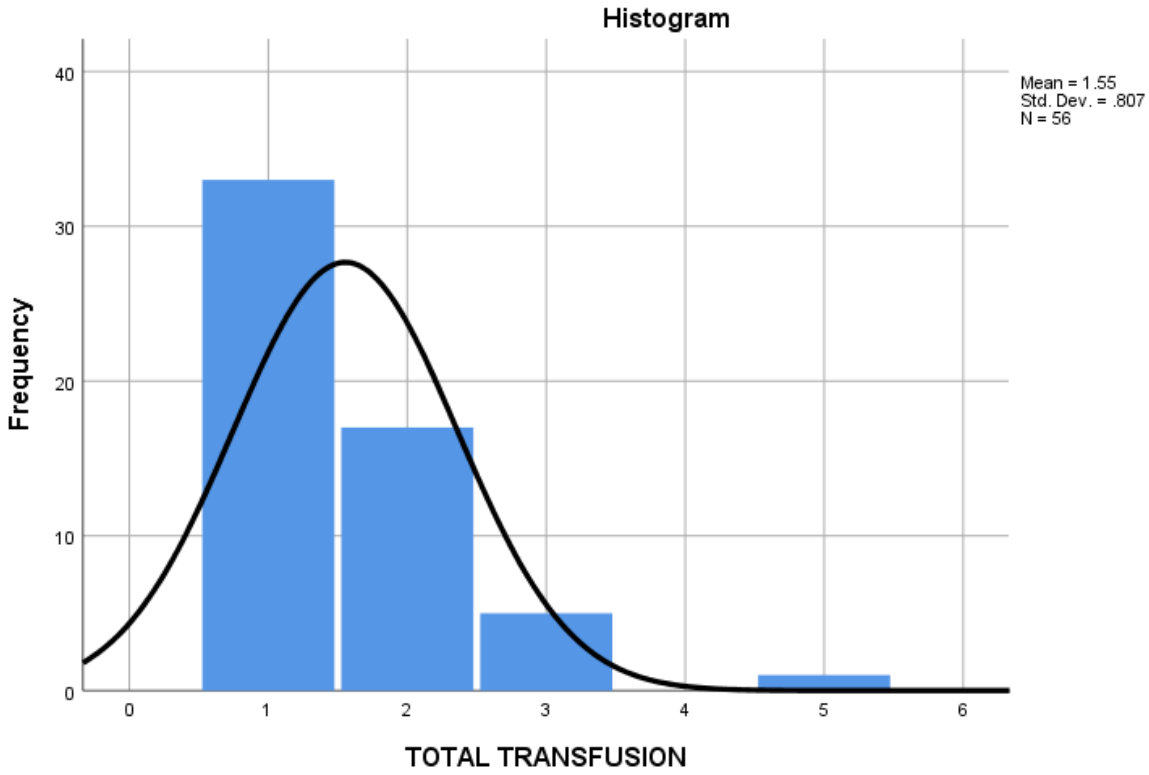


Figure 41. Histogram of total transfusions

The mean number of transfusions in the tranexamic was 1.55. The Median was 1 and the standard deviation was 0.807.

XIV B.) TRANSFUSIONS IN NON TRANEXAMIC ACID GROUP

Statistics

TOTAL TRANSFUSION

N	Valid	82
	Missing	0
Mean		.28
Median		.00
Std. Deviation		.634

Table 42. Transfusions in non tranexamic acid group

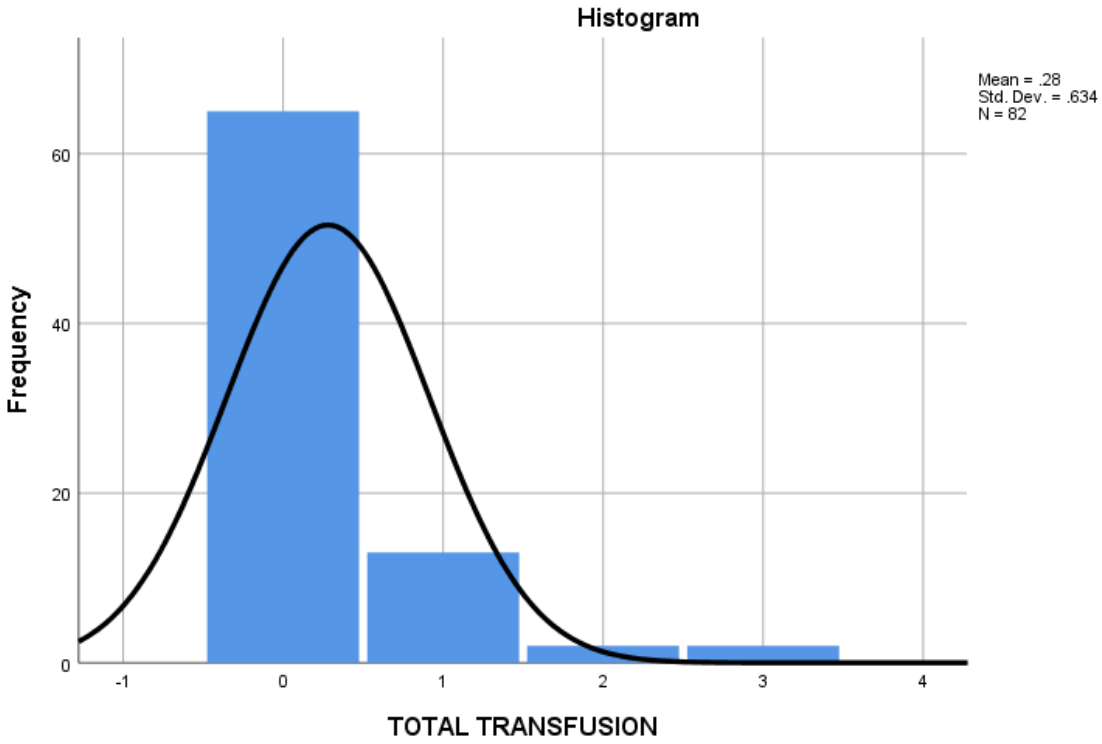


Figure 41. Histogram of transfusions in non tranexamic acid group

The mean number of transfusions in the tranexamic was 0.28. The Median was 0 and the standard deviation was 0.63

XIV C.) TRANSFUSIONS ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
TOTAL TRANSFUSION	TRANEXAMIC ACID	56	1.5536	.80723	.10787
	NONTRANEXAMIC ACID	82	.2805	.63391	.07000

Table 43 Descriptive statistics of transfusions between the cohort group

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
TOTAL TRANSFUSION	Equal variances assumed	6.893	.010	10.356	136	.000	1.27308	.12293	1.02998	1.51619
	Equal variances not assumed			9.900	99.141	.000	1.27308	.12859	1.01793	1.52824

Table. 44 Independent Samples test of transformation transfusions between cohort groups

There was a significant requirement of transfusions in the tranexamic acid group when compared to the non tranexamic acid group (**$p<0.05$**)

XV) CRITICAL CARE LENGTH OF STAY

Statistics

		CRITICAL CARE STAY
N	Valid	138
	Missing	0
Mean		3.87
Median		4.00
Std. Deviation		.739

Table 45. Critical care length of stay

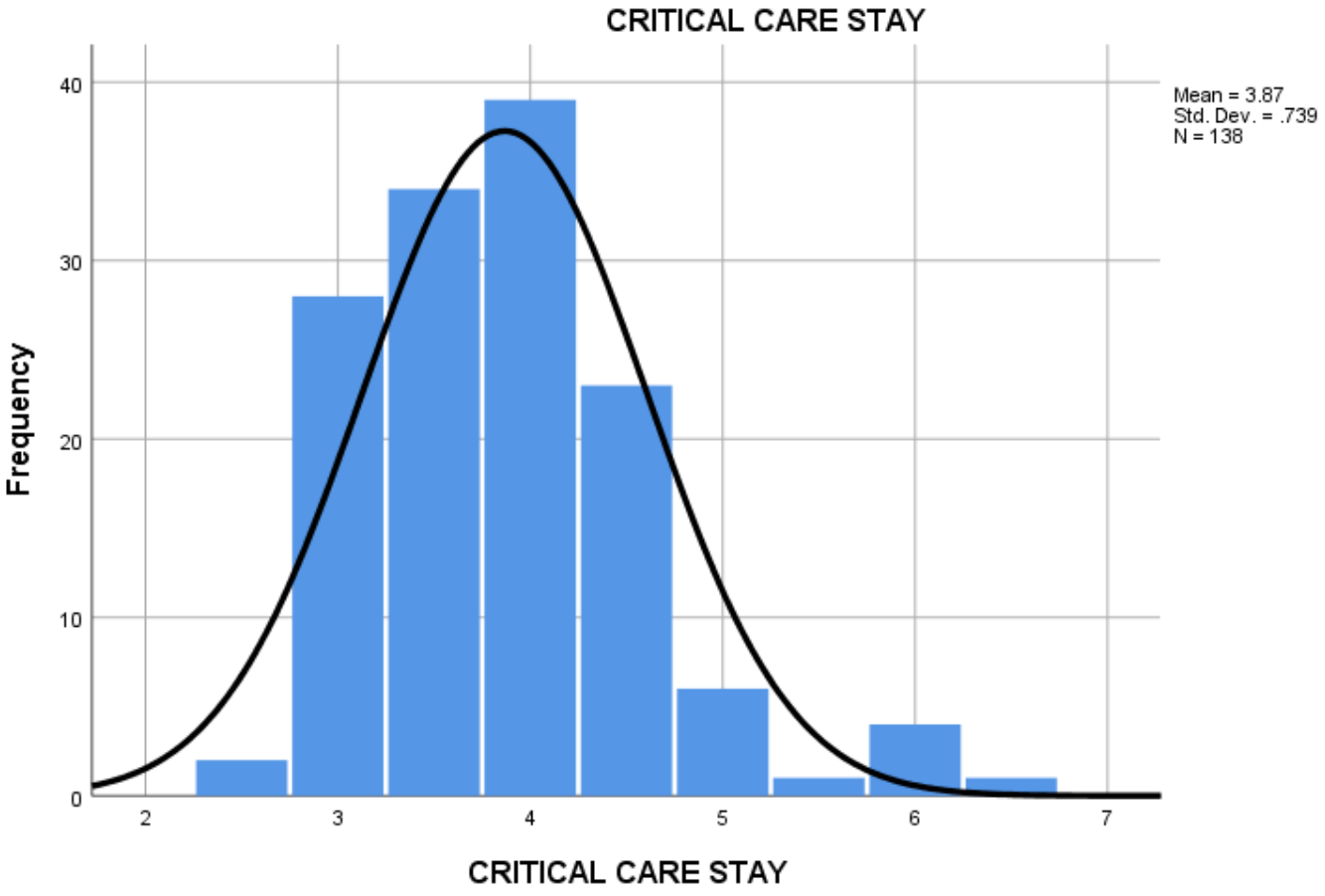


Figure 42. Histogram of critical care length of stay

The mean length of stay in the emergency department was 3.87 hours. The Median was 4 **hours** and the standard deviation was 0.739 hours

XV A) CRITICAL CARE LENGTH OF STAY IN TRANEXAMIC ACID GROUP

Statistics

CRITICAL CARE STAY

N	Valid	56
	Missing	0
Mean		3.99
Median		4.00
Mode		4
Std. Deviation		.806

Table 47. Critical care length of stay in tranexamic acid group

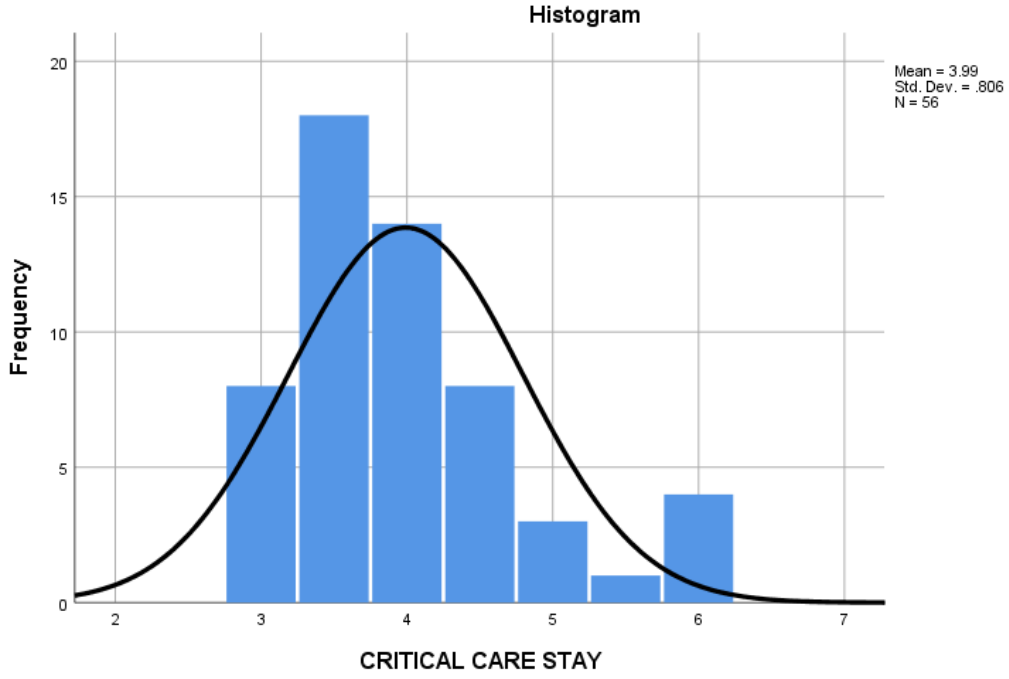


Figure 43. Histogram of Tranexamic group critical care length of stay

The mean length of stay in the emergency department was 3.99 hours. The Median was 4 **hours** and the standard deviation was 0.806 hours

XV B) CRITICAL CARE LENGTH OF STAY IN NON TRANEXAMIC ACID GROUP

Statistics

CRITICAL CARE STAY

N	Valid	82
	Missing	0
Mean		3.78
Median		4.00
Std. Deviation		.681

Table 48 Critical care length of stay in non tranexamic acid group

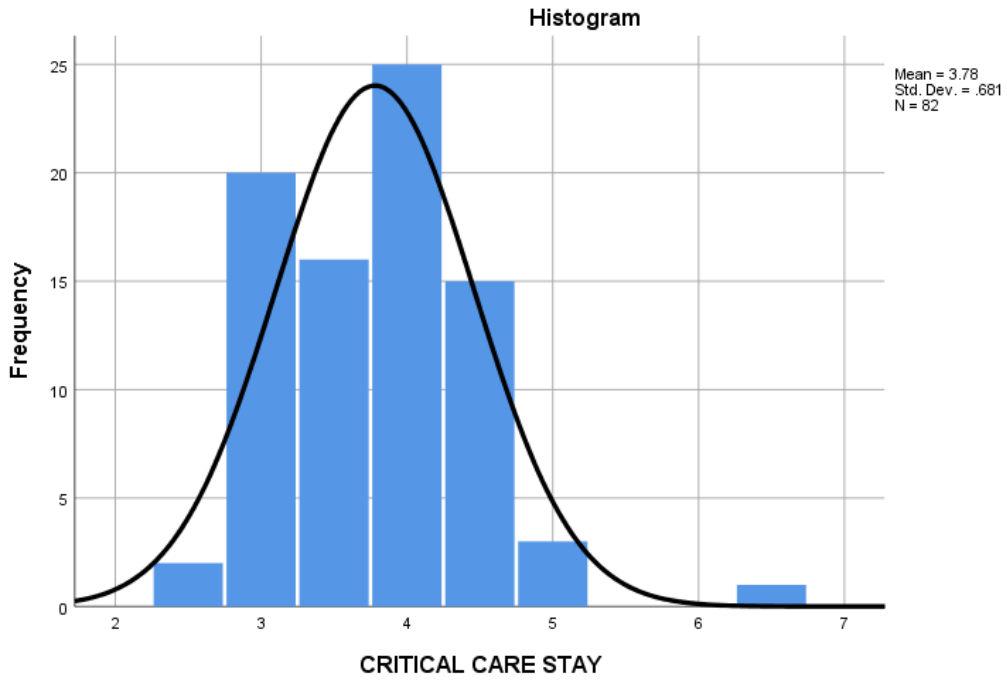


Figure 44. Histogram of critical care length of stay in non tranexamic acid group

The mean length of stay in the emergency department was 3.78 hours. The Median was 4 **hours** and the standard deviation was 0.681 hours

XV C.) CRITICAL CARE STAY DURATION ANALYSIS BY UNPAIRED T TEST

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
CRITICAL CARE STAY	TRANEXAMIC ACID	56	3.9911	.80618	.10773
	NONTRANEXAMIC ACID	82	3.7805	.68086	.07519

Table 49 Descriptive statistics of critical care stay duration between the cohort group

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
CRITICAL CARE STAY	Equal variances assumed	.343	.559	1.655	136	.100	.21058	.12726	-.04109	.46226
	Equal variances not assumed			1.603	104.755	.112	.21058	.13137	-.04991	.47108

Table 50 Independent Samples test of critical care stay between cohort groups

Compared with the non tranexamic acid group, there was no significant difference between the critical care stay duration of the tranexamic acid group **(p>0.05),** indicating that though the tranexamic cohort was more severely injured the critical care requirement is shortened.

SUMMARY OF RESULTS

1. The most common mode of injury was Road Traffic Accidents(RTA) contributing to 89 cases(64.5%) , followed by assault with 21 cases(15.2%). There were 12 cases(8.7%) due to accidental injury and 12 cases(8.7%) due to accidental fall. 3 cases were due to self inflicted injury (2.2%) and 1 case due to Train Traffic Accident(0.7%)
2. The most common mode of injury in tranexamic group-was due to penetrating trauma with 34 cases(60.7%) followed by blunt trauma with 22 cases (39.3%).
3. The average duration from time of injury to administration of loading dose tranexamic acid was **124 minutes**.
4. The Mean Age was **38.49 years.** The Median Age was **36.50 years** . Trauma was found to be most commonly affecting the middle aged group.
5. Tranexamic acid group cases had more significant severity of injury (**$p<0.05$**) when compared to the non tranexamic acid group
6. The Tranexamic acid group patients had significantly tachycardia ($p<0.05$) owing to the volume low due to active bleeding and increased acuity of injury.
7. The Tranexamic acid group was found to be significantly tachypneic during admission when compared to the non tranexamic acid group (**$p< 0.05$)**
8. The Tranexamic acid group was found to be slightly hypoxic when compared to the non tranexamic acid group which was found to be statistically significant ($p <0.05$)

9. The Tranexamic acid group was found to have higher intravenous fluids requirement when compared to the non tranexamic acid group(**p <0.05)**
10. There was a significant requirement of transfusions in the tranexamic acid group when compared to the non tranexamic acid group **(p<0.05)**
11. Compared with the non tranexamic acid group, there was no significant difference between the critical care stay duration of the tranexamic acid group **(p>0.05),** indicating that though the tranexamic cohort was more severely injured the critical care requirement is shortened.
12. No adverse drug reactions were observed during the period of study
13. No mortality was observed during the period of study

DISCUSSION

What do the studies demonstrate?

CRASH-2 was a randomized and prospective study. It took place in 247 hospitals throughout 40 countries, with a large number of patients (N=20,211). The primary outcome measured was death at four weeks with intention to treat. The investigators found, that all causes of mortality decreased by 10% (RR 0.91, 95% CI 0.85-0.97). Furthermore, the risk of death from bleeding decreased by 15% (RR 0.85, 95% CI 0.76-0.96). The data also noted that there was increased mortality when the initial dose of Tranexamic acid was given past three hours.

MATTERs was a retrospective, observational trial that compared Tranexamic acid administration with non-Tranexamic acid administration in combat casualties receiving at least 1 unit blood. They also evaluated a subset of patients receiving a massive transfusion. There were 896 consecutive patients with a combat wound, of which 293 received Tranexamic acid. Their findings in the Tranexamic acid group demonstrated a lower unadjusted mortality (17.4% vs. 23.9%; p=0.03); benefit greater in patients receiving Massive transfusion (14.4% vs. 28.1%; p=0.04) Their conclusion said Tranexamic acid was independently associated with survival (OR=7.7228; CI 3.016-17.322).

Military use of tranexamic acid in combat trauma: Does it matter? was a much larger retrospective study from Afghanistan over a 3-year, 6-month period

from October 1, 2010, to March 31, 2014. It included combat wounded, those admitted to a Role 3, and received at least one unit of blood or blood component. The overall patient population was 3,773. For results purposes, the casualties groups, 1. patients requiring a massive transfusion (N = 784), 2. a propensity score-matched sample (N = 1,030), and 3. NATO members (N=1262). Of those casualties receiving a massive transfusion (HR, 0.84; 95% CI, 0.46–1.56; p = 0.51), the propensity-matched sample (HR, 0.68; 95% CI, 0.27–1.73; p = 0.34), or US/NATO military sample (HR, 0.76; 95% CI, 0.30–1.92; p = 0.48) did not indicate a significant association between Tranexamic acid administration and mortality. The use of Tranexamic acid was associated with increased risk of pulmonary embolism in the total sample, massive transfusion sample, and the NATO military sample. Tranexamic acid was also associated with increased risk of deep vein thrombosis in the total sample and the NATO military sample.

Prehospital Administration of Tranexamic Acid by Ground Forces in Afghanistan: The Prehospital Trauma Registry Experience is the only study from the Tactical Combat Casualty Care perspective. This retrospective review from January 2013 to September 2014, there were 272 patients who met inclusion criteria. Most injuries (97.8%; n = 266) were battle injuries. Of the 111 patients who met criteria to receive prehospital Tranexamic Acid only 51 (18.8%) received Tranexamic Acid, the remaining 221 (81.2%) did not.

There was a sub-analysis of four interventions,

1. Hemostatic dressing

2. Pressure dressing

3. Tourniquet and

4. IV fluids.

Though the numbers were small, there was a significant difference in Tranexamic Acid administration versus not receiving Tranexamic Acid for casualties with hemostatic dressings, pressure dressings, and tourniquet placement. Oddly, the proportion of patients receiving IV fluids was higher among the non-Tranexamic group.

The mechanism of Tranexamic Acid in the trauma has been debated. A sub-analysis of the CRASH-2 trial suggested Tranexamic acid's impact is due to anti-fibrinolytic effects, whereas results from the MATTERs trial supported an anti-inflammatory effect mechanism.

There are physicians and scientists who believe Tranexamic acid may not beneficial to everyone and in some cases, maybe harmful. This issue may have to do with certain types of gene expression and/or the injury pattern. Some studies recommend holding off on Tranexamic acid administration until they arrive at the hospital and can conduct the proper lab work. As previously mentioned, giving Tranexamic acid after three hours is harmful, there is no debate about that claim.

This prospective study to evaluate the use of tranexamic acid as a part of massive haemorrhage protocol in severely injured trauma patients over a period of one year in the

emergency department of Stanley Medical College, Chennai. A total of 138 cases were evaluated. 56 cases received Tranexamic acid and 82 cases were grouped into the non tranexamic acid group.

The results of the study show that the cohort who received tranexamic acid were more severely injured when compared to the non tranexamic acid group. They also present with significant tachycardia, tachypnea and lower oxygen saturation. These can be attributed to the increased acuity of injuries in the tranexamic acid cohort. Despite the severity, the Tranexamic acid group had comparable duration of critical care stay when compared to the non tranexamic acid group.

The study was limited to only a single center experience. Larger multi centre studies may be necessary to evaluate the efficacy and side effects of use of tranexamic acid in trauma as well as pretrauma setup.

For now, tranexamic acid may be useful to improve the survival of actively bleeding severely injured trauma patients.

CONCLUSION

1) Tranexamic acid can be used as a part of massive haemorrhage protocol in a trauma care setup with shorter duration of critical care stay and improved survival.
2) Tranexemic acid administration is not associated with any adverse drug reactions.
3) There is no risk of thrombosis due to use of tranexamic acid within 3hrs of trauma in severely injured trauma patients.

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

DISSERTATION TOPIC:

"TRANEXAMIC ACID USE IN SEVERELY INJURED PATIENTS WITH SIGNIFICANT HAEMORRHAGE- A PROSPECTIVE COHORT STUDY"

PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI
NAME AND ADDRESS OF PATIENT:

I, _____ have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.
I understand that I will not get any payment for taking part in this study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Volunteer:

Signature/Thumb impression of the Volunteer
Date:

Witnesses:
(Signature, Name & Address)
Date:

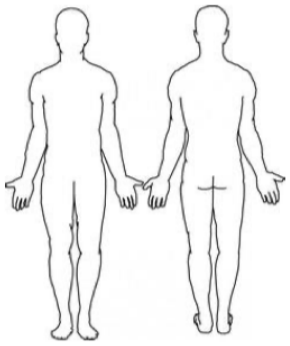
Name and signature of investigator: (Dr. PRAVEEN KUMAR.S):

PROFORMA

Name: Age: Sex:
Date of Injury : Time of Injury:
Date of Admission: Time of Admission:
Mode of Injury:

Mechanism of Injury: Blunt/Penetrating
Active Bleeding : Yes/ No

List of Injuries:



Injury Severity Score:

AIS Score	Injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical
6	Maximum

Region	Injury Description	AIS	Square top three
Head & Neck			
Face			
Chest			
Abdomen			
Extremity			
External			
TOTAL(Injury Severity Score)			

Glasgow Coma Score: E( ) V( ) M( )= /15

VITALS:

BP: PR: RR: SPO2:

Tranexamic acid 1g iv bolus followed by 1g infusion over 8hrs administered : Yes/No

Total intravenous fluids transfused(ml):

INVESTIGATIONS: **Details of Transfusions given:**

Hb		B.Sugar		T.B		Whole Blood(units)	
PCV		Urea		D.B		PRBC(units)	
WBC		Creatinine		OT		Platelets(units)	
Platelets				PT		FFP(units)	
INR				ALP		Cryoprecipitate(units)	

RADIOLOGICAL INVESTIGATION FINDINGS

DIAGNOSIS :

ACTIVE SURGICAL INTERVENTION : Yes / No
If Yes , Procedure done with date , time :
TIME TO ACTIVE SURGICAL INTERVENTION :
ANY MORTALITY : Yes/No
CAUSE OF MORTALITY :

DATE AND TIME OF DISCHARGE/EXPIRY : DURATION OF HOSPITAL STAY:
DURATION OF CRITICAL CARE STAY :

MASTER CHART

S.NO	ID	AGE	SEX	DURATION (minutes)	MODE	TYPE OF INJURY	GCS	ISS	ACTIVE BLEED	SBP	PR	RR	SPO2	TRANEX (A-YES B-NO)	IVF INFUSED	INITIAL HB	WB	PRBC	PLATELET	FFP	CRYO	TOTAL TRANSF	POST TRANS HB(24 HR)	MORTALITY	DURATION OF STAY IN ED
1	TX001	34	M	120	RTA	BLUNT	13	20	YES	100	90	20	94	A	300	10	1	2				3	12.5	NO	6
2	TX002	26	M	100	ASSAULT	PENETRATING	14	16	YES	90	104	22	93	A	400	12.5		1				1	13.2	NO	3.5
3	TX003	30	M	168	SELF INFLICTED INJURY	PENETRATING	15	16	YES	100	96	20	96	A	600	12.6		1				1	12.4	NO	3
4	TX004	36	M	120	ACCIDENTAL FALL	BLUNT	13	17	NO	100	98	22	96	B	500	10		2				2	11.2	NO	6.5
5	TX005	42	M	170	RTA	BLUNT	15	15	YES	110	92	20	94	A	1000	11		2				2	12.4	NO	5
6	TX006	64	F	160	ACCIDENTAL FALL	BLUNT	15	15	YES	110	94	20	95	A	800	10.4		1				1	11.2	NO	4
7	TX007	29	F	100	ASSAULT	PENETRATING	15	17	YES	90	102	22	94	A	1000	10.2		1		1		2	10.8	NO	3
8	TX008	27	M	146	RTA	BLUNT	14	19	YES	100	96	21	94	A	1000	11.4		2				2	12.6	NO	5
9	TX009	38	F	110	ACCIDENTAL INJURY	PENETRATING	15	15	YES	102	96	20	96	A	1000	10.4		2				2	11.5	NO	5.5
10	TX010	46	M	146	RTA	BLUNT	15	17	NO	120	88	19	96	B	500	12.4						0	12.3	NO	3
11	TX011	52	F	98	ACCIDENTAL INJURY	PENETRATING	14	15	YES	112	90	22	94	A	850	11.6		1				1	12.1	NO	3
12	TX012	56	M	90	ACCIDENTAL FALL	BLUNT	15	15	NO	100	90	20	96	B	1000	11.4		1				1	12.1	NO	3
13	TX013	18	M	86	RTA	BLUNT	15	16	NO	100	86	20	97	B	500	12.4						0	12.1	NO	3
14	TX014	25	M	104	RTA	BLUNT	15	17	YES	100	94	22	95	A	800	12		1				1	11.8	NO	3
15	TX015	42	M	104	RTA	BLUNT	14	15	NO	110	94	18	94	B	800	11.2		1				1	11	NO	3.5
16	TX016	25	F	86	RTA	BLUNT	15	21	YES	90	108	20	96	A	1500	10.8		2				2	11	NO	4.5

17	TX017	54	M	110	RTA	BLUNT	15	15	NO	120	88	18	96	B	500	12.4						0	12.3	NO	3
18	TX018	42	F	130	ACCIDENTAL INJURY	PENETRATING	15	22	YES	100	96	18	98	A	1500	10.7		1		1		2	11.4	NO	3.5
19	TX019	37	M	86	RTA	BLUNT	15	15	NO	110	90	20	94	B	800	13.2						0	13	NO	3
20	TX020	32	M	110	RTA	BLUNT	11	15	NO	100	96	21	95	B	1000	11.8						0	11.6	NO	3
21	TX021	46	M	80	RTA	BLUNT	14	18	YES	100	94	21	94	A	1200	12		1				1	12.3	NO	4.5
22	TX022	28	M	130	ACCIDENTAL FALL	BLUNT	13	16	NO	108	96	22	97	B	950	12.5						0	12.3	NO	3.5
23	TX023	24	M	127	RTA	BLUNT	12	21	YES	92	106	24	92	A	1200	11.4		2				2	12.2	NO	3.5
24	TX024	70	F	120	RTA	PENETRATING	14	17	YES	110	98	19	97	A	1200	12.8		1				1	12.7	NO	3
25	TX025	32	M	90	RTA	BLUNT	12	15	NO	98	108	22	97	B	1100	13						0	13	NO	3
26	TX026	28	F	160	ACCIDENTAL INJURY	PENETRATING	15	20	YES	96	116	21	96	A	1500	11.8		2				2	12.2	NO	4.5
27	TX027	21	F	120	SELF INFLICTED INJURY	PENETRATING	15	16	YES	100	98	19	99	A	900	12.4		1				1	12.8	NO	3.5
28	TX028	21	F	160	RTA	PENETRATING	12	21	YES	88	120	24	94	A	1200	11.5		1				1	12	NO	4
29	TX029	46	F	68	RTA	BLUNT	16	20	YES	90	116	25	92	A	1000	11.4		2				2	12.2	NO	4.5
30	TX030	48	M	120	RTA	BLUNT	13	18	YES	100	94	21	96	A	900	11.8		1				1	12.5	NO	3
31	TX031	34	F	126	ASSAULT	BLUNT	10	21	YES	86	124	24	94	A	1100	11.6		1				1	11.8	NO	4
32	TX032	56	M	100	ACCIDENTAL INJURY	PENETRATING	15	16	NO	96	110	20	95	B	1000	13.2						0	12.9	NO	4
33	TX033	35	F	84	ASSAULT	PENETRATING	15	21	YES	96	121	26	92	A	1250	11.6		1				1	11.8	NO	4
34	TX034	36	M	140	RTA	BLUNT	14	18	NO	94	114	20	97	B	950	12.4						0	12	NO	4.5
35	TX035	37	F	156	RTA	BLUNT	15	24	YES	90	113	24	95	A	1300	9.6		2		1		3	11.5	NO	6
36	TX036	65	F	150	RTA	PENETRATING	16	18	YES	106	98	20	97	A	1100	10.6		1		1		2	12	NO	3.5
37	TX037	37	M	160	TTA	PENETRATING	15	27	YES	86	120	26	89	A	2000	9.6		2	1	2		5	11.4	NO	6
38	TX038	49	M	90	RTA	BLUNT	12	15	NO	110	88	19	95	B	950	12.6						0	12.4	NO	3.5
39	TX039	22	F	134	ASSAULT	BLUNT	15	20	YES	88	120	23	92	A	1300	10.8		2				2	11.3	NO	4
40	TX040	58	M	76	ASSAULT	BLUNT	11	21	YES	84	116	24	94	A	1600	11.8		1				1	12.2	NO	4

41	TX041	36	F	170	RTA	PENETRATING	12	19	YES	92	106	22	94	A	1000	11.4		1				1	11.7	NO	4
42	TX042	66	M	84	ACCIDENTAL FALL	BLUNT	13	15	NO	108	86	20	95	B	1000	12.4						0	12	NO	3
43	TX043	26	M	74	RTA	BLUNT	13	16	NO	112	84	21	94	B	960	12.2						0	11.8	NO	3.5
44	TX044	28	F	140	ASSAULT	PENETRATING	14	18	YES	100	98	22	97	A	800	11.2		1				1	11.5	NO	3.5
45	TX045	34	F	126	RTA	PENETRATING	15	24	YES	80	122	28	94	A	1400	9.6		2		1		3	11.7	NO	6
46	TX046	36	M	80	RTA	PENETRATING	12	20	YES	96	104	21	98	A	1200	11.6		1				1	12	NO	3.5
47	TX047	52	M	148	ASSAULT	BLUNT	14	18	NO	114	90	20	96	B	800	13.1						0	12.6	NO	4.5
48	TX048	34	M	144	ASSAULT	BLUNT	15	16	NO	110	84	19	99	B	900	12.3						0	12.1	NO	4
49	TX049	54	F	164	ASSAULT	BLUNT	13	16	YES	102	94	24	96	A	900	11.5		1				1	11.8	NO	3.5
50	TX050	34	F	175	RTA	PENETRATING	14	19	YES	96	114	26	91	A	1000	11.3		1				1	11.7	NO	4
51	TX051	30	M	130	RTA	BLUNT	15	20	YES	110	98	18	99	A	1400	11.7		1		1		2	12.3	NO	3.5
52	TX052	24	F	157	ACCIDENTAL INJURY	PENETRATING	14	20	YES	92	114	24	89	A	1200	10.2		1				1	10.6	NO	4.5
53	TX053	32	M	120	ASSAULT	PENETRATING	14	18	NO	100	88	22	95	B	950	12.5						0	12	NO	4.5
54	TX054	26	M	96	ASSAULT	PENETRATING	13	17	YES	100	92	19	99	A	1000	12.1		1				1	12.8	NO	3
55	TX055	35	M	143	RTA	BLUNT	15	15	NO	120	86	21	94	B	800	14.2						0	13.8	NO	4
56	TX056	67	M	153	ASSAULT	PENETRATING	14	18	YES	98	100	22	96	A	1100	10.7		2				2	11.4	NO	4.5
57	TX057	22	F	142	RTA	BLUNT	14	15	NO	120	88	20	96	B	800	14						0	13.8	NO	3
58	TX058	30	M	124	ASSAULT	PENETRATING	13	16	NO	116	90	22	96	B	1000	11.9		1				1	12.9	NO	4
59	TX059	42	M	135	RTA	BLUNT	12	15	NO	118	92	21	97	B	800	12.8						0	12.5	NO	3.5
60	TX060	46	M	98	RTA	BLUNT	11	16	NO	104	98	22	97	B	600	12.5						0	12	NO	3
61	TX061	38	F	134	RTA	BLUNT	15	17	NO	110	94	18	98	B	800	12.6						0	12.4	NO	2.5
62	TX062	34	M	94	ASSAULT	BLUNT	14	20	YES	94	106	20	98	A	1200	11.3		1		1		2	12	NO	4.5
63	TX063	37	F	95	RTA	BLUNT	14	15	NO	110	84	19	96	B	500	11.8						0	11.4	NO	2.5
64	TX064	36	M	138	ASSAULT	PENETRATING	15	18	NO	100	96	19	98	B	800	13.4						0	13.2	NO	4
65	TX065	57	M	140	RTA	BLUNT	15	16	NO	104	110	18	95	B	900	12.8						0	12.4	NO	4

66	TX066	60	F	120	ACCIDENTAL FALL	BLUNT	15	15	NO	110	84	18	96	B	500	11.6						0	11.4	NO	3
67	TX067	36	M	127	RTA	BLUNT	15	16	NO	104	100	20	95	B	1100	12.5						0	12.5	NO	4.5
68	TX068	37	M	150	RTA	BLUNT	14	20	YES	100	104	18	98	A	1300	12.2		1				1	12	NO	3.5
69	TX069	62	F	124	RTA	BLUNT	10	15	NO	106	94	20	94	B	800	12.2						0	12	NO	3
70	TX070	49	M	150	RTA	BLUNT	15	16	NO	118	96	19	95	B	900	11.9		1				1	11.6	NO	3.5
71	TX071	19	F	130	RTA	BLUNT	11	15	NO	112	94	20	96	B	900	11.9						0	11.7	NO	3
72	TX072	39	M	96	RTA	PENETRATING	12	19	YES	100	102	18	98	A	1400	12.1		1				1	12.3	NO	4
73	TX073	41	M	75	ACCIDENTAL FALL	BLUNT	13	16	NO	110	86	20	98	B	1000	11.8						0	11	NO	3.5
74	TX074	26	F	110	RTA	BLUNT	14	16	NO	96	106	21	96	B	900	12		1				1	12.5	NO	3.5
75	TX075	65	M	80	RTA	BLUNT	13	18	NO	100	88	21	98	B	1000	12.6						0	11.6	NO	4
76	TX076	42	F	150	RTA	BLUNT	14	16	NO	106	96	18	96	B	900	13.1						0	12.4	NO	4
77	TX077	27	M	150	RTA	BLUNT	14	16	NO	112	88	21	96	B	900	13.2						0	12.4	NO	3.5
78	TX078	56	M	178	RTA	BLUNT	14	18	NO	100	94	21	96	B	1100	11.8		1				1	12.3	NO	4
79	TX079	24	M	114	RTA	BLUNT	15	18	NO	108	98	18	94	B	1000	11.7						0	11.4	NO	4.5
80	TX080	40	F	115	RTA	BLUNT	13	18	NO	104	86	21	95	B	850	13.6						0	13	NO	4
81	TX081	37	M	123	RTA	BLUNT	13	16	NO	110	96	22	95	B	850	12.6						0	12	NO	4
82	TX082	60	F	160	RTA	BLUNT	13	18	NO	120	85	18	98	B	1000	11.9		1				1	12.2	NO	4.5
83	TX083	60	M	150	ACCIDENTAL FALL	BLUNT	15	17	NO	120	88	19	98	B	800	12.3						0	11.9	NO	4
84	TX084	29	M	147	RTA	BLUNT	13	16	NO	108	86	20	97	B	800	13.2						0	12.5	NO	3.5
85	TX085	29	F	85	RTA	BLUNT	15	20	NO	90	112	20	96	B	1200	13.1						0	12.4	NO	5
86	TX086	36	M	159	RTA	PENETRATING	15	16	NO	104	92	21	97	B	750	13.4		1				1	13	NO	3.5
87	TX087	24	F	160	RTA	BLUNT	15	17	NO	114	88	20	95	B	1100	12.3						0	11.8	NO	4.5
88	TX088	40	M	72	RTA	BLUNT	14	19	YES	94	112	28	90	A	1000	12.1		1				1	12.4	NO	4
89	TX089	51	M	96	RTA	BLUNT	15	16	NO	120	90	20	96	B	500	16.7						0	15	NO	3
90	TX090	29	M	138	RTA	PENETRATING	11	24	YES	80	131	22	95	A	1400	10.2		2		1		3	11.2	NO	3.5

91	TX091	58	F	75	RTA	BLUNT	12	18	NO	94	121	20	96	B	1500	13.2						0	12.8	NO	4.5
92	TX092	32	M	112	RTA	BLUNT	12	18	NO	112	98	20	95	B	900	12.8						0	12.4	NO	4
93	TX093	24	M	67	RTA	BLUNT	10	16	NO	120	96	21	96	B	800	13.2						0	12.8	NO	4.5
94	TX094	27	F	134	ACCIDENTAL FALL	BLUNT	15	18	NO	106	108	19	94	B	1000	12.6						0	12.5	NO	3.5
95	TX095	67	M	116	ACCIDENTAL FALL	BLUNT	13	20	YES	100	96	24	94	A	1200	11.4		1				1	12	NO	4
96	TX096	50	F	98	ASSAULT	BLUNT	13	18	NO	106	98	21	96	B	600	13.2						0	13	NO	4
97	TX097	34	M	148	RTA	BLUNT	14	17	NO	116	86	18	96	B	1000	14		1				1	13.4	NO	4.5
98	TX098	61	F	140	ACCIDENTAL FALL	BLUNT	14	18	NO	110	94	18	96	B	1000	12		1				1	11.4	NO	4
99	TX099	36	M	123	ASSAULT	BLUNT	15	19	YES	98	118	21	98	A	1000	10.4		2				2	11.6	NO	3.5
100	TX100	30	M	114	RTA	BLUNT	10	18	YES	100	96	20	98	A	1200	11.4		1		1		2	12.2	NO	3.5
101	TX101	37	F	142	RTA	BLUNT	14	15	NO	118	86	22	97	B	950	12.4						0	11.9	NO	3
102	TX102	26	M	86	RTA	PENETRATING	12	16	NO	116	88	19	98	B	1100	13.6						0	13	NO	4
103	TX103	24	M	156	RTA	BLUNT	14	16	NO	114	90	21	97	B	850	11.6						0	11.5	NO	4
104	TX104	20	M	148	ACCIDENTAL INJURY	PENETRATING	15	19	YES	86	114	24	92	A	1200	11.6		1				1	11.6	NO	4
105	TX105	18	M	86	RTA	BLUNT	15	18	NO	110	96	20	96	B	900	11.8						0	11.4	NO	4
106	TX106	56	M	86	RTA	BLUNT	10	16	NO	106	94	21	98	B	940	12.4						0	11.4	NO	3
107	TX107	42	M	86	RTA	BLUNT	13	15	NO	108	94	22	97	B	950	13						0	12.8	NO	3.5
108	TX108	40	M	120	RTA	PENETRATING	14	16	YES	95	110	24	93	A	900	12.1		1				1	12.4	NO	3
109	TX109	29	M	137	ACCIDENTAL INJURY	PENETRATING	15	18	YES	98	112	24	96	A	950	11.4		1				1	11.9	NO	3.5
110	TX110	34	M	124	RTA	BLUNT	13	17	NO	104	86	19	98	B	500	13.4						0	13	NO	3.5
111	TX111	60	M	134	ACCIDENTAL FALL	BLUNT	13	19	NO	96	82	18	99	B	1200	10.1		2		1		3	11.4	NO	4.5
112	TX112	54	F	145	RTA	BLUNT	15	15	NO	118	94	18	98	B	1000	14.5						0	13.5	NO	4
113	TX113	26	M	84	RTA	BLUNT	15	15	NO	120	92	20	98	B	750	11.4						0	11.2	NO	3
114	TX114	20	M	86	ACCIDENTAL INJURY	PENETRATING	15	18	YES	98	108	23	95	A	1000	11.5		1				1	11.8	NO	3.5
115	TX115	30	F	120	RTA	PENETRATING	15	18	NO	120	86	20	99	B	800	12.5						0	12.4	NO	5

116	TX116	18	M	95	SELF INFLICTED INJURY	PENETRATING	10	20	YES	92	116	25	95	A	1200	10.3		2				2	12	NO	4.5
117	TX117	45	M	110	RTA	BLUNT	13	18	NO	110	90	21	99	B	850	11.7		1				1	12.5	NO	3.5
118	TX118	40	M	126	RTA	BLUNT	10	16	NO	123	86	19	97	B	900	12.6						0	12.2	NO	4
119	TX119	41	M	135	ASSAULT	PENETRATING	12	16	YES	100	100	20	97	A	800	12.2		1				1	12.5	NO	3.5
120	TX120	38	F	134	RTA	BLUNT	15	18	NO	100	90	20	98	B	860	12.8						0	12.5	NO	4
121	TX121	52	F	162	RTA	BLUNT	11	16	NO	112	88	18	99	B	800	11.8		1				1	12.5	NO	3
122	TX122	52	M	158	RTA	BLUNT	11	19	NO	114	90	21	97	B	1000	13.1						0	12.8	NO	5
123	TX123	32	M	164	ACCIDENTAL INJURY	PENETRATING	15	20	YES	90	120	28	90	A	1350	10.7		2		1		3	12	NO	5
124	TX124	37	F	157	RTA	BLUNT	12	18	NO	114	84	18	97	B	650	11.1		1				1	11.3	NO	4
125	TX125	51	M	154	RTA	BLUNT	13	16	NO	112	94	23	96	B	1100	11.8						0	11.9	NO	3.5
126	TX126	37	M	163	ASSAULT	PENETRATING	15	17	NO	116	90	20	97	B	1000	10.9		2		1		3	11.5	NO	3
127	TX127	29	M	124	RTA	BLUNT	13	18	NO	112	86	20	98	B	800	11.6						0	11.4	NO	4.5
128	TX128	37	F	128	RTA	BLUNT	14	18	NO	116	88	20	98	B	960	13.2						0	13	NO	4.5
129	TX129	22	M	158	ASSAULT	PENETRATING	15	19	YES	110	98	23	96	A	1000	11.6		1				1	11.8	NO	4
130	TX130	29	F	168	RTA	BLUNT	12	18	NO	119	92	22	96	B	1100	12.7						0	12.6	NO	4
131	TX131	37	M	160	RTA	BLUNT	14	15	NO	100	96	18	97	B	950	12.6						0	12.4	NO	3
132	TX132	42	M	89	ACCIDENTAL INJURY	PENETRATING	10	16	YES	100	94	20	95	A	800	12.4		1				1	12.8	NO	3.5
133	TX133	37	M	92	RTA	PENETRATING	16	18	YES	98	104	25	93	A	850	12.1		1				1	12.4	NO	4
134	TX134	35	F	168	RTA	BLUNT	11	16	NO	114	94	21	99	B	900	11.7						0	11.6	NO	4
135	TX135	21	M	96	ACCIDENTAL INJURY	PENETRATING	15	16	YES	100	96	24	95	A	750	12.5		1				1	12.8	NO	3.5
136	TX136	36	F	142	RTA	BLUNT	12	17	NO	104	94	20	97	B	800	11.9						0	11.2	NO	4.5
137	TX137	46	F	98	RTA	BLUNT	13	16	NO	98	106	18	98	B	840	10.8		2				2	11.9	NO	4
138	TX138	40	F	157	RTA	BLUNT	13	18	NO	100	104	19	97	B	750	13						0	12.4	NO	4.5

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