AUTOTRANSFUSION OF HAEMOTHORACES AND HAEMOPERITONEUMS: A REPORT ON TRAUMA AND RUPTURED ECTOPIC PREGNANCY PATIENTS.

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A DISSERTATION SUBMITTED TO THE FACULTY OF MEDICINE, UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG FOR THE DEGREE OF MASTER OF MEDICINE (SURGERY).

JOHANNESBURG, 1993.

Abstract

During the period June 1985-December 1989, 77 patients were accumulated for the autotransfusion trial, 21 of which were control patients. These patients were managed at three institutions namely Hillbrow (64), Coronation (1) and Shongwe (12) Hospitals. Of these 77 patients, 65 were involved in penetrating or blunt injuries, and 12 were ruptured ectopic pregnancies. The ages of all patients ranged from 16 yrs to 65 yrs.

The patients were divided into four groups:

1 banked blood only (controls), 21

2 autotransfused blood only, 27

3 combined banked and autotransfusion, 17

4 ruptured ectopic pregnancies, 12.

Investigated were the effects of autotransfused or banked blood volumes on the following parameters:

1 White cell counts: admission and post-transfusion day 1

2 Platelet counts: post-transfusion days 1 and 2

3 Haemoglobin: admission and post-transfusiondays 1 and 2

4 Prothrombin Index:post-transfusion days 1, 2, and 3

5 Partial thromboplastin Times: post-transfusion days 1 and 2

6 Fibrinogen Degradation products: post-transfusion day 1

7 Haptoglobin levels: post-transfusion day 1

8 Haemopexin levels: post-transfusion day 1

9 Fibrinogen levels: post-transfusion days 1 and 2

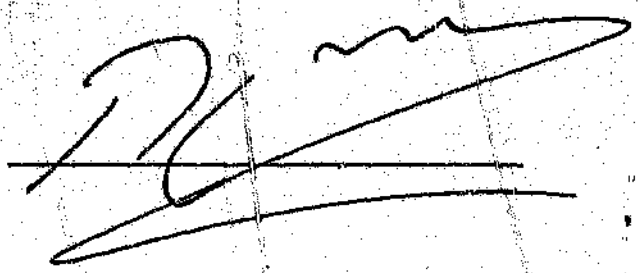
Four salvage techniques were utilised.

Complications were analysed for each transfusion group.

Autotransfusion of salvaged blood from haemothoraces and haemoperitoneums is safe, efficacious, and cost effective, provided that certain guidelines are followed.

DECLARATION

I declare that this dissertation is my own, unaided work. It is being submitted for the degree of Master of Medicine in Surgery to the University of the Witwatersrand, Johannesburg. It has not been previously submitted for any degree or examination to any other University.



25th. day of March 1993.

GROOTE SCHUUR HOSPITAL/HOSPITAAL, OBSERVATORY
Ex Officio: Commissioner of Oaths, C.P./Kommissaris van Ede, K.P.

J. Van Rensburg

Acknowledgements:

1 Dr Ken Boffard for agreeing to be my supervisor. Your approach to trauma has guided my career into trauma.

2 Professor Rosemary Hickman, UCT, without whose encouragement I would not have restarted the MMed.

3 Professor John Knottenbelt, who started me on PC's, without which this work would not have been possible. He has allowed me the use of his own designed database and statistical software programs.

4 Professor Giraud for the original idea while I was a registrar at Hillbrow Hospital.

5 My wife, Dr Dee McCormack, for all her patience and support. She has been a computer widow for months while I worked on this dissertation.

Glossary

#	Fracture
Abn	Abnormal
alpha-FP	Alpha fetoprotein
Anastom	Anastomosis
ARDS	Adult Respiratory Distress Syndrome
AUTO	Group receiving Autotransfused blood only
AUTOXFUSE	Autotransfuse(d)
BANK	Group receiving Banked blood only
BANKED	Group receiving Banked blood only
CAH	Chronic Active Hepatitis
CMV	Cytomegalovirus
DPL	Diagnostic peritoneal lavage
DIC	Disseminated intravascular coagulation
EBV	Epstein-Barr Virus
Ecto	Ectopic Group
FIBRINO1	First post-transfusion fibrinogen levels
FIBRINO2	Second post-transfusion fibrinogen levels
GIT,SCA+V	Gastrointestinal tract & subclavian vessels
HAEMOPEX	Day 1 post-transfusion haemopexin levels
HAPTOGLOB	Day 1 post-transfusion haptoglobin levels
Hepatec	Hepatectomy
HCC	Hepatocellular Carcinoma
MVA	Motor vehicle Accident
HB	Hepatitis B
Hb0	Admission Hb
Hb1	Day 1 Hb
Hb2	Day 2 Hb
HBsAg	Hepatitis B surface antigen
HCVH	Hepatitis C Viral Hepatitis
HIV	Human Immunodeficiency Virus
HTLV	Human T-cell Lymphotropic Virus
HVGSW	High Velocity Gunshot Wound
ITartery	Internal thoracic artery
LAP	Laparotomy
livr/spln	Liver & spleen
LO	Low
MIX	Group receiving Banked and autotransfused
MRC	Medical Research Council
NANB(H)	Non-a, Non-B (Hepatitis)
PI0	First post-transfusion PI
PI1	Second post-transfusion PI
PI2	Third post-transfusion PI
PLT0	First post-transfusion Platelet count
PLT1	Second post-transfusion Platelet count
Pt	Patient
PTT0	First post-transfusion PTT
PTT1	Second post-transfusion PTT
spln+Diaf	Spleen & diaphragm
SA	South Africa(n)
Thoracot	Thoracotomy
VLO	Very low
WCC0	Admission White cell Count
WCC1	Day 1 White cell Count

Contents:

AUTOTRANSFUSION DATA SHEET:

NAME:_____ HOSPNO:_____
AGE/DOB:_____ SEX:_____ RACE:_____
ADMITTED:

ADMISSION/PREOP:

BP:_____/_____ ; PULSE:_____ ; CVP:_____
PERFUSION:Good/intermed/poor ; HAEMATURIA:_____
CXR:_____
AXR:_____
OTHER:_____

INJURIES/PROBLEM AREAS:

EMERGENGY / ELECTIVE ; INJURY-->OP PERIOD:_____
HEAD:NO / YES:_____
ABDOMEN:NO/YES:_____
CHEST:NO/YES:_____
LIMB:NO/YES:_____
DPL:NO / CLEAR / PINK / POSITIVE / OTHER:_____
FRACTURE: NO / YES: CLOSED / OPEN / COMMINUTED / DEPRESSED
PELVIS/HUMERUS/RAD-ULNA/PELVIS/FEMUR/TIB-FIB/SPINE:_____
BURNS:NO / YES : SUP / PT / FT / %: ;FIRE / CHEM/RESP

PREOP INVESTIGATIONS:

Hb_____	WCC_____	PLT_____	PI_____	PTT_____
pH_____	PO_2_____	PCO_2_____	SBC_____	FiO_2_____

OPERATIVE FINDINGS:

ABDOMEN:_____
CHEST:

UNITS AUTOTRANSFUSED:

0-5 6-10 11-15 16+ ( )

BANKED UNITS TRANSFUSED:

ANTICOAGULANT:

HEPARIN ACD VENTURI BOLUS

INTRAOP RESULTS:

Hb_____	WCC_____	PLT_____	PI_____	PTT_____
pH_____	PO_2_____	PCO_2_____	SBC_____	FiO_2_____

COMPONENT THERAPY:

PLATELETS units FFP units

POSTOP RESULTS:

	DAY-1	DAY-2	DAY-3
Hb	*	*	*
WCC	*	*	*
PLT	*	*	*
FDPs	*	*	*
HAPTOGLOBIN	*	*	*
HAEMOPEXINS	*	*	*
PI	*	*	*
PTT	*	*	*
pH	*	*	*
PO2	*	*	*
PCO2	*	*	*
Na	*	*	*
K	*	*	*
U	*	*	*
Cr	*	*	*

COMPLICATIONS:

CHEST: _____
ABD: _____
OTHER: _____

MORTALITY: NO / YES

	INTRAOP	POSTOP
REASON:		
WHEN:		

COMPOSITION OF SALVAGED BLOOD:

HAEMOTHORAX (HT)	HAEMOPERITONEUM (HP)

INVESTIGATIONS ON HT/HP:

FIBRINOGEN: _____
PLT: _____
Hb: _____
WCC: _____

EFFECT OF MICROFILTRATION:

	BEFORE	*	AFTER
PLT:		*	
WCC:		*	

COMMENTS:

1. INTRODUCTION

Trauma today is a national epidemic, accounting for one of the most serious health care problems within this country [1 2]. The indications are that only AIDS will compete with trauma as the number one community health hazard in the foreseeable future [3].

Progress in developing countries usually results in increased urbanization, improved health care, and a reduction in malnutrition and infectious diseases. The reverse is true of trauma. Exposure to urban life and technology increases trauma rates, precipitating a high demand for trauma services.

Trauma is the principal cause of death between the ages of 1 and 40 in South Africa [4]. In terms of overall mortality, trauma occupies a disturbing and close second place behind all circulatory related deaths [3]. It is also the leading cause of death and disability in North Americans less than 38 years of age [5], and the fourth leading cause of death in Americans of all ages.

A 4 year review of the Cook County Medical Examiner's records, Chicago, Illinois, 1986-1989, indicate that trauma is the leading cause of non-maternal maternal death (46.3%) [6]. By non-maternal death is meant any cause of death not from pregnancy, labour, delivery, postpartum, nor any pre-existing health problems exacerbated by pregnancy.

The World Health Organization (WHO) estimates that non-natural deaths, almost all due to trauma, represent 5.2% of all global fatalities, ***whereas in South Africa, these figures represent 16% of national deaths*** [3]!

There is ar increasing motor vehicle incident rate, subsequent to alcohol use, unroadworthy vehicles, inadequate training of drivers, and a rising anarchistic behavior on the road. Poor legislation and often inadequate or total lack of law enforcement also contributes [7].

There is a not unreasonable reluctance on the part of law enforcement officers to deal with the rising minibus problem, associated with a significant number of unlicensed firearms in the hands of drivers and passengers. There are presently (1992) an estimated 122 000 minibuses in the PWV area, of which only 70 000

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There is a not unreasonable reluctance on the part of law enforcement officers to deal with the rising minibus problem, associated with a significant number of unlicensed firearms in the hands of drivers and passengers. There are presently (1992) an estimated 122 000 minibuses in the PWV area, of which only 70 000

are licensed with a minibus association. (Mr L Katz, Deputy Director; Operations, Brixton Ambulance and Fire Dept.).

There is a high incidence of pedestrian injuries, particularly in the Cape Peninsula. Many of these incidents are alcohol related, as are many MVA-driver related events. These incidents often occur in outlying areas lacking in trauma expertise. Consequently, there is a high incidence of poorly managed, shocked patients arriving at major trauma centres [8].

In 1989, the MRC conducted a survey of trauma patients in the Greater Cape Town Metropolitan Area. Included were all state institutions, clinics, and private practices of general practitioners, specialists, and dentists. A clinical (not blood alcohol level) estimate of alcohol related trauma was 30%.

The MRC figures included patients having died within 4 hours of MVA related incidents.

68% were pedestrians. Of these, only 24.2% were alcohol free. 3.3% had levels <0.08g%, 14.2% had levels between 0.08 and 0.19g%, while 58.3% had levels exceeding 0.2g%.

Considering MVA-drivers, 47.4% were found to be blood alcohol negative, 5.2% <0.08g%, 58.5% between 0.08 and 0.19g%, while 15% had levels exceeding 0.2g% (Dr J W van der Spuy, MRC.).

A rising level of person to person violence is also responsible for an overwhelming number of injuries and deaths, affecting a significant proportion of South African society. It is estimated that more than 5% of people require treatment annually as a result of trauma [9]. A 1989 population and patient analysis of the Cape Peninsula estimates a rate of 6% [3]. This tragically includes a paediatric and young adult population. It has assumed such proportions that Muckart has called it the *"malignant epidemic"* [10]. This supersedes the statement made by Prof. S P Baker *"injuries-the neglected epidemic"* [11]. Claude Organ from the USA commented in a Editorial recently that ***"trauma is the motor end plate of violence"*** [12].

The increase in trauma admissions to medical institutions has been so marked in recent years that it threatens the very infrastructure of medical services in this country!

The medical and nursing staff work under tremendous pressure, resulting in overwork, and stress [13]. This leads to rapid burnout and an inevitable high staff turnover.

In many institutions, staff to patient ratios have decreased to worrying levels over the last 5 years, leading to the undermining of South African medical standards. This is particularly noticeable in peak demand times. Public holidays and religious holidays are usually so busy as to overwhelm existing resources. This leads to the inevitable death of patients when the staff is too busy with other emergencies. There are other drawbacks; in an attempt to deal with increased numbers of patients, medical staff have to reduce the time spent on each patient, thus leading to a reduction in medical standards and increasing the incidence of missed pathology.

The economic consequences of trauma are massive. In South Africa, the composite cost of vehicular incidents and occupational injuries accounted for R20 million per day in 1988 [14].

Studies in the USA, involving injuries and resulting costs, showed that in 1985 alone, 57 million people were injured, costing $157.6 billion. This excluded deaths.

When deaths were considered, trauma related deaths accounted for $335 000.00 per death, which was three times the cost of deaths from cancer and cardiovascular diseases combined. Trauma causes a far greater loss of working years than all forms of cancer and heart disease combined. It occupies an enormous national resource in terms of hospital beds, days lost at work, and significant permanent disability [15].

It is estimated that trauma cost the USA $180 billion in 1988, which exceeded all other disease categories [16].

The cost of managing such patient loads in South Africa, is outstripping the budgets allocated to the medical services, at a time where political reforms and a severe economic depression is limiting available financial resources.

The realities of trauma in South Africa indicate that an increasing load will be placed on curative services. This obviously conflicts with the sociopolitical aspects of this country, where a greater emphasis is to be placed on preventative services. However, in the

case of trauma, no amount of preventative health care will change this.

Just how far the health budget cutbacks go is seen in the recent announcements (October, 1992) regarding the laying off of health workers and the acceleration of patient discharges in Natal, where a low grade civil war is currently being waged.

Hypovolaemic shock accounts for a significant proportion of surgical and gynaecological admissions.

Many hospitals do not incorporate a blood bank and a significant delay may exist between the presentation of a trauma victim, and the arrival of ordered banked blood.

Other hospitals have small blood banks on their premises, but with limited supplies. Whatever the situation, a delay always exists from the time required to run compatibility tests until the blood is issued. Many institutions stock limited quantities of group O Rh +ve or O Rh -ve blood for circumstances in which blood is urgently required, but these supplies are often insufficient in the event of severe blood loss from trauma.

Having worked at the Hillbrow Hospital as a surgical registrar for 2 years during the period 1984-1985, I was dismayed to find that no emergency blood was allowed in the trauma casualty; furthermore, although this hospital was virtually across the street from the South African Blood Transfusion Service, considerable delays were often encountered from the time of ordering to the arrival of blood. This in many instances was the result of an inefficient and disinterested transport system employed by the hospital. Consequent to the above problems, autotransfusion appeared to be an important source of alternative emergency blood supply for many patients with hypovolaemic shock. This may eliminate or reduce the requirements for banked homologous blood transfusions. This stimulated my investigation into the feasibility of introducing an autotransfusion facility to the surgical department.

For this to be successful and practical, simple apparatus and ease of operation were essential. Furthermore, the logical and practical sources of autogenous blood would be trauma related haemothoraces and haemoperitoneums.

As an incidental event, we saved the life of a young woman admitted with a ruptured ectopic pregnancy at Hillbrow hospital. She had a hypovolaemic cardiac arrest soon afte. arrival. Autotransfusion of the blood during her cardiopulmonary resuscitation almost certainly contributed to her survival and discharge within a week of admission.

This led to the following questions which required elucidation:

1. Is autotransfusion safe?
2. Is autotransfusion efficacious in the management of hypovolaemia?
3. Is autotransfusion cost effective?
4. Does it represent an immediate source of blood?
5. Can it prevent the potential hazards of homologous banked blood transfusions?

By definition, autotransfusion is a method of blood conservation where a patient's blood is collected and subsequently returned to that individual's circulation. This may be either before, during, or after an operative procedure. The blood may be processed in various ways, the simplest being filtration and anticoagulation, or the erythrocytes are separated from the salvaged blood, washed and returned as a concentrated cell suspension in a crystalloid fluid.

2. LITERATURE REVIEW

2.1. ORIGINS

The use of autotransfusion was first reported in 1818 by James Blundell. He had witnessed the death of a woman from uncontrolled uterine hemorrhage and this led him to conduct a series of experiments in which dogs were bled to shock states and then reinfused with the shed blood [17]. He then used this technique for the first time in London in 1818 for post partum haemorrhage, which, at those times and with available techniques, was associated with a mortality rate of 50% [18]. William Highmore attended a patient with fatal postpartal haemorrhage in 1874 and called for the future collection and use of the exsanguinated blood [19 20 21].

The complications of haemolysis, intravascular coagulation, air and particle embolism [22] and technical problems, such as blood clotting before transfusion [22], limited the widespread adoption of this technique.

Autotransfusion in the setting of chest injuries was used by Elmendorff in a wartime situation in 1917 and in civilian life in 1931 [18].

The earliest American report of autotransfusion was by Lockwood in 1917, where intraoperative reinfusion was undertaken during a splenectomy [23]. Early reports in the 1920's and 1930's described autotransfusion more commonly used in haemoperitoneums than in ruptured ectopic pregnancies and traumatic abdominal vascular injuries [18]. There was a general resistance to the procedure because of possible contamination of the blood.

After Landsteiner and Roce described the blood group system and when methods for preventing clotting were discovered, blood banks were established in 1935. This led to the decline of autotransfusion.

In 1969, Symbas was responsible for autotransfusion gaining acceptance in haemothoraces following thoracic trauma [24].

Only in the early 1970's did autotransfusion get the stamp of credibility from Breuer et al and Brewster et al, when certain safeguards were put forward [25 26].

It is only in the last two decades that autotransfusion has become an established blood conservation technique.

3. REASONS FOR USE

3.1. BLOOD BANK SHORTAGES

Difficulties in obtaining adequate stocks of banked blood, as a result of increasing demand for banked whole blood [27], donor shortages, particularly during public holidays and school holidays, rare blood groups and expiry, frequently leads to periods of decreased blood availability with consequent cancellations of elective surgical procedures and at worst, death from hypovolaemia [28 29]. Autotransfusion thus reduces, and in some instances, may even eliminate the demand for banked homologous blood [30 31 32].

3.2. TRANSMISSION OF DISEASES

Blood transfusions pose the same dangers as any other tissue transplant, namely the transmission of infectious diseases from the donor to the recipient. These infectious agents range from the age old treponemal disease, syphilis, to the latest esoteric group of viruses, HTLV 1 and 2. It is, however, the appearance of the HIV agent which has forever changed world attitudes. This applies not only to blood products, but also to the injured patient management and the potential hazards to the attending staff.

Transfusion-transmitted infective agents, eg viruses, are classified into two classes: those in which a cell-free viraemia occurs, and those in which the viraemia is cell-associated (intracellular). Intracellular viruses include the cytomegalovirus and the Epstein Barr virus, while cell-free viraemias are primarily those responsible for post-transfusion hepatitis [33]. These transmission modes have important implications regarding control of viral transmission in blood transfusions.

3.2.1. HEPATITIS B

Hepatitis from the B-virus has been markedly reduced, but not completely eliminated by the routine testing for the Hepatitis B surface antigen (HBsAg), antibody (anti-HBsAg, anti-HBcAg), and elevated transaminases. Hepatitis B accounts for less than 9% of all transfusion acquired hepatitides [28 34].

The frequency of transfusion transmitted HBV infection in the USA is estimated to be 1 in 50 000 per recipient, or about 1 in 200 000 per unit [35].

The HBV, a DNA virus of the hepadnavirus family, is an extremely infectious agent, present in large numbers in blood and other body fluids of infectious patients [36]. These include individuals with acute HB and chronic active hepatitis (CAH). HB is associated with a 10% mortality rate in the acute phase, while approximately 20% of clinical cases of HB progress to chronic active hepatitis (CAH). In some series, 25%-50% of patients developing CAH die within 5 years [12]. In the light of such figures, it is essential to prevent spread of the HBV.

3.2.2. HEPATITIS-C

Hepatitis C, before the discovery of the hepatitis C virus, HCV, was known as Non-A, Non-B Hepatitis, NANBH. Articles concerning NANBH are still appearing in the literature, and the term NANBH is probably still familiar to most in the academic world.

More than 15 years have elapsed since viral hepatitis was recognized, and it was only in 1974 that NANBH was identified as a clinical entity, yet transfusion associated hepatitis remains a high priority concern even today [34]. Recently, it has been estimated that 7-10% of all patients receiving banked blood will develop hepatitis, icteric or anicteric. Of these, 95% are caused by the NANB type [28 34 37 29]. This represents about 300 000 patients [38].

This frequency of hepatitis occurs even among recipients of volunteer banked blood; in recipients of paid donor blood, the incidence is 30-40% [34].

Seventy five percent of acute NANB hepatitis are asymptomatic, as are a majority of those whose disease progresses and becomes chronic. Chronic hepatitis (persistence of elevated aminotransferases for at least 6 months) evolves in about 50% of acute NANB hepatitis patients. Cirrhosis develops in up to 20% of patients with chronic NANB transfusion associated hepatitis, or 1% of all transfused persons, within 10 years after an acute illness [28 29 34].

In 1989, Choo et al isolated a cDNA clone from a parenterally transmitted NANBH viral genome and designated it as the hepatitis C

virus HCV [39] [40]. Since that discovery, and the development of an antibody test, rapid advances have been made with regard to the epidemiology and oncogenicity of the HCV. The first-generation antibody tests have the disadvantage of giving false positive results, in as much as 50-70% of cases [41] [42], while it may take a year after exposure before seroconversion occurs; the second generation tests are more accurate and sensitive. All of these tests are based on the detection of antibodies against non-structural viral proteins [43], c100 and c33. A polymerase chain reaction is now available to detect HCV RNA in liver and serum.

The HCV is a single positive-stranded RNA virus distantly related to the flaviviridae, [43] and appears to be less infective than the HBV.

In contrast to the HBV, the HCV is not transmitted as well by sexual contact [44] [45].

Recently 2 distinct strains have emerged and may eventually require the designation HCV 1 and HCV 2 [46].

It has also become apparent that the HCV is oncogenic, far more so than the HBV [47] [48]. The chronicity and cirrhosis rates for HCV is identical to that of NANBH [36], however, there appears to be a sequential development of cirrhosis and hepatocellular carcinoma (HCC). Chronic HCVH with cirrhosis is associated with elevations of alpha-FP, even in the absence of HCC [49].

The frequency of HCV in NANBH shows a wide variation. Studies using anti-HCV range from 50%-66% [36], 90% [50], 91% [40], 95% [43] to 100% [39]. There is also a difference in positivity rates between acute and chronic HCVH, the rate increasing with chronicity. The reason for this is the delay in rising antibody titres in HCVH.

Comparing NANB HCV positive disease to HBV disease, we see the following: [51]

	chronic hepatitis	cirrhosis	HCC
HCV	90%	86%	94%
HBV	6%	17%	35%

When the HCV RNA test is used, the presence of HCV is detected in half the cases of chronic NANB liver disease where anti-HCV tests are negative [52].

In the case where HCV and HIV coexist, the immunosuppressive consequences of AIDS lead to a more rapid onset of cirrhosis [53].

HCV is endemic in many populations: Western Europe 0.7%, Japan 1.3%, New York City 1.4% and 5.3% in Riyadh, Saudi Arabia. The Cape Peninsula appears to have a very low incidence of HCV carriers. In half the cases no known source of infection is present; it is unknown how such large numbers of people become HCV carriers [43 54]. In one study, it is noted that the HBV was found in 7.7% of a trauma population, thus posing another threat to trauma staff [55]. There was a relationship between cocaine use, HIV positivity, and HCV positivity. In the discussion following the presentation of this data at a congress, a 10% HCV prevalence was noted in another trauma population. These figures contrast with rates of 1.2% with HBV, and 0.8% with HIV in the same trauma population.

With available tests, it is estimated that the frequency of HCV transmission is less than 1 in 3 000 units of transfused blood components. However, these figures are already outdated in that newer, more sensitive tests for the HCV antibodies have been introduced [35].

HCV may also be transmitted by blood components, FFP, Factor VIII etc.

The economic and health related aspects of HCV infection are enormous. In the USA, there are at least 170 000 cases of NANBH annually, of which 85 090 cases of chronic hepatitis will follow. Large numbers of HCC may follow. This progression is slow, but decompensation eventually occurs.

Interferon therapy has recently been reported to of value in HCV infection. Reports of viral eradication and normalization of transaminases in acute HCVH have emerged. If that is the case, progression to chronicity and HCC may be prevented [39 43 47].

Ribavirin appears to be the first oral drug effective against the HCV [56].

3.2.3. ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)

The next greatest transfusion borne scourge is AIDS [22 29 30 31 34 57 58 59 60].

Although all blood donated in developed countries since 1986 is tested for HIV antibodies, false negative tests occur in 1 per 30 000 to 1 per 300 000 units (0.003%-0.0001%). More recent

estimates of HIV risks are of the order of 1 in 225 000 per unit, although risk rates of 1 in 60 000 to 1 in 40 000 may occur in areas of high HIV prevalence [67].

South African figures vary according to the type of population studied, and the area within the country. At Baragwanath Hospital, 1% of antenatal positive Wasserman positive sera were HIV positive in 1990, while 2.7% of women were HIV positive at STD clinics. Ciskei antenatal HIV positivity was up to 2.3% in 1990; STD attenders in Durban in 1989 were found to have a 3.95% seroprevalence [61]. Other figures from the same institution show seroprevalence rates in pregnant women to have increased from 0.32% in 1988 to 0.82% by the end of 1990 [62]. The South African Blood Transfusion Service has also become involved in antenatal HIV testing. During the period 1 May 1987 - 31 October 1988, 104683 samples from pregnant women resident in the southern Transvaal were tested. The HIV 1 seroprevalence increased from 0.036% in 1987, to 0.217% in 1988, a doubling time of 5 months on the basis of ELISA reactors [63].

As the seroprevalence of a population rises, so the risk of transfusion related HIV transmission will increase proportionally. In the South African context, several studies have looked at HIV seropositivity. Not only are trauma staff at risk of HIV exposure, but here and in the USA, the rising incidence of HIV associated TB is another concern. Where occupational HIV exposure is by blood exposure, TB is by aerosol exposure, a far greater risk [64].

The commonest cause of transfusion associated HIV transmission remains the individual who has not yet seroconverted 31. An individual may take 3-24 months to develop the marker antibody, although virtually all seroconvert by 6 months.

Another possibility is viral mutation leading to antigen alteration making produced antibodies undetectable with existing kits.

A third possibility is the HIV 2 virus. No HIV 2 positivity was found in 18 million units of blood during 1987-1989; HIV-2 is presently not screened for.

By 1984, in the U.S.A., about 1% of AIDS cases were transfusion related, by 1986 it had risen to 2% of adults and 13% of childhood cases.

Blood component therapy was also responsible for the transmission of AIDS, as in clotting factor concentrates for haemophiliacs[59].

By March 1987, over 550 known cases of transfusion associated AIDS have been documented[37]. In 1987, just under 1200 new cases were reported and peaked at 1300 new cases in 1988. Since then there has been a slow but steady decline in annual reporting, and in 1990 accounted for 1276 cases. This follows the introduction of routine HIV screening of donated blood, in 1985[65].

As a result of media coverage of HIV and AIDS, patients may now refuse blood transfusions and many blood donors have ceased donating blood out of the unfounded fear of acquiring AIDS during the donation[60]. This and general concern, even hysteria about AIDS, is another stimulus for the use of autotransfusion.

With regard to haemophiliacs, blood transfusions, and organ transplant recipients, HIV screening has now almost eliminated HIV transmission.

AIDS is now the second commonest cause of death of adult males in the USA in the age range of 24-45 years, trauma leading by a small margin only.

The HIV, actually HIV 1, formerly known as HTLV III and LAV, an RNA agent, is a retrovirus of the lentivirus family. It selectively infects helper T-cells of the CD4 subtype, using reverse transcriptase to manufacture DNA from the RNA template. This proviral DNA is integrated into the chromosomal DNA (genome) of the host cell. Here it remains dormant for extended periods, but proviral DNA eventually replicates to produce new viruses which infect other CD4 cells, leading to a gradual reduction in the numbers of these cells until a critical level of 200/mm^3 is reached. At this stage, a deficient cell mediated immunity develops. This takes 5-8 years and the patient becomes susceptible to opportunistic infections .

Recent findings suggest that one or more HIV superantigens may be responsible for anergy and cell loss, in addition to direct viral activity[66].

Regarding HIV infection in the light of 1992 knowledge, the following may be said; there is an inevitable progression and will ultimately be fatal in virtually all patients 7-12 years after infection. The HIV disease has 3 distinct clinical expressions;

consequences of the HIV itself, immune complex disease opportunistic infections and tumours.

HIV 2 is also a retrovirus causing disease similar to HIV 1. It is sufficiently different in antigenicity from HIV 1 that current HIV kits based on anti-HIV 1 will miss up to 40% of these cases. It is found virtually only in West Africa or in persons originating from that region.

In 1992 the CDC is expected to revise the definition of AIDS as the presence of anti-HIV and a CD4 count of less than 200/mm^3 [67].

HIV antigen screening of donor blood is the next step and may lessen the chances of HIV transmission [22].

3.2.4. HUMAN T-CELL LYMPHOTROPIC VIRUS (HTLV) 1 & 2:

These viruses were discovered a short time before the HIV, which was previously called HTLV 3. They are retroviruses belonging to the subfamily Oncovirinae which is distinct from the subfamily Lentivirinae, which includes the HIV [68].

The HTLV 2 is currently a virus in search of a disease, in contrast to the HTLV 1, which is linked etiologically to adult T-cell leukaemia/ lymphoma (ATLL) and HTLV 1 associated myelopathy/ tropical spastic paraparesis (HAM/ TSP).

An overall seropositivity rate of 0.025% has been found in the USA. Most of the tests presently used do not distinguish between HTLV 1 and 2. Thus HTLV seropositivity is of both viruses unless specified otherwise. 60-80% of positive tests are due to the HTLV 2 virus.

HTLV 1 is endemic in many populations around the world and is spread by similar routes to HIV and HBV. Transfusion spread is the most efficient method with seroconversion rates of up to 60% following exposure to HTLV 1 contaminated blood products. Sexual transmission also occurs, particularly from male to female.

Although ATLL has not been linked to previous transfusions, the transfusion of HTLV 1 contaminated blood has caused HAM/ TSP within 1 month to 3 years [68].

The lifetime risk of ATLL in HTLV 1 carriers is 1-6%, having a long latent period [69] [70]. There is an incidence of 0.1% of HAM/ TSP in

carriers of the virus. Twenty five percent of HAM/ TSP in Japan are transfusion related, while 2% of donors are HTLV 1 positive.

The transmission of HTLV 1 via blood products has been virtually stopped since the introduction of routine screening of donated blood in 1986.

The USA has also introduced HTLV 1 screening of donated blood in reaction to the potential diseases caused by this agent. On the other hand, non-endemic areas in Europe have not implemented screening until further epidemiological data becomes available.

3.2.5. NON-A, NON-B, NON-C HEPATITIS

At present it is unclear whether this condition is of any importance and exactly what agents may be involved.

One group found this to be a transient subclinical event [71], while another group found hepatitis E virus (HEV) RNA in the serum of a patient with fulminant hepatic failure [72]. It is spread in a similar fashion to the HAV. It is a single positive stranded RNA virus [73].

There is a possibility of a HFV and more because a large proportion of HCV patients are negative when other tests are utilized.

3.2.6. HEPATITIS D / DELTA VIRUS (HDV)

This agent is spread in similar fashion to all of the above mentioned viruses.

It is a hybrid organism, consisting of a single stranded RNA core, surrounded by HBsAg. Its genome does not code for a viral capsule and thus relies on the HBV for capsular provision. As a result of this interaction, the HDV can only infect hosts in which HBV is already present. This implies that HDV infection may occur as a coinfection or as a superinfection. The agent is endemic in various parts of the world, but like all of the viruses already discussed, it can easily be spread by modern transport. In first world countries it is spread primarily by means of blood products, sexual contact and contaminated needles.

Coinfections usually result in acute hepatitis only, while superinfections result in acute hepatitis D, usually followed by chronic infection. There is an incidence as high as 60% of chronic

active hepatitis. Fulminant hepatitis which is often fatal, may occur with either coinfection or with superinfection.

The routine testing of blood for HBsAg etc has markedly reduced the transmission via blood products [74].

3.2.7. SYPHILIS

A treponemal disease which uncommonly can be transmitted via blood products [22 30 32 60].

3.2.8. MALARIA, BABESIOSIS

Both are protozoan organisms able to be transmitted by blood transfusions. Malaria is a common condition in Southern Africa and can be transmitted in the case of active disease at the time of blood donation. Plasmodium falciparum, ovale, malariae, and vivax are all a possibility in South Africa [22 30 32 60].

Babesia microti and B devergens are rare complications of transfusions. There have been only 7 documented cases [75].

3.2.9. CYTOMEGALOVIRUS, EPSTEIN-BARR VIRUS

These are uncommon but important agents transmitted by homologous blood transfusion [30 32 22 60].

The CMV is a herpes virus with a DNA core, surrounded by a complex double capsid. Leucocytes have been implicated as the source of CMV in blood transfusions. The exclusion of seropositive units eliminates the risk, but this is impractical in populations where the prevalence of CMV > 70% .

The EBV is also a herpes virus. It may be transmitted by leucocyte containing blood products. Little is known about transfusion related disease and is probably minimal.

The EBV is now thought to cause CNS lymphomas in HIV infected patients [76].

3.3. TRANSFUSION REACTIONS

This includes febrile non-haemolytic reactions, allergic reactions, haemolytic reactions, and allo-immunizations [28 29 30 31 22 60 77]. Febrile reactions occur in at least 2% of all transfusions [31].

3.4. IMMUNOSUPPRESSION BY HOMOLOGOUS TRANSFUSIONS

There is little doubt today that homologous blood transfusions are immunosuppressive [78]. It is only since 1973 that blood transfusions have become accepted as an immunosuppressive modality in human transplantation [78]. The mechanisms are only partially understood.

Transfusions alter macrophage behavior, decreasing chemotaxis. There is an increase in eiconasoid synthesis, particularly that of prostaglandin E_2, [78], (PGE_2), which is a potent immunosuppressive, inducing a broad based immune inhibition. It inhibits lymphocyte proliferation, natural killer cells and activates suppressor cell networks [79]. The PGE's also suppress B lymphocytes, thereby reducing immunoglobulin synthesis [80].

Perioperative blood transfusion in patients undergoing surgery for malignancy, has been reported as having a higher recurrence rate of cancer [81]. Several studies suggest this is so, one, indicates a significantly earlier tumour recurrence associated with decreased survival [82]. This is true for animal models, but has not been conclusively proved in humans with prospective studies [83]. This is supported by a study where a non-significant effect was seen [84]. Until hard evidence becomes available, no recommendations concerning the use of blood in patients with malignancies are justified [85].

Transfusions resulting in immunosuppression may contribute to postoperative sepsis and septic related mortality [86]. There is good evidence in animal models of transfusion related sepsis, although this has not been confirmed by every study. Clinically, there is support for this finding. A Spanish group found a relationship between transfusion and postoperative sepsis in surgery for gastric carcinoma [87]. These findings are supported by a Danish study, where whole blood was associated with depression of natural killer cells, and 23% postoperative sepsis, compared to 2% sepsis in patients

transfused with filtered blood free from white cells and platelets [88].

There is evidence of long term, even life-long immunomodulation, in patients having had whole blood transfusions in that significant reductions in helper cells were found [89].

Although transfusions may have an immunosuppressive effect, shock is probably more important and has greater effects on immune function [90] [91]. Elevated cortisol levels as a result of stress are known to reduce neutrophil chemotaxis for up to 10 days. This is another mechanism by which immunosuppression may occur [92] [146].

In addition to shock, burns, sepsis, major tissue trauma, aspiration, pulmonary contusions, multiple major transfusions and drug overdoses are associated with profound immunological alterations. All these events initiate prostaglandin (PG) synthesis and release. This by itself predisposes to infection. Although immune cell counts are normal in trauma patients with immunosuppression, cell functions are suppressed; cells are also resistant to exogenously administered cytokines such as IL-2 [93]. However other more recently discovered events occur.

The same insults as above result in the translocation of viable and non-viable (endotoxin) gut organisms into the portal venous and lymphatic systems. This translocation has important effects on the host.

Although thermal injury is the model used to study translocation phenomena, other major insults to the living organism probably have similar sequelae. It is postulated that hypovolaemia and splanchnic vasoconstriction lead to relative intestinal ischaemia, activation of various factor cascades, neutrophil activation, with subsequent injury to the mucosal barrier [94] [95]. Ischaemic insults to endothelial cells reduce their endocrine function of nitric oxide (NO) release. Reduction in this endogenous vasodilator leads to unopposed vasoconstrictor activity by the endogenous endothelin peptides ET 1, ET 2, and ET 3 [96] [97] [98] [99] [100] [101]. Neutrophils are less compliant than erythrocytes, have a greater diameter, (7.5μ), than capillaries (5μ), and when activated, become even less compliant. Reduction in capillary diameter contributes to capillary plugging by white cells.

Gastrointestinal mucosal barrier disruption leads to rapid and extensive migration of organisms, the mechanism has been extensively investigated and is now believed to occur in the following manner.

Bacteria and fungi adhere to gut epithelium, pass through the cell membrane and enter the cytoplasm. They are not enveloped by cell membrane and the process is therefore not that of phagocytosis. The organisms are then extruded from the cell at the basement membrane, to lie free in the basement membrane, or within venules or lymphatics. The organisms are often phagocytosed by macrophages or neutrophils at this stage and transported within these cells to more central areas.

Bacteria are to be found in peritoneal fluid, and in regional lymph nodes, spleen and liver. Viable organisms are to be found mainly in the lungs, liver, and to a lesser extent, in the spleen and lymph nodes [102]. Of all translocating organisms, only 1% are alive in the peritoneal fluid, while 0.01% are to be found to be viable in lymph nodes and spleen. 1% reach the liver alive and 0.5% the lungs.

These organisms cause stimulation of macrophages which in turn release cytokines such as interleukin-1 (IL-1), IL-2 and tumuor necrosis factor (TNF). The interleukins, of which 9 are now known, stimulate the brain to release cortisol via neuromediators.

Complement activation results in the appearance of immunosuppressive degradation products like C3b, C3c, C3d and C1 split products. C3b can inhibit neutrophil phagocytosis and bacterial killing. C3a and C5a activate neutrophils, mast cells, endothelial cells and causes the conversion of hypoxanthine dehydrogenase to xanthine oxidase. This produces a respiratory burst, releasing toxic products such as superoxide (O_2^-), H_2O_2 and HO·. Histamine is also released, increasing the activity of xanthine oxidase by a positive feedback mechanism.

Much attention has been focussed on free radicals and their effects. Early reperfusion is an absolute requirement for tissue survival, however, reperfusion has been called the "*double edged sword*" in that reperfusing ischaemic tissue carries with it an injury component called "*reperfusion injury*".

Ischaemia increases intracellular Calcium, activating a calcium dependent, possibly calmodulin regulated, protease, which converts xanthine dehydrogenase to xanthine oxidase. This forms $\cdot O_2-$ and

H_2O_2. Activated neutrophils have a NADPH dependent oxidase system on their surface. This is normally inactive, but when activated, it catalyses the reduction of O_2 to O_2^- and H_2O_2 (the respiratory burst). The following then occur:

H_2O_2	+ H^+ + Cl^-	--->	H_2O	+ HOCl		
H_2O_2	+ HOCl	--->	H_2O	+ H^+ + Cl^-	+	$^1O_2$
PGG_2	+ Fe^{3+}	--->	PGH_2	+ FeO^{3+}		
PGG_2	+ FeO^{3+}	--->	PGH_2	+ Fe^{3+}	+	$^1O_2$
$\cdot O_2^-$	+ $\cdot O_2^-$ + $2H^+$	--->	H_2O_2	+ O_2		
H_2O_2	+ $\cdot O_2^-$	--->	$\cdot OH$	+ $\cdot OH$	+	$^1O_2$
H_2O_2	+ H_2O_2	--->	$2H_2O$	+ $^1O_2$		

It is now believed that the superoxide ion, $\cdot O_2^-$, is a low reactive species, as is H_2O_2 at physiological concentrations. The dangers of tissue accumulation of superoxide and hydrogen peroxide is that these are precursors for the formation of the $\cdot OH$ radical. This latter radical is very reactive, and results in the formation of singlet oxygen $^1O_2$. This is extremely reactive and severely inhibits a wide variety of enzyme systems [103].

Endotoxin (free or attached to non-viable organisms) which is rapidly released from the gut in significant stress, also activates neutrophils, mast cells, monocytes and endothelium.

Large numbers of neutrophils are normally adherent to endothelium, 2-3 times that of circulating cells. This is a reversible phenomenon, TNF acts on endothelium, causing expression of surface antigens which increase neutrophil binding. Local macrophages produce C5a, IL-1, IL-6, IL-8, TNF, and platelet activating factor (PAF), which all increase leucocyte adherence, chemotaxis and activation. This all results in free radical and protease release with injury to local tissues. Many of these factors feed back positively thereby amplifying the entire process. A new monokine, macrophage inflammatory protein (MIP) also causes the release of IL-1 and IL-6 from macrophages. In addition to endothelial damage, free radicals injure erythrocytes, causing haemolysis and increased osmotic fragility [79 104 105 106 107 108 109 110 111].

Experimentally it is possible to prevent or minimize the reperfusion injury using leucocyte depleted reperfusion of organs. This is further evidence of the role of neutrophil mediated tissue injury [112].

3.5. INTERFERENCE WITH HEALING

As transfusions affect the immune system, and as the immune response has effects on healing, investigations have been performed on the relationship of transfusion and healing. A study from the Netherlands concludes that experimentally at least, blood transfusions are associated with the impairment of the healing of bowel anastomoses and confirms an increase in septic complications [113].

3.6. HYPOTHERMIA AND OXYGEN AFFINITY

Blood is stored at 4^0 C and if unwarmed, causes hypothermia with resultant hypotension, decreased cardiac output, increased peripheral resistance, bradycardia, and reduction of coronary blood flow. Left shift of the oxygen-haemoglobin association curve may reduce tissue oxygenation. This all contributes to myocardial dysfunction and ultimate collapse from hypothermic arrhythmias or ischaemia [114]. Banked blood contains low erythrocytic 2,3-DPG (diphosphoglyceric acid) levels also shifting the oxygen-dissociation curve to the left, with resulting tissue hypoxia [115].

3.7. HYPOCALCAEMIA

The citrate within banked blood chelates Ca^{++} and may cause hypocalcaemia with its consequences [114], in the case of large transfusions.

3.8. CITRATE

In addition to the above, citrate inhibits the enzyme phosphofructokinase, which depletes platelets of energy and causes platelet dysfunction [116].

The citrate may not be metabolized in hypovolaemic and hypothermic states [22]. Banked blood has a pH of 6.6-6.9 because of excess citrate in the anticoagulant [114]. This may be of importance in patients with significant metabolic acidosis and large volume transfusions.

Citrate is metabolized to bicarbonate, and is responsible for post-transfusion metabolic alkalosis. This shifts the oxygen dissociation

curve to the right and may interfere with oxygen delivery in critically ill patients.

3.9. HYPERKALAEMIA

As banked blood ages, there is a slow proportional rise in potassium levels; in fact, potassium levels of 19mM/L are not uncommon [21 32].

3.10. ACIDOSIS

Banked blood often has a pH of 6.6-6.9 because of excess citrate in the anticoagulant. Elevated lactate levels of 120mg/L may occur from cellular metabolism. The low pH also contributes to a moderate reduction of bicarbonate content and increased levels of dissolved carbon dioxide; these all result from the attempt by the stored blood to buffer the fixed acid content of its anticoagulant and from the anaerobic metabolic products arising from the cellular component of the blood [114].

3.11. DELAYED METABOLIC ALKALOSIS

This is a common feature after the transfusion of anticoagulated blood and blood products. The elevated bicarbonate levels are the metabolic products of successful citrate biodegradation by the liver.

3.12. REDUCED OXYGEN TRANSPORT

Homologous banked blood erythrocytes undergo a reduction in 2,3 diphosphoglycerate (2,3 DPG) content. This shifts the oxygen-dissociation curve to the left and is responsible for reduced oxygen off-loading at tissue levels.

Further impairment of tissue oxygenation results from poor myocardial performance owing to hypothermia from cold intravenous fluids. If significant coronary artery disease is present in a trauma patient, this may result in a reduced and fixed cardiac output. In both instances, tissue perfusion cannot be increased to compensate for reduced oxygen off-loading [21 22 32 117].

In the case of salvaged blood, levels of 2,3 DPG are normal. Tissue oxygen off-loading is not a problem with autotransfusion [118].

3.13. IMMEDIATE AVAILABILITY

Blood salvage may start on initial presentation, preoperatively or intraoperatively. The availability is hastened by systems where minimal blood processing is required by the apparatus used [18 22 21 28 60 116].

The use of blood salvage in ruptured ectopic pregnancies is simple, safe, immediately available, and can be practised by the unsophisticated departments [119].

3.14. RELIGIOUS OBJECTIONS

Jehovah's Witnesses refuse banked blood, however, certain types of autotransfusion techniques are acceptable. If a closed system is used in order to maintain blood contact with the patient, the blood is then considered not to have left the host and can be returned to the patient. This technique is not always possible in the emergency situation. This is safer than intraoperative haemodilution [28 29 32 120 121].

3.15. COST EFFECTIVENESS

For low cost and user friendliness in blood salvage, simple apparatus eg Sorenson device, which use inexpensive software and hardware are needed. Even very sophisticated (and expensive) apparatus and software become cost effective above 1 000ml of salvaged blood [18 22 28 29 30 122].

The cost of a unit of blood in public hospitals is about R150.00. The cost of a 2 000ml inner liner for the sorenson device is about R32.50. If 500-2 000ml of blood is salvaged and autotransfused, the cost is about R35.00, as opposed to R150.00 multiplied by the same volume of blood.

In the event of blood salvage by abdominal swab filtration, and/or salvage in a glass transfusovac bottle, the procedure is still cost effective with little extra cost to the hospital or patient.

3.16. HAEMOLYTIC DISEASE OF THE NEWBORN

In an increasing number of cases, haemolytic disease of the newborn is caused by sensitization of the mothers to non-self antigens during previous homologous blood transfusions [31]. It is important to remember that during the resuscitation of young females, if banked blood is ordered in the emergency situation, O, Rh -ve blood be transfused. Autotransfusion in this case would certainly avoid haemolytic disease in the neonate.

3.17. GRAFT VERSUS HOST DISEASE

In susceptible individuals graft vs host disease can be transmitted by viable immunologically competent cells in homologous blood transfusions [123]. This only occurs in patients with immunosuppressive disease or therapy.

4. AUTOTRANSFUSION PROBLEMS; ACTUAL AND POTENTIAL

4.1. BACTERIAL DISSEMINATION AND OTHER SEPSIS

Bacterial contamination in body space blood collections following trauma is always cause for concern. However, the use of banked blood is not without similar risk; Kluge et al found a 4% incidence of positive blood cultures taken from the blood bottles at the time of transfusion [28].

In previous times, bacterial contamination of blood intended for salvage, was an absolute contraindication to its use, both in washing and non-washing cell-savers. This, however, is no longer the case [71].

Many authors report few if any complications with minor contamination, while patients have survived with few problems when transfused with blood grossly contaminated with faeces, so much so that one autotransfusion was terminated only when faecal material occluded the venous cannula [124 125].

Now even the systems that wash the red cells and concentrate them for use can clear more than 80-95% of bacteria; those remaining are largely adsorbed onto the red cell membranes [124 126]. The opinion now is that mild faecal contamination will do no harm if processed by washing apparatus although mild contamination is acceptable in any unprocessed form if severe hypotension exists with no forthcoming blood supplies. Gross faecal contamination is viewed by many as a contraindication; there is therefore no justification for contaminated autotransfusions except in circumstances where the use of such blood will be life saving [28 124 125].

A group from New Orleans compared in a 5 year prospective study, two groups of patients. The first group, consisting of patients autotransfused with washed peritoneal blood contaminated by bowel contents, was compared with a second group with similar injuries transfused with banked blood. No significant septic differences were found between the two groups. This indicates that autotransfusion of washed contaminated blood is a safe procedure [127].

4.2. HAEMATOLOGICAL CONSEQUENCES

It is known, or has been claimed that thrombocytopaenia, leucopaenia, reduced red cell life, haemolysis, DIC, and hypofibrinogenaemia may occur with autotransfusion of salvaged blood.

In one study, haematological effects of experimental haemothorax autotransfusions in dogs were investigated. Of the haemothorax blood itself, the following were found: very prolonged PI and PTT, very low platelets and fibrinogen, mildly elevated FDP, very low coagulation factors, all causing inability to clot. Post-transfusion consequences were as follows: normal PI and PTT, 25% decrease in platelets, 31% fibrinogen drop, 20% reduction in clotting factors. It was concluded that the clotting mechanism was insignificantly affected [128].

4.2.1. THROMBOCYTOPAENIA & PLATELET DYSFUNCTION

Autotransfused blood is never normal. Contact with mesothelial cavities, plastic tubing etc will cause profound deviation from its original makeup.

Thrombocytopaenia is a common finding in individuals after autotransfusion, although this is not a constant feature [122].

The thrombocytopaenia may be mild to profound. Early literature on autotransfusion attributed this to haemodilution [21] by blood containing few platelets [129]; platelet content of salvaged blood ranges from 0-70%. Platelets respond in a fixed and limited manner to a variety of stimuli, many of which, in the autotransfusion context, result from extravascular tissue contact [130]. This triggers a rapid set response comprising morphological alterations, release of granular contents (degranulation), thromboxane A2 production, fibrinogen activation and binding, adhesion to collagen, and aggregation [116].

Although dilution may occur with small or large volumes of autotransfused blood, it is certainly true for large transfusions of banked blood [127]. In addition, large transfusions will also cause dilutional reduction of clotting factors.

Thrombocytopaenia arises from a different mechanism in autotransfusion.

Experiments in dogs being autotransfused show a rapid drop in circulating platelets, to as low as 30%, increasing during the course of transfusions and again declining after the cessation of blood administration. Thereafter, a steady rise occurred with a rebound phenomenon of up to 150% [131]. Baseline levels in humans are reached by 48-72 hours following autotransfusion [18]. Interleukin 6 (IL 6) is a cytokine released by activated monocytes, macrophages, and endothelium. It is a growth factor for megakaryocytes and is probably partially responsible for the later increases in platelets [132].

These rapidly fluctuating levels can only be explained by an effect of the autotransfused blood on the platelets themselves. Certain of the changes may be reversible ie adherence to endothelium and aggregation. Not only are platelet numbers affected, but also platelet function. Within minutes of initiation of autotransfusion, function declined rapidly, to as low as 10%. A parallel sequence of events occurs as in platelet numbers [131 133].

It must be remembered that hypovolaemia shock can itself induce platelet dysfunction [133]. Shock, as previously discussed, causes neutrophil activation and the subsequent release of O_2^-, H_2O_2 and $OH\cdot$. This has an effect of injuring erythrocytes and platelets in the circulation. Steroids have been used for haemotological protection with conflicting results [130 133].

The prolonged thrombocytopaenia is often, however, not present after moderate autotransfusion volumes [28].

The platelet content of salvaged blood is usually low to undetectable because of platelet activation, aggregation etc by extravascular tissue contact and is contributed to the further contact with plastic piping and bags. In addition, a physiological assault on the platelets takes place. This is probably similar to the mechanism causing leucopaenia.

The remaining platelets have abnormal function in that aggregation and degranulation are markedly reduced on platelet stimulation [18].

4.2.2. LEUKOPAENIA

A rapid drop after the start of autotransfusion occurs, returning to normal or supernormal levels. This probably results from margination and clumping which are reversible [131]. A major cause for this is probably activation of the alternate pathway of complement.

As previously discussed under the immunological effects of transfusion, 67% of all leucocytes are marginated. This number increases as a result of activation of endothelium and neutrophils. In addition, increased movement into the tissues occurs with activation, further reducing circulating numbers.

4.2.3. RED CELL SURVIVAL

Although a previous study detected reduced erythrocyte survival, autotransfusion has either a minimal effect on red cell survival or a mild reduction of life span equivalent to that of banked homologous erythrocytes [18 28 131 134].

In contrast to the study showing reduced erythrocyte survival, survival studies on autotransfused cells showed no difference between autotransfused and non-autotransfused erythrocytes [135]. There was no early uptake of labelled cells by the reticuloendothelial system, nor was there evidence of a shortened life-span 24 hours after transfusion.

4.2.4. HAEMOLYSIS AND RENAL FAILURE

Destruction of red blood cells may occur during the salvage of blood, by excessive suction pressure [18], or prolonged contact with extravascular tissue [130], which either lysis the cells, or alters their membrane surface leading to sequestration and destruction in the spleen and other reticuloendothelial organs after autotransfusion.

Newer salvage devices have largely overcome the suction haemolysis problem, by maintaining a negative pressure within a safe range [28].

Until quite recently, it has been accepted that free circulating haemoglobin caused renal failure by precipitation within the renal tubules. This has now been disproved. Free haemoglobin, by itself is not toxic, but the empty erythrocyte membrane shells and the stromal

elements are the cause [18 28]. Pre-existing renal pathology predisposes to renal failure, so the presence of renal insufficiency should be a relative contraindication to unwashed autotransfused blood. This hazard is, of course, eliminated by devices that include erythrocyte washing in the processing of salvaged blood [18 28 29].

4.2.5. DISSEMINATED INTRAVASCULAR COAGULATION (DIC)

During contact with extravascular tissues and to a lesser extent, with the materials forming the autotransfusion device, the blood undergoes numerous physical alterations.

Platelet activation and aggregation releases cytokines, bioamines and procoagulation phospholipids which can activate the clotting system. Contact with nonvascular foreign material, particularly mesothelial lined cavities [18 133], will also activate the coagulation pathway [116].

Haemolysis of erthrocytes releases the phospholipid erythrocytin, which can initiate the clotting system [136 137]. Proteases released by white cells are also capable of activating the clotting pathways [137].

Leukoattractants are also released during the above cellular reactions [18].

Large retroperitoneal haematomas undergoing slow haemolysis are promoters of coagulation pathway activation, thus autotransfusions and homologous transfusions will cause more profound haematological effects [22].

Procoagulants and leucoattractants in autotransfused blood may contribute to ARDS and renal failure seen occasionally in blood salvage [18].

One must bear in mind that transfused banked blood is not in the same physical state as when originally collected, and is probably as capable as salvaged blood in activating the clotting and plasminogen cascades.

During the storage of whole blood, there is degradation of white cells and platelets. This releases multiple potentially dangerous cellular components. Proteolytic enzymes are able to activate the complement pathway. This may also be caused by blood contact with plastic surfaces [138].

Fibrinogen is an acute phase reactant, and levels can become elevated in trauma, sometimes to supernormal levels. The interleukins, in particular IL 6, induce the liver to synthesize acute phase reactants such as fibrinogen, alpha-1-antitrypsin, C reactive protein (CRP), and a reduction of albumin synthesis [132].

Dog experiments indicate that excessive fibrinogen levels predispose to DIC [139]. It was further found that for DIC to develop, 2 conditions were required.

1 slow capillary flow (shock),
2 clot promoting substances in the circulation, all of which have been listed above.

Neither was effective in triggering a DIC alone [139]. Although on paper, a DIC type of picture is present in many patients after autotransfusion, there appears to be no clinical effect in the majority of patients. These alterations, when found, are not an indication for component therapy; replacement therapy is only indicated in the presence of overt bleeding [29].

Hypofibrinogenaemia may occur as a result of a DIC type of picture but is usually transient.

Hypofibrinogenaemia in salvaged blood is the rule, particularly in blood exposed to mesothelium. Owing to as yet unknown mechanisms, mesothelium defibrinates blood in contact with it [18 28 133]. This defibrination occurs rapidly and even 1 hour's contact is usually sufficient to render a haemothorax uncoaguable.

Elevated levels of fibrinogen degradation products (FDP's) occur both in a DIC type of abnormality and in salvaged blood. These substances have an inhibiting effect on platelets, further promoting a bleeding tendency [116]. In the haematological workup of autotransfused patients, the potential abnormalities discussed above need identification [140]. Thus required investigations are:

FBC and platelets,
PI and PTT,
Fibrinogen levels,
FDP levels.

The measurement of FDP's detects breakdown products of fibrinogen, fibrin monomers, and fibrin polymers. More specific information about fibrin breakdown is now available by measuring D-dimer

crosslinked fibrin degradation products (XDP's), which are specific degradation products from insoluble crosslinked fibrin clots [141]. At the time of this study's data collection, XDP's were not yet available.

The initial post-transfusion haematological investigations of this study were collected about 12 hours after transfusion. This ensured that the effects of heparin in salvaged blood would no longer have been present.

In a recent publication, it was found that heparin, administered before induction of shock, maintains microvascular patency in rats, during and after hemorrhage [142]. As a slight digression, this raises significant questions regarding the validity of animal experiments for the investigation of drug therapy for shock, where preheparinization forms part of the trial. Heparin appears to have several potentially useful effects in the trauma situation ie microvascular protection, hypoxia resistance, suppression of noradrenalin levels, endothelial stability, anti-inflammatory and bacteriostatic effects. Our decision to use heparin in this study was an arbitrary decision, not influenced by any reports in the literature.

Even blood salvage in trauma, utilizing a cell washing and packing device, is associated with coagulation defects and reduction in peripheral cell counts [143]. The abnormalities increased in relation to the number of transfused units. There was a sepsis rate of between 25% to 44% in patients who received salvaged washed blood

4.3. ADULT RESPIRATORY DISTRESS SYNDROME

The presence of microaggregated matter in banked and salvaged blood can be associated with various pathophysiological phenomenon and theoretically requires the use of in line blood microfilters to prevent pulmonary microembolic complications [18 21 28 29 122 144 145].

Microemboli may arise as follows:

1 Platelet-fibrin-leukocyte microaggregates,
2 fat microemboli from traumatized tissue,
3 cellular debris from lysed cells,
4 tissue debris from trauma and surgery.

These micro-emboli, if infused, must traverse the pulmonary vasculature and result in diffuse pulmonary micro-embolization.

If present in large quantities, large scale pulmonary microvascular occlusion may occur with potential pulmonary hypertension, dyspnea, hypoxia etc. Once trapped, important pathophysiological consequences occur.

Microemboli, which consist of activated neutrophils, platelets and chemically active clot, are sources of cytokines, complement, and activated clotting factors.

Complement C3a and C5a activate neutrophils, mast cells, endothelial cells, converting hypoxanthine dehydrogenase to xanthine oxidase. This produces toxic products such as superoxide (O_2^-), H_2O_2 and HO·. Histamine, which increases the activity of xanthine oxidase, is also released.

Large numbers of neutrophils, 2-3 times that of circulating cells, are normally adherent to endothelium. This is a reversible phenomenon in that catecholamines and cortisol can demarginate these cells and acutely increase circulating numbers [146].

Cytokines are polypeptides from activated lymphocytes, macrophages, endothelium, fibroblasts, monocytes, lymphocytes, keratinocytes, retinal pigmented epithelium and hepatocytes [153], which stimulate cell growth, initiate immunological, endocrinological and metabolic responses. They are also responsible for the acute-phase responses to trauma/stress. The cytokines interleukins 1, 2, 6 (IL-1, IL-2, IL-6) and tumour necrosis factor (TNF), are particularly involved in this response. The majority of these cytokines have short half-lives of minutes only.

TNF-a and IL-1b are responsible for the non hepatic manifestations of pyrexia, PG synthesis, leucocytosis, tachycardia and increased catabolism. TNF also increases hepatic cell mass, by inducing IL-6, previously called hepatocyte stimulating factor or β_2-interferon. IL-6 is primarily responsible for the hepatic component of the reaction, namely the synthesis of the acute phase reactants. IL-6 and IL-1ß levels correlate with the degree of injury, while IL-6 levels are maximal in patients going on to develop major complications. TNF increases cardiac function initially, and is then associated with depression of cardiac function [147 148 149 150].

TNF acts on endothelium, causing expression of surface antigens like *endothelial leukocyte adhesion molecule 1* (ELAM-1) or MAC-1 which increase neutrophil margination [151] [152]. Endothelial cells when stimulated by cytokines such as TNF and IL-1, increase the expression of their own adhesive factors like *intercellular adhesion molecule 1*. IL-8 is produced by the cells of the alveolar capillary wall (alveolar macrophages, fibroblasts, and endothelial cells), and is a potent neutrophil chemoattractant and activator. IL-8 is produced in response to TNF, IL-1, and bacterial cell wall products [153]. In contrast to the other cytokines, IL-8 has a half-life of several hours. In view of these interactions, it is thus obvious that endothelial cells do not play a passive role in endothelial-neutrophil interactions.

Local activated macrophages produce C5a, IL-1, IL-6, IL-8, TNF, and platelet activating factor (PAF), which all increase leucocyte adherence, chemotaxis and activation. Also released, is leukotriene-B_4 (LTB_4), a parallel, 5-HPETE product of the prostaglandin pathway, a potent neutrophil chemotactic agent. Macrophages are not the only sources of these cytokines.

This all results in free radical, cytokine, prostaglandin product, and protease release with injury to local tissues. Many of these factors feed back positively, thereby amplifying the entire process. A new monokine, macrophage inflammatory protein (MIP) also causes the release of IL-1 and IL-6 from macrophages. In addition to endothelial damage, free radicals injure erythrocytes and platelets [79] [154] [155] [156] [157] [158] [159] [160] [161].

This local interaction of cells, tissues, cytokines and free radicals causes oedema, cellular infiltration, collagen synthesis, which may become permanent leading to fibrosis, and activation of the coagulation, complement, prostaglandin pathways. A host of tissues may be involved in this microvascular damage cascade, however, the lungs are particularly vulnerable in that their anatomic position ensures that they receive the initial and main shower of microaggregates and activated cascades from transfusions.

The "*salvaged blood syndrome*" when it occurs in the lungs, causes ARDS and a range of dysfunction in the kidneys. An identical syndrome may occur with large transfusions of banked blood [18].

Here, histologically, are found marginated leukocytes, fibrin networks and occlusive thrombi in the microcirculation, particularly

of arterioles. Intra-alveolar and perivascular haemorrhages are also found [18 145].

20µ filters are said to prevent these effects. Intraoperative or non-operative salvaged, unfiltered blood rarely causes microembolic problems [29].

4.4. MALIGNANT CELL DISSEMINATION

The presence of a regional malignancy has up to now been regarded as an absolute contraindication to the use of blood salvage techniques.

Salvage of neoplastically contaminated blood will result in malignant cells entering the circulation. There is no evidence of an increased incidence of disseminated disease in patients where this has occurred [28 77 81].

This will occur even with cell washing autotransfusor devices which concentrate the neoplastic cells with the erthrocytes [81]. The feeling is that certainly for genitourinary malignancy, autotransfusion can be safely used [77].

The use of white cell filters with autotransfusion appear to remove malignant cells. This may allow safe autotransfusion in surgery for malignancies [162].

4.5. AIR EMBOLISM

This potentially lethal complication may occur both in autotransfusion and with homologous banked blood transfusions. This occurs as a result of blood being pumped in by pressure, with inattention when the blood bottle is empty. Fatal air embolism was a problem with the Bentley autotransfusor which has subsequently been withdrawn from the market [18 26 28 29 144]. Since then, the occurrence of this has been virtually nonexistent in autotransfusions. Prolonged embolism of microscopic bubbles causes mechanical obstruction of pulmonary vessels and secondary obstruction when thrombosis supervenes; the air-fluid interphase causes activation of the coagulation and complement pathways [144] and SA Diving Med Course. This in turn leads to the ARDS cycle, where leukocytes are attracted, activated, and cause further tissue damage by the release of free radicals.

This produces obstruction to right ventricular outflow. In massive air embolism, cardiac filling is severely interfered with. In man, an estimated 200ml of air, delivered at 70-105 ml per second is required to cause death. In the presence of a patent foramen ovale, volume of as little as 20 ml can be fatal.

4.6. NOT COST EFFECTIVE; TOO SLOW

To be cost effective, a minimum of 1,5-2l of blood needs to be salvaged in advanced autotransfusor devices, in particular, those that wash and subsequently concentrate red cells; below this, the cost of the disposables exceeds that of the salvaged blood, equivalent to an equal banked blood volume. The costs are discussed in section 3.15. The costs are discussed in detail in the discussion at the end of this dissertation. Although some papers regard autotransfusion to be too slow and not cost effective in trauma and non-trauma situations [126], the majority of available literature supports the cost effectiveness and speed of salvaged blood becoming available [18 20 22 29 30 58 60 122 136].

5. CONCLUSIONS

The use of autotransfusion in trauma and non-trauma situations is an effective, efficacious, safe and cost effective method of maintaining cardiovascular homeostasis. A reduction in, or the elimination in the use of banked blood, with all its potential complications, is made possible.

6. PURPOSE / AIMS

To determine the safety, efficacy and cost effectiveness of unprocessed autotransfused blood in the trauma patient or in patients with ruptured ectopic pregnancies.

7. PATIENTS, MATERIALS, METHODS

7.1. PATIENTS

Only patients under the care of the author were entered into a prospective trial, to investigate the effects of autotransfused and banked blood transfusions. Potential candidates were patients with hypovolaemia from blood loss from trauma or ruptured ectopic pregnancies.

7.1.1. PATIENT SELECTION

To reduce the risks of sepsis, no blood collections older than 12 hours would be utilized for autotransfusion.

Any haemoperitoneums associated with small & large bowel penetrating injuries would be discarded.

7.1.1.1. Penetrating Chest Injuries

7.1.1.1.1. Stabs

7.1.1.1.2. Low velocity gunshots

Patients in each group must have a haemothorax, requiring evacuation by intercostal drainage

The stab sites or gunshot wounds must be of the left upper chest & any site of Right chest. This is to ensure that the haemothorax is uncontaminated with bowel contents, which may occur if diaphragmatic penetration has occurred.

7.1.1.2. Penetrating Abdominal injuries

7.1.1.2.1. Stabs

7.1.1.2.2. Low velocity gunshots

Any significant acute haemoperitoneum of adequate volume to justify salvage. Contamination from bowel penetrating injuries must be excluded at laparotomy, before autotransfusion is effected.

7.1.1.3. Blunt injuries of chest with haemothorax

This group of patients should consist primarily of haemothoraces, requiring evacuation, arising from rib fractures. In the case of polytrauma and a haemothorax, a normal and non tender abdomen is mandatory. This is to avoid contamination by bowel contents in the case of a ruptured diaphragm with possible hollow visceral injuries.

7.1.1.4. Blunt abdominal haemoperitoneums

This group of patients comprises those with injuries arising from assaults or polytrauma. In both instances, contamination from perforated bowel must be excluded at laparotomy before autotransfusion is commenced.

7.1.1.5. Acute ruptured ectopic pregnancy

Significant volumes of intra-peritoneal blood, arising from the rupture of an ectopic pregnancy, may be salvaged and subsequently autotransfused.

7.1.2. PATIENT EXCLUSION

7.1.2.1. Penetrating Chest Injuries

7.1.2.1.1. Stabs

7.1.2.1.2. Low velocity gunshots

Small haemothoraces not requiring evacuation, or any haemothorax older than 12 hours requiring evacuation. In this case, the blood is likely to be contaminated as a result of the penetrating injury.

Stab sites or gunshot wounds lying within the left lower chest area. The haemothorax may be contaminated with bowel contents, in the case of diaphragmatic penetration.

7.1.2.2. Penetrating Abdominal Injuries

7.1.2.2.1. Stabs

7.1.2.2.2. Low velocity gunshots:

7.1.2.2.3. High velocity gunshot injuries:

Any haemoperitoneums of adequate volume to justify salvage associated with penetrating bowel injuries.

HVGSW's of the abdomen should also be excluded. There is a high incidence of bowel injuries, and owing to the temporary cavitation effect, contamination by bacteria, environmental and tissue debris is common.

7.2. METHODS

7.2.1. MATERIALS / EQUIPMENT

7.2.1.1. The autotransfusion system

A Sorenson cell saver was acquired by Hillbrow Hospital for blood salvage, but simpler and cheaper apparatus were equally effective.

7.2.1.1.1. Sorenson Autotransfusion Device

This comprises a disposable sterile inner liner (reservoir), within a rigid reusable outer cylinder.

Disposable suction tubing with an inbuilt venturi device for anticoagulation is attached to the reservoir.

Wall vacuum operates the unit and salvaged blood is filtered through an in line 170µ screen filter in the reservoir. The full inner liner is removed, suspended and the blood transfused via an administration set spiked into the built in port, on the inferior portion of the reservoir. This system allows whole blood to be collected, anticoagulated, filtered and, retransfused.

This device can be used for body cavity blood salvage in the emergency room or intraoperatively.

7.2.1.1.2. Transfusovac bottles

50ml of sterile saline is in the intercostal bottle; 500u of heparin are added and once full, the blood decanted into an emptied transfusovac bottle. The sterile top is replaced and the blood is then autotransfused.

7.2.1.1.3. Vacolitre bag

Gross haemoperitoneums diagnosed by peritoneal lavage are drained into an emptied vacolitre bag and 500u heparin added. The full bag is then transfused once bowel integrity has been established.

7.2.1.1.4. Abdominal Swab Filtration

Gross haemoperitoneums at laparotomy were ladled into a sterile bowl, filtered through an abdominal swab, decanted into an emptied, sterile transfusovac bottle and 500u heparin added; the blood was then autotransfused if no contamination is present.

A microfilter was used during the administration of the salvaged blood.

Only blood collections less than 12 hours old and where contamination was excluded, were used for this procedure.

7.2.2. DATA COLLECTION

Data collection: Refer to protocol page. Every possible effort was made to record all preoperative results, preop status and injuries with intraop findings.

All banked & autotransfused blood was recorded with the type of blood salvage and anticoagulant used.

All postoperative haematology & complications were firmly recorded.

7.2.2.1. ADMISSION

If circumstances permitted, acute patients would have the following haematological investigations performed on admission:

Haemoglobin (Hb),
White Cell Count (WCC),
Platelets (PLT),
Prothrombin Index (PI),
Partial Thromboplastin Time (PTT).

7.2.2.2. POST TRANSFUSION

The morning following the transfusions, further specimens would be drawn to determine the haematological effects of autotransfused and banked blood.

Hb, WCC, PLT, PI, PTT, FDP's,
Haptoglobin, Haemopexin, and Fibrinogen Levels,
U&E and Creatinine

7.2.2.3. SALVAGED BLOOD COMPOSITION

Whenever possible, the following investigations would be performed on the autotransfused blood itself:

Hb, WCC, PLT's, PI, PTT, FDP's, and Fibrinogen levels.

7.2.3. THERAPEUTIC

All autotransfusions would be covered with Penicillin G, 2.5MU, intravenously, in non-allergic patients.

Platelets and Fresh Frozen Plasma would be administered to patients after every 6 units of transfusion.

Protamine would be administered to reverse coagulopathies induced by heparin.

7.3. RUPTURED ECTOPIC PREGNANCY

7.3.1. DIAGNOSTIC

As an alternative diagnostic measure, percutaneous diagnostic peritoneal lavage (DPL) would be employed.

Identical blood testing to the above patients to be performed.

7.3.2. THERAPEUTIC

As in the above section, THERAPEUTIC, 7.2.3.

7.4. TECHNICAL PROBLEMS

1 As the author was virtually the only person interested and trained in its use, it was not possible with dire emergencies, to set up the apparatus and be involved in the management of a patient.

2 Difficulty in persuading staff to use sterile saline and not water in the intercostal drain bottle to prevent haemolysis of salvaged blood.

7.4. TECHNICAL PROBLEMS

1 As the author was virtually the only person interested and trained in its use, it was not possible with dire emergencies, to set up the apparatus and be involved in the management of a patient.

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8. RESULTS OF AUTOTRANSFUSION STUDY

Between January 1984 and December 1990, a prospective trial on autotransfusion was performed on 77 patients at the Hillbrow, Coronation and Shongwe Hospitals.

Shongwe is a rural hospital in the Nkomazi district of Kangwane, a self-governing homeland, bordering South Africa, Swaziland, and Mozambique.

The hospital is 10 km from the Swazi border, 100 km from Nelspruit, and 35 km from Malalane.

It supplies medical services to over 300 000 people. It has 450 inpatients and 150 outpatients per day. The nearest referring institution which covered my requirements was 450 kilometers away. It has an in house first world laboratory facility, good X-ray and sonar facilities. A CT scanner service was available nearby when required. Modern theatres are present with a three bed ICU in the theatre complex. A modern endoscopy service also operated from this complex.

8.1. PATIENT NUMBERS

Of the 77 study patients, 65 were involved in penetrating or blunt injuries, and 12 were ruptured ectopic pregnancies.

8.2. SEX

There were 48 males and 17 females. The trauma group comprised 48 males and 5 females. The ectopic group obviously consisted of 12 females.

8.3. AGE

The ages ranged from 16 years to 65 years. The average age was 29.91 years. The 26-30 year group comprised the largest number of patients.

8.4. GROUPS

The patients were divided into four groups:

1 banked blood only (controls),
2 autotransfused blood only,
3 combined banked and autotransfusion,
4 ruptured ectopic pregnancies.

8.5. TRIAL GROUP

There were 44 patients who were autotransfused. Of these, 27 received autotransfused blood only, while 17 had a combination of banked blood and autotransfused blood.

8.6. CONTROL GROUP

Twenty one patients were used as controls in that only banked blood was used.

8.7. HOSPITAL AND PATIENT DISTRIBUTION

	Total	Trauma	Ectopics
Hillbrow	64	63	1
Coronation	1	1	0
Shongwe	12	1	11

9. ANALYSIS OF RESULTS

9.1. BANKED BLOOD ONLY, TRAUMA

There were 21 patients who functioned as controls in that all transfusions were of banked blood.

9.1.1. TRANSFUSION VOLUMES

Name	n	%	Lo	Hi	Anticoagulation
BANKED	21	100	1	17	ACD
* ONE	1	4.76	1	1	ACD
* TWO	7	33.33	2	2	ACD
* THREE	4	19.05	3	3	ACD
* FOUR	3	14.29	4	4	ACD
* FIVE	1	4.76	5	5	ACD
* SIX	1	4.76	6	6	ACD
* SEVEN	1	4.76	7	7	ACD
* EIGHT	1	4.76	8	8	ACD
* TEN	1	4.76	10	10	ACD
* SEVENTEEN	1	4.76	17	17	ACD

9.1.2. INJURIES, SURGERY, FINDINGS

Pt	CHEST	ABDOMEN	OTHER	OPERATION	FINDINGS
9	0	0	head/#'s	#.fix.	0
39	0	0	head	0	0
48	Blunt	0	# ribs	0	0
49	GSW	0	0	0	0
50	Stab	0	0	0	0
51	0	Blunt	0	Lap.	Liver
52	Stab	0	0	0	0
53	Blunt	0	0	0	0
54	0	Blunt	0	Lap.	Spleen
66	0	GSW	0	Lap.	Bowel+bladder
67	Stab	0	0	0	0
68	0	Blunt	0	Lap.	Spleen
69	0	GSW	0	Lap.	GIT,livr,kidny
70	GSW	GSW	0	Lap.	GIT,liver
71	0	Blunt	0	Lap.	Liver
72	0	Blunt	0	Lap.	bowel
73	0	Blunt	0	Lap.	Liver
74	0	0	#,other	#.fix.	0
75	0	Stab	0	Lap.	liver
76	0	GSW	0	Lap.	bowel
77	0	0	#,other	#.fix.	0

9.1.3.3. Haptoglobins, Haemopexins, Fibrinogen

Pt	HAPTOGLOB	HAEMOPEX	FIBRINO1	FIBRINO2
9	100	427	185	0
39	90	510	280	328
48	122	629	260	282
49	76	595	283	315
50	40	850	340	312
51	122	629	629	0
	140	580	221	272
	50	612	178	270
	0	890	270	320
		728	265	0
		701	358	0
		884	300	250
		207	1250	0
		569	145	659
		820	424	288
		483	510	0
		707	346	0
		634	256	0
		535	298	0
		650	147	0
		563	343	0

ON HB

formation

Min	Max	O/Range	Missing
0	99	0	0
1	5		
6	10		
11	14		
15	20		

Lo	Hi	Mean	S.D.	S.E.M
7	14	11.62	1.962	0.428
7	10	9.29	1.254	0.4738
12	14	12.79	0.893	0.2366

9.1.3. HAEMATOLOGY RESULTS

9.1.3.1. Hb, WCC, PLT

Pt	HB0	HB1	HB2	WCC0	WCC1	PLT0	PLT1
9	10	12	0	22	11	207	0
39	8	9	0	28	17	380	363
48	13	14	0	17	16	160	172
49	14	14	0	16	11	120	154
50	14	14	0	13	13	160	176
51	12	11	0	25	15	217	246
52	12	12	0	10	9	385	350
53	10	10	0	11	13	124	252
54	12	11	0	14	10	250	160
66	12	14	0	15	7	215	0
67	7	10	0	15	12	67	197
68	10	9	0	17	10	37	74
69	13	13	0	16	10	85	123
70	12	11	0	24	21	159	0
71	10	10	0	26	19	315	0
72	14	9	0	17	15	287	57
73	12	0	0	18	0	326	0
74	10	8	0	15	5	191	53
75	14	0	0	10	0	100	0
76	13	12	0	18	1	108	0
77	12	11	0	17	14	120	0

9.1.3.2. PI, PTT, FDP's

Pt	PI0	PI1	PI2	PTT0	PTT1	FDP
9	81	0	0	48	0	10-40
39	99	99	0	35	32	10-40
48	99	99	0	34	33	10-40
49	74	86	94	47	35	10-40
50	97	99	0	45	31	<10
51	80	91	98	32	35	10-40
52	75	86	94	46	33	<10
53	67	81	96	56	31	120-240
54	71	85	94	42	30	<10
66	72	83	94	63	44	80-120
67	81	93	0	43	36	<10
68	71	77	0	65	48	10-40
69	69	66	70	70	45	80-120
70	75	86	0	54	0	80-120
71	61	82	0	41	35	<10
72	83	33	0	77	0	80-120
73	83	0	0	39	0	10-40
74	58	0	0	42	0	80-120
75	52	0	0	50	0	80-120
76	62	0	0	57	0	10-40
77	81	0	0	43	0	10-40

9.1.3.3. Haptoglobins, Haemopexins, Fibrinogen

Pt	HAPTOGLOB	HAEMOPEX	FIBRINO1	FIBRINO2
9	100	427	185	0
39	90	510	280	328
48	122	629	260	282
49	76	595	283	315
50	40	850	340	312
51	122	629	629	0
52	140	580	221	272
53	60	612	178	270
54	0	890	270	320
66	70	728	265	0
67	100	701	358	0
68	60	884	300	250
[illegible]	30	207	1250	0
[illegible]	90	569	145	659
71	280	820	424	288
72	41	483	510	0
73	50	707	346	0
74	30	634	256	0
75	20	535	298	0
76	50	650	147	0
77	59	563	343	0

9.1.4. ADMISSION HB

9.1.4.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
HB0		21	0	99	0	0
*	VLO	0	1	5		
*	LO	7	6	10		
*	N	14	11	14		
*	HI	0	15	20		

9.1.4.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HB0	21	100	7	14	11.62	1.962	0.428
* VLO	0	0					
* LO	7	33.33	7	10	9.29	1.254	0.4738
* N	14	66.67	12	14	12.79	0.893	0.2386
* HI	0	0					

9.1.5. FIRST POST-TRANSFUSION HB

9.1.5.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
HB1		19	0	99	0	0
*	VLO	0	1	5		
*	LO	7	6	10		
*	N	12	11	14		
*	HI	0	15	20		

9.1.5.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HB1	19	100	8	14	11.26	1.91	0.4382
* VLO	0	0					
* LO	7	36.84	8	10	9.29	0.756	0.2857
* N	12	63.16	11	14	12.42	1.311	0.3786
* HI	0	0					

9.1.5.3. Effect of Transfusion Volume on Hb

Group name	n	%	Mean	Std.Error
ONE	1	5	12	0
TWO	7	37	11.9	0.86
THREE	3	16	12	1.15
FOUR	3	16	10.7	0.33
FIVE	1	5	13	0
SIX	1	5	8	0
SEVEN	1	5	10	0
EIGHT	1	5	11	0
TEN	EXCLUDED - 0 Cases			
SEVENTEEN	1	5	9	0
TOTAL	19	100	11.3	0.44

Significance of BANKED group effect on HB is 1.108142

9.1.6. ADMISSION WCC

9.1.6.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
WCC0		21	0	99	0	0
*	VLO	0	1	2		
*	LO	0	2.1	4		
*	N	3	4.1	11		
*	HI	13	11.1	20		
*	VHI	5	20.1	99		

9.1.6.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
WCC0	21	100	10	28	17.33	5.063	1.1048
* VLO	0	0					
* LO	0	0					
* N	3	14.29	10	11	10.33	0.577	0.3333
* HI	13	61.9	13	18	16	1.528	0.4237
* VHI	5	23.81	22	28	25	2.236	1

9.1.6.3. Effect of Transfusion Volume on WCC

Group name	n	%	Mean	Std.Error
ONE	1	5	10	0
TWO	7	33	19.1	2.02
THREE	4	19	16.5	2.33
FOUR	3	14	21.3	3.71
FIVE	1	5	16	0
SIX	1	5	15	0
SEVEN	1	5	15	0
EIGHT	1	5	17	0
TEN	1	5	10	0
SEVENTEEN	1	5	17	0
TOTAL	21	100	17.3	1.1

Significance of BANKED group effect on WCC is 1.173992

9.1.7. POST-TRANSFUSION WCC

9.1.7.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
WCC1		19	0	99	0	0
*	VLO	1	1	2		
*	LO	0	2.1	4		
*	N	8	4.1	11		
*	HI	9	11.1	20		
*	VHI	1	20.1	99		

9.1.7.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
WCC1	19	100	1	21	12.05	4.79	1.0988
* VLO	1	5.26	1	1	1		
* LO	0	0					
* N	8	42.11	5	11	9.12	2.1	0.7425
* HI	9	47.37	12	19	14.89	2.205	0.7349
* VHI	1	5.26	21	21	21		

9.1.7.3. Effect of Transfusion Volume on WCC

Group name	n	%	Mean	Std.Error
ONE	1	5	9	0
TWO	7	37	12.6	2.07
THREE	3	16	10.3	1.76
FOUR	3	16	16.7	3.38
FIVE	1	5	10	0
SIX	1	5	5	0
SEVEN	1	5	12	0
EIGHT	1	5	14	0
TEN	EXCLUDED - 0 Cases			
SEVENTEEN	1	5	10	0
TOTAL	19	100	12.1	1.1

Significance of BANKED group effect on WCC is 1.209324

9.1.8. FIRST POST-TRANSFUSION PLT

9.1.8.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
PLT0		21	0	999	0	0
*	LO	8	1	140		
*	N	13	141	400		
*	HI	0	401	999		

9.1.8.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PLT0	21	100	37	385	191.1	101.039	22.0485
* LO	8	38.1	37	124	95.12	30.549	10.8009
* N	13	61.9	159	385	250.15	80.942	22.4493
* HI	0	0					

9.1.8.3. Effect of Transfusion Volume on PLT

Group name	n	%	Mean	Std.Error
ONE	1	5	385	0
TWO	7	33	204.6	37.22
THREE	4	19	218	41.46
FOUR	3	14	241.3	45.24
FIVE	1	5	85	0
SIX	1	5	191	0
SEVEN	1	5	67	0
EIGHT	1	5	120	0
TEN	1	5	100	0
SEVENTEEN	1	5	37	0
TOTAL	21	100	191.1	22.05

Significance of BANKED group effect on PLT is 0.253505

9.1.9. SECOND POST-TRANSFUSION PLT

9.1.9.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
PLT1		13	0	999	0	0
*	LO	4	1	140		
*	N	9	141	400		
*	HI	0	401	999		

9.1.9.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PLT1	13	100	53	363	182.85	99.587	27.6206
* LO	4	30.77	53	123	76.75	32.149	16.0747
* N	9	69.23	154	363	230	79.745	26.5816
* HI	0	0					

9.1.9.3. Effect of Transfusion Volume on PLT

Group name	n	%	Mean	Std.Error
ONE	1	8	350	0
TWO	6	46	194.7	41.81
THREE	1	8	252	0
FOUR	1	8	160	0
FIVE	1	8	123	0
SIX	1	8	53	0
SEVEN	1	8	197	0
EIGHT	EXCLUDED - 0 Cases			
TEN	EXCLUDED - 0 Cases			
SEVEN TEN	1	8	74	0
TOTAL	13	100	182.8	27.62

Significance of RANKED group effect on PLT is 1.045045

9.1.10. FIRST POST-TRANSFUSION PI

9.1.10.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
PI0		21	0	99	0	0
*	VLO	0	1	50		
*	LO	18	51	89		
*	N	3	90	100		

9.1.10.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PI0	21	100	52	99	75.76	12.668	2.7645
* VLO	0	0					
* LO	18	85.71	52	83	72	9.152	2.1572
* N	3	14.29	97	99	98.33	1.155	0.6667

9.1.10.3. Effect of Transfusion Volume on PI

Group name	n	%	Mean	Std.Error
ONE	1	5	75	0
TWO	7	33	84.9	5.38
THREE	4	19	75.7	3.77
FOUR	3	14	69	4.16
FIVE	1	5	69	0
SIX	1	5	58	0
SEVEN	1	5	81	0
EIGHT	1	5	81	0
TEN	1	5	52	0
SEVENTEEN	1	5	71	0
TOTAL	21	100	75.8	2.76

Significance of BANKED group effect on PI is 0.290504

9.1.11. SECOND POST-TRANSFUSION PI

9.1.11.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
PI1		15	0	99	0	0
*	VLO	1	1	50		
*	LO	9	51	89		
*	N	5	90	100		

9.1.11.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PI1	15	100	33	99	83.07	16.46	4.2489
* VLO	1	6.67	33	33	33		
* LO	9	60	66	86	81.33	6.481	2.1602
* N	5	33.33	91	99	96.2	3.899	1.7436

9.1.11.3. Effect of Transfusion Volume on PI

Group name	n	%	Mean	Std.Error
ONE	1	7	86	0
TWO	6	40	84.5	10.53
THREE	2	13	82	1
FOUR	3	20	84.3	1.2
FIVE	1	7	66	0
SIX	EXCLUDED - 0 Cases			
SEVEN	1	7	93	0
EIGHT	EXCLUDED - 0 Cases			
TEN	EXCLUDED - 0 Cases			
SEVENTEEN	1	7	77	0
TOTAL	15	100	83.1	4.25

Significance of BANKED group effect on PI is 1.43872

9.1.12. THIRD POST-TRANSFUSION PI

9.1.12.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
PI2		7	0	99	0	0
*	VLO	0	1	50		
*	LO	1	51	89		
*	N	6	90	100		

9.1.12.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PI2	7	100	70	98	91.43	9.572	3.6178
* VLO	0	0					
* LO	1	14.29	70	70	70		
* N	6	85.71	94	98	95	1.673	0.6831

9.1.12.3. Effect of Transfusion Volume on PI

Group name	n	%	Mean	Std.Error
ONE	1	14	94	0
TWO	2	29	96	2
THREE	2	29	95	1
FOUR	1	14	94	0
FIVE	1	14	70	0
SIX	EXCLUDED - 0 Cases			
SEVEN	EXCLUDED - 0 Cases			
EIGHT	EXCLUDED - 0 Cases			
TEN	EXCLUDED - 0 Cases			
SEVENTEEN	EXCLUDED - 0 Cases			
TOTAL	7	100	91.4	3.62

Significance of BANKED group effect on PI is 0.016051

9.1.13. FIRST POST-TRANSFUSION PTT

9.1.13.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT0		21	0	99	0	0
*	NORMAL	4	28	39		
*	ABN	17	40	99		

9.1.13.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PTT0	21	100	32	77	49	12.021	2.6232
* NORMAL	4	19.05	32	39	35	2.944	1.472
* ABN	17	80.95	41	77	52.29	10.884	2.6399

9.1.13.3. Effect of Transfusion Volume on PTT

Group name	n	%	Mean	Std.Error
ONE	1	5	46	0
TWO	7	33	46.7	6.06
THREE	4	19	51.5	5.17
FOUR	3	14	45.7	4.18
FIVE	1	5	70	0
SIX	1	5	42	0
SEVEN	1	5	43	0
EIGHT	1	5	43	0
TEN	1	5	50	0
SEVENTEEN	1	5	65	0
TOTAL	21	100	49	2.62

Significance of group effect on PTT0 is 1.302169

9.1.14. SECOND POST-TRANSFUSION PTT

9.1.14.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT1		13	0	99	0	0
*	NORMAL	10	28	39		
*	ABN	3	40	99		

9.1.14.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PTT1	13	100	30	48	36	5.859	1.6251
* NORMAL	10	76.92	30	36	33.1	2.079	0.6574
* ABN	3	23.08	44	48	45.67	2.082	1.2019

9.1.15. POST-TRANSFUSION FDP

9.1.15.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
FDP		21	0	99	0	0
*	<10	5	1	1		
*	10-40	9	2	2		
*	40-80	0	4	4		
*	80-120	6	8	8		
*	120-240	1	16	16		
*	>240	0	32	32		

9.1.15.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FDP	21	100	1	16	4.14	4.004	0.8737
* <10	5	23.81	1	1	1		
* 10-40	9	42.86	2	2	2		
* 40-80	0	0					
* 80-120	6	28.57	8	8	8		
*120-240	1	4.76	16	16	16		
* >240	0	0					

9.1.16. POST-TRANSFUSION HAPTOGLOBINS

9.1.16.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
HAPTOGLOB		20	0	999	0	0
*	VLO	7	1	50		
*	LO	7	51	99		
*	N	6	100	300		
*	HI	0	301	999		

9.1.16.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HAPTOGLOB	20	100	20	280	81.5	57.565	12.8719
* VLO	7	35	20	50	37.29	11.176	4.2242
* LO	7	35	59	90	72.14	13.692	5.1752
* N	6	30	100	280	144	68.34	27.8998
* HI	0	0					

9.1.16.3. Effect of Transfusion Volume on Haptoglobins

Group name	n	%	Mean	Std.Error
ONE	1	5	140	0
TWO	7	35	77.3	13.46
THREE	4	20	70	10.8
FOUR	2	10	185	95
FIVE	1	5	30	0
SIX	1	5	30	0
SEVEN	1	5	100	0
EIGHT	1	5	59	0
TEN	1	5	20	0
SEVENTEEN	1	5	60	0
TOTAL	20	100	81.5	12.87

Significance of group effect on HAPTOGLOB is 0.276547

9.1.17. POST-TRANSFUSION HAEMOPEXINS

9.1.17.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
HAEMOPEX		21	0	999	0	0
*	LO	3	1	500		
*	N	18	501	999		

9.1.17.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HAEMOPEX	21	100	207	890	628.71	160.469	35.0171
* LO	3	14.29	207	483	372.33	145.895	84.2325
* N	18	85.71	510	890	671.44	119.404	28.1438

9.1.17.3. Effect of Transfusion Volume on Haemopexins

Group name	n	%	Mean	Std.Error
ONE	1	5	580	0
TWO	7	33	620.9	45.11
THREE	4	19	610.5	68.64
FOUR	3	14	759.7	97.45
FIVE	1	5	207	0
SIX	1	5	634	0
SEVEN	1	5	701	0
EIGHT	1	5	563	0
TEN	1	5	535	0
SEVENTEEN	1	5	884	0
TOTAL	21	100	628.7	35.02

Significance of group effect on HAEMOPEX is 0.148855

9.1.18. FIRST POST-TRANSFUSION FIBRINOGEN

9.1.18.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO1		21	0	9999	0	0
*	LO	4	1	199		
*	N	13	200	400		
*	HI	4	401	9999		

9.1.18.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FIBRINO1	21	100	145	1250	347.05	236.728	51.6582
* LO	4	19.05	145	185	163.75	20.71	10.3552
* N	13	61.9	221	358	293.85	41.865	11.6111
* HI	4	19.05	424	1250	703.25	374.065	187.0327

9.1.18.3. Effect of Transfusion Volume on Fibrinogen

Group name	n	%	Mean	Std.Error
ONE	1	5	221	0
TWO	7	33	349.9	62.15
THREE	4	19	243.5	39.46
FOUR	3	14	279.7	80.69
FIVE	1	5	1250	0
SIX	1	5	256	0
SEVEN	1	5	358	0
EIGHT	1	5	343	0
TEN	1	5	298	0
SEVENTEEN	1	5	300	0
TOTAL	21	100	347	51.66

Significance of group effect on FIBRINO1 is 0.007437 (Significant)

9.1.19. SECOND POST-TRANSFUSION FIBRINOGEN

9.1.19.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO2		10	0	9999	0	0
*	LO	0	1	199		
*	N	9	200	400		
*	HI	1	401	9999		

9.1.19.2. Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FIBRINO2	10	100	250	659	329.6	118.474	37.4647
* LO	0	0					
* N	9	90	250	328	293	26.842	8.9474
* HI	1	10	659	659	659		

9.1.19.3. Effect of Transfusion Volume on Fibrinogen

Group name	n	%	Mean	Std.Error
ONE	1	10	272	0
TWO	4	40	309.2	9.72
THREE	1	10	270	0
FOUR	3	30	422.3	18.69
FIVE	EXCLUDED - 0 Cases			
SIX	EXCLUDED - 0 Cases			
SEVEN	EXCLUDED - 0 Cases			
EIGHT	EXCLUDED - 0 Cases			
TEN	EXCLUDED - 0 Cases			
SEVENTEEN	1	10	250	0
TOTAL	10	100	329.6	37.46

Significance of group effect on FIBRINO2 is 1.197741

9.2. AUTOTRANSFUSED BLOOD ONLY, TRAUMA

This group comprised 27 patients.

9.2.1. TRANSFUSION VOLUME

Units	n	%	Anticoagulant
1	1	3.7	heparin
2	13	48.15	heparin
3	5	18.52	heparin
4	4	14.81	heparin
5	2	7.41	heparin
6	1	3.7	heparin
7	1	3.7	heparin

9.2.2. INJURIES, SURGERY, FINDINGS

Pt	CHEST	ABDOMEN	OTHER	OPERATION	FINDINGS
2	0	blunt	0	lap	spleen
3	blunt	0	#ribs	0	0
4	GSW	0	#ribs	0	0
11	stab	0	head	lap	spln+Diaf.
12	stab	0	0	0	0
13	stab	0	0	0	0
15	0	blunt	0	lap	liver
17	stab	0	0	0	0
20	0	blunt	0	lap	spleen
21	stab	0	0	0	0
22	stab	0	0	0	0
24	0	blunt	0	lap	spleen
25	stab	0	0	0	0
28	stab	0	0	0	0
29	0	blunt	0	lap	livr/spln
30	stab	0	0	thoracot	lung
32	stab	stab	0	lap	liver
33	0	blunt	0	lap	spleen
34	0	blunt	0	lap	spleen
35	0	stab	0	lap	liver
36	0	blunt	0	lap	spleen
37	0	stab	0	lap	liver
38	blunt	blunt	rib+other	lap	spleen
41	stab	0	0	0	0
42	stab	0	0	thoracot	lung
44	stab	0	0	0	0
45	stab	0	0	0	0

9.2.3. HAEMATOLOGY RESULTS

9.2.3.1. Haemoglobin, platelets, white cell count

Pt	HB0	HB1	HB2	PLT0	PLT1	WCC0	WCC1
2	6	12	0	100	0	17	16
3	13	14	0	160	0	17	16
4	14	14	0	120	0	16	0
11	8	8	0	132	187	26	12
12	14	15	0	190	212	20	14
13	14	14	0	160	193	13	13
15	12	11	0	217	255	25	15
17	9	11	0	135	163	28	9
20	12	13	0	54	96	8	7
21	9	11	0	142	176	15	9
22	11	10	0	210	224	18	10
24	11	12	0	228	274	17	12
25	14	17	15	295	279	19	10
28	13	14	0	120	222	13	19
29	16	13	0	180	170	18	12
30	10	12	0	120	174	20	13
32	10	13	0	144	190	20	12
33	16	12	0	150	178	19	10
34	11	14	0	210	272	23	20
35	13	13	0	162	186	18	12
36	14	13	0	160	183	20	13
37	11	11	0	150	174	17	11
38	11	8	0	81	57	10	2
41	11	13	0	210	230	10	8
42	12	12	0	383	403	10	11
44	12	12	0	186	248	16	21
45	14	14	0	230	214	9	3

9.2.3.2. PI, PTT, FDP

Pt	PI0	PI1	PI2	PTT0	PTT1	FDP	AUTOXFUSE
2	10	0	0	40	0	10-48	4
3	1	0	0	45	0	10-48	2
4	74	0	0	47	0	10-48	2
11	81	88	93	39	31	<10	6
12	88	93	95	33	31	<10	2
13	97	99	0	45	36	<10	2
15	80	86	93	32	36	10-48	2
17	79	87	93	37	35	10-48	2
20	70	91	0	42	36	10-48	3
21	99	99	0	30	34	10-48	3
22	55	86	94	31	33	<10	3
24	79	90	96	91	44	<10	2
25	94	74	89	31	40	120-240	2
28	99	98	0	30	31	<10	2
29	99	0	0	32	0	120-240	4
30	92	97	0	34	32	10-48	2
32	86	94	95	35	30	10-48	4
33	92	99	0	34	32	10-48	3
34	99	99	0	28	30	80-120	5
35	92	99	0	32	34	80-120	4
36	99	99	0	34	32	10-48	5
37	99	0	0	34	0	10-48	2
38	55	40	0	45	76	>240	7
41	86	92	96	38	31	<10	3
42	75	88	96	46	33	<10	1
44	86	92	97	34	31	<10	2
45	85	93	99	37	36	<10	2

9.2.3.3. Haptoglobins, Haemopexins, Fibrinogen

Pt	HAPTOGLOB	HAEMOPEX	FIBRINO1	FIBRINO2	BANKED
2	150	840	126	0	0
3	122	629	240	0	0
4	76	595	283	280	0
11	110	646	198	312	0
12	100	662	227	235	0
13	40	850	340	372	0
15	122	629	300	346	0
17	90	760	210	281	0
20	130	442	250	270	0
21	136	520	80	261	0
22	310	615	262	273	0
24	320	826	222	273	0
25	180	523	240	260	0
28	300	660	240	280	0
29	120	720	200	320	0
30	110	800	260	294	0
32	150	680	290	341	0
33	130	720	260	304	0
34	210	521	495	538	0
35	80	821	372	360	0
36	120	756	190	224	0
37	120	687	220	255	0
38	0	0	0	0	0
41	100	680	267	280	0
42	140	580	221	272	0
44	90	510	205	222	0
45	150	610	340	372	0

9.2.4. FIRST POST-TRANSFUSION PLATELET COUNT

9.2.4.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PLTO		27	0	999	0	0
*	LO	8	1	140		
*	N	19	141	400		
*	HI	0	401	999		

9.2.4.2. Platelet Count

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PLTO	27	100	54	383	171.44	66.43	12.7845
* LO	8	29.63	54	135	107.75	27.891	9.8611
* N	19	70.37	142	383	198.26	59.24	13.5907
* HI	0	0					

9.2.4.3. Effect of autotransfusion volume on PLT

Group name	n	%	Mean	Std.Error
ONE	1	4	383	0
TWO	13	48	177.8	14.76
THREE	5	19	153.2	28.66
FOUR	4	15	146.5	17.15
FIVE	2	7	185	25
SIX	1	4	132	0
SEVEN	1	4	81	0
TOTAL	27	100	171.4	12.78

Significance of group effect on PLT is 0.013958 is (Significant)

9.2.5. SECOND POST-TRANSFUSION PLATELET COUNT

9.2.5.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PLT1		24	0	999	0	0
*	LO	2	1	140		
*	N	21	141	400		
*	HI	1	401	999		

9.2.5.2. Platelet Count

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PLT1	24	100	57	403	206.67	66.405	13.5549
* LO	2	8.33	57	96	76.5	27.577	19.5
* N	21	87.5	163	279	209.71	37.438	8.1697
* HI	1	4.17	403	403	403		

9.2.5.3. Effect of autotransfusion volume on PLT

Group name	n	%	Mean	Std.Error
ONE	1	4	403	0
TWO	11	46	218.9	12.32
THREE	5	21	180.8	23.99
FOUR	3	13	182	6.11
FIVE	2	8	227.5	44.5
SIX	1	4	187	0
SEVEN	1	4	57	0
TOTAL	24	100	206.7	13.55

Significance of group effect on PLT is 0.001562=(Significant)

9.2.6. ADMISSION WHITE CELL COUNT

9.2.6.1. Variable information

Name	group	n	Min	Max	O/Range	Missing
WCC0		27	0	99	0	0
	VLO	0	1	2	0	0
	LO	0	2.1	4		
	N	5	4.1	11		
	HI	18	11.1	20		
	VHI	4	20.1	99		

9.2.6.2. WCC Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
WCC0	27	100	8	28	17.11	5.109	0.9832
* VLO	0	0					
* LO	0	0					
* N	5	18.52	8	10	9.4	0.894	0.4
* HI	18	66.67	13	20	17.39	2.2	0.5185
* VHI	4	14.81	23	28	25.5	2.082	1.0408

9.2.7. FIRST POST-TRANSFUSION WHITE CELL COUNT

9.2.7.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
WCC1		26	0	99	0	0
*	VLO	1	1	2		
*	LO	1	2.1	4		
*	N	9	4.1	11		
*	HI	14	11.1	20		
*	VHI	1	20.1	99		

9.2.7.2. WCC Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
WCC1	26	100	2	21	11.92	4.454	0.8734
* VLO	1	3.85	2	2	2		
* LO	1	3.85	3	3	3		
* N	9	34.62	7	11	9.44	1.333	0.4444
* HI	14	53.85	12	20	14.21	2.665	0.7124
* VHI	1	3.85	21	21	21		

9.2.7.3. Effect of autotransfusion volume on WCC

Group name	n	%	Mean	Std.Error
ONE	1	4	11	0
TWO	12	46	13	1.36
THREE	5	19	8.8	0.58
FOUR	4	15	13	1
FIVE	2	8	16.5	3.5
SIX	1	4	12	0
SEVEN	1	4	2	0
TOTAL	26	100	11.9	0.87

Significance of group effect on WCC is 0.077418

9.2.8. FIRST POST-TRANSFUSION PI

9.2.8.1. Variable information

Name	Group	n	Min	Max
Totals		27	0	99
PI0	Very Low	0	1	50
	Low	14	51	89
	Normal	13	90	99

9.2.8.2. PI Results

Group	n	%	Lo	High	Mean	S.D.
Total	27	100	55	99	86.89	12.774
Very Low	0	0				
LOW	14	51.85	55	88	77.07	10.789
NORMAL	13	48.15	92	99	96.85	3.105

9.2.8.3. Effect of autotransfusion volume on PI

Group	n	%	Mean	Std.Error
1	1	4	75	0
2	13	48	88.5	2.44
3	5	19	80.4	7.95
4	4	15	94	3.14
5	2	7	99	0
6	1	4	81	0
7	1	4	55	0
Total	27	100	86.6	2.46

Significance of volume groups on PI = 0.038917 (Significant)

9.2.9. SECOND POST-TRANSFUSION PI

9.2.9.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI1		22	0	99	0	0
*	VLO	1	1	50		
*	LO	6	51	89		
*	N	15	90	99		

9.2.9.2. PI Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PI1	22	100	40	99	90.14	12.811	2.7313
* VLO	1	4.55	40	40	40		
* LO	6	27.27	74	88	84.83	5.382	2.1972
* N	15	68.18	90	99	95.6	3.501	0.904

9.2.9.3. Effect of autotransfusion volume on PI

Group	n	%	Mean	Std.Error
1	1	4	88	0
2	13	48	69.9	11.2
3	5	19	93.4	2.5
4	4	15	48.2	27.88
5	2	7	99	0
6	1	4	88	0
7	1		40	0
Total	27	100	73.3	7.22

Significance of autotransfused volume on PI is 1.083701

9.2.10. THIRD POST-TRANSFUSION PI

9.2.10.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI2		12	0	99	0	0
*	VLO	0	1	50		
*	LO	1	51	89		
*	N	11	90	99		

9.2.10.2. PI Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PI2	12	100	89	99	94.67	2.535	0.7317
* VLO	0	0					
* LO	1	8.33	89	89	89		
* N	11	91.67	93	99	95.18	1.888	0.5692

9.2.10.3. Effect of autotransfusion volume on PI

Group name	n	%	Mea..	Std.Error
ONE	1	8	96	0
TWO	7	58	94.6	1.23
THREE	2	17	95	1
FOUR	1	8	95	0
FIVE	EXCLUDED - 0 Cases			
SIX	1	8	93	0
SEVEN	EXCLUDED - 0 Cases			
TOTAL	12	100	94.7	0.73

Significance of group effect on PI is 1.49845

9.2.11. FIRST POST-TRANSFUSION PTT

9.2.11.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT0		27	0	99	0	0
*	NORMAL	19	28	39		
*	ABN	8	40	99		

9.2.11.2. PTT Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PTT0	27	100	28	91	38.37	11.878	2.2859
* NORMAL	19	70.37	28	39	33.42	2.912	0.6681
* ABN	8	29.63	40	91	50.12	16.669	5.8932

9.2.11.3. Effect of autotransfusion volume on PTT

Group	n	%	Mean	Std.Error
1	1	4	46	0
2	13	48	40.8	4.47
3	5	19	35	2.24
4	4	15	34.7	1.89
5	2	7	31	3
6	1	4	39	0
7	1	4	45	0
Total	27	100	38.4	2.29

Significance of autotransfused volume on PI is 1.416186

9.2.12. SECOND POST-TRANSFUSION PTT

9.2.12.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT1		22	0	99	0	0
*	NORMAL	19	28	39		
*	ABN	3	40	99		

9.2.12.2. PTT Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PTT1	22	100	30	76	35.64	9.634	2.054
* NORMAL	19	86.36	30	36	32.84	2.141	0.4912
* ABN	3	13.64	40	76	53.33	19.732	11.392

9.2.12.3. Effect of autotransfusion volume on PTT

Group name	n	%	Mean	Std.Error
ONE	1	5	33	0
TWO	10	45	35.2	1.36
THREE	5	23	33.2	0.86
FOUR	2	9	32	2
FIVE	2	9	31	1
SIX	1	5	31	0
SEVEN	1	5	76	0
TOTAL	22	100	35.6	2.05

Significance of group effect on PTT is 0.000004 (significant).

9.2.13. ADMISSION HB

9.2.13.1. Variable information

Name	Group	n	Min	Max	O/Range	Missing
Total		27	0	99	0	0
Hb0	Very Low	0	1	5		
	Low	6	6	10		
	Normal	19	11	14		
	High	2	15	20		

9.2.13.2. Hb Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HB0	27	100	6	16	11.89	2.342	0.450
* VLO	0	0					
* LO	6	22.22	6	10	8.67	1.506	0.6146
* N	19	70.37	11	14	12.47	1.264	0.2899
* HI	2	7.41	16	16	16		

9.2.14. FIRST POST-TRANSFUSION HB

9.2.14.1. Variable information

Name	Group	n	Min	Max
Total		27	0	99
Hb1	Very Low	0	1	5
	Low	3	6	10
	Normal	22	11	14
	High	2	15	20

9.2.14.2. Hb; Looking at Hb>0

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HB1	27	100	8	17	12.44	1.948	0.3749
* VLO	0	0					
* LO	3	11.11	8	10	8.67	1.155	0.6667
* N	22	81.48	11	14	12.64	1,093	0.233
* HI	2	7.41	15	17	16	1.414	1

9.2.14.3. Effect of autotransfusion volume on Hb

Group name	n	%	Mean	Std.Error
ONE	1	4	12	0
TWO	13	48	13.2	0.5
THREE	5	19	11.8	0.58
FOUR	4	15	12.7	0.25
FIVE	2	7	13.5	0.5
SIX	1	4	8	0
SEVEN	1	4	8	0
TOTAL	27	100	12.4	0.37

Significance of group effect on HB is 0.014644 = Significant

9.2.15. SECOND POST-TRANSFUSION HB

9.2.15.1. Variable information

Name	Group	n	Min	Max
Total		27	0	99
Hb2	Very Low	0	1	5
	Low	0	6	10
	Normal	0	11	14
	High	1	15	20

9.2.15.2. Hb; Looking at Hb>0

Group	n	%	Lo	High	Mean	S.D.
Very Low	0	0				
Low	0	0				
Normal	0	0				
High	1	3.7	15	15	15	NA
Total	27	100	0	15	NA	NA

9.2.16. POST-TRANSFUSION FDP'S

9.2.16.1. Variable Information

Name	Group	n	Min	Max	O/Range	Missing
Total		27	0	99	0	0
FDP	<10	10	1	1		
	10-40	12	2	2		
	40-80	0	4	4		
	80-120	2	8	8		
	120-240	2	16	16		
	>240	1	32	32		

9.2.16.2. FDP Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FDP	27	100	1	32	4.22	6.908	1.3294
* <10	10	37.04	1	1	1		
* 10-40	12	44.44	2	2	2		
* 40-80	0	0	0	0	0		
* 80-120	2	7.41	8	8	8		
*120-240	2	7.41	16	16	16		
* >240	1	3.7	32	32	32		

9.2.17. POST-TRANSFUSION HAPTOGLOBINS

9.2.17.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HAPTOGLOB		26	0	999	0	0
*	VLO	1	1	50		
*	LO	4	51	99		
*	N	19	100	300		
*	HI	2	301	999		

9.2.17.2. Haptoglobins Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HAPTOGLOB	26	100	40	320	142.54	70.226	13.7725
* VLO	1	3.85	40	40	40		
* LO	4	15.38	76	90	84	7.118	3.559
* N	19	73.08	100	300	142.11	46.774	10.7306
* HI	2	7.69	310	320	315	7.071	5

9.2.17.3. Effect of transfusion volume on Haptoglobins

Group name	n	%	Mean	Std.Error
ONE	1	4	140	0
TWO	13	50	140	22.97
THREE	5	19	161.2	37.73
FOUR	4	15	125	16.58
FIVE	2	8	165	45
SIX	1	4	110	0
SEVEN	EXCLUDED - 0 Cases			
TOTAL	26	100	142.5	13.77

Significance of group effect on HAPTOGLOB is 1.526343

9.2.18. POST-TRANSFUSION HAEMOPEXINS

9.2.18.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HAEMOPEX		26	0	999	0	0
*	LO	1	1	500		
*	N	25	501	999		

9.2.18.2. Haemopexins Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HAEMOPEX	26	100	442	850	664.69	112.06	21.9768
* LO	1	3.85	442	442	442		
* N	25	96.15	510	850	673.6	104.555	20.9109

9.2.18.3. Effect of transfusion volume on Haemopexins

Group name	n	%	Mean	Std.Error
ONE	1	4	580	0
TWO	13	50	672.4	30.16
THREE	5	19	595.4	51.13
FOUR	4	15	765.2	38.74
FIVE	2	8	638.5	117.5
SIX	1	4	646	0
SEVEN	EXCLUDED - 0 Cases			
TOTAL	26	100	664.7	21.98

Significance of group effect on HAEMOPEX is 0.329233

9.2.19. FIRST POST-TRANSFUSION FIBRINOGEN

9.2.19.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO1		26	0	9999	0	0
*	LO	4	1	199		
*	N	21	200	400		
*	HI	1	401	999		

9.2.19.2. Fibrinogen Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FIBRINO1	26	100	80	495	251.46	79.23	15.5383
* LO	4	15.38	80	198	148.5	55.89	27.9449
* N	21	80.77	200	372	259.48	47.17	10.2932
* HI	1	3.85	495	495	495		

9.2.19.3. Effect of transfusion volume on Fibrinogen

Group name	n	%	Mean	Std.Error
ONE	1	4	221	0
TWO	13	50	255.9	12.8
THREE	5	19	223.8	36.06
FOUR	4	15	247	53.48
FIVE	2	8	342.5	152.5
SIX	1	4	198	0
SEVEN	EXCLUDED - 0 Cases			
TOTAL	26	100	251.5	15.54

Significance of group effect on FIBRINO1 is 1.158822

9.2.20. SECOND POST-TRANSFUSION FIBRINOGEN

9.2.20.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO2		24	0	9999	0	0
*	LO	0	1	199		
*	N	23	200	400		
*	HI	1	401	999		

9.2.20.2. Fibrinogen Results

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FIBRINO2	24	100	222	538	301.04	66.238	13.5208
* LO	0	0					
* N	23	95.83	222	372	290.74	43.861	9.1456
* HI	1	4.17	538	538	538		

9.2.20.3. Effect of transfusion volume on Fibrinogen

Group name	n	%	Mean	Std.Error
ONE	1	4	272	0
TWO	12	50	289.2	14.28
THREE	5	21	277.6	7.27
FOUR	3	13	340.3	11.55
FIVE	2	8	381	157
SIX	1	4	312	0
SEVEN	EXCLUDED - 0 Cases			
TOTAL	24	100	301	13.52

Significance of group effect on FIBRINO2 is 0.418372

9.3. MIXED GROUP (BANKED AND AUTOTRANSFUSED)

This group comprised 17 patients.

9.3.1. TRANSFUSION VOLUME

9.3.1.1. Combined, Grouped

Units	1-5	6-8	9-10	11-15	16-20	21-25	26-30
Nos	6	7	2	0	1	0	1

9.3.1.2. Combined, Specific, and %

Total units	4	5	6	7	9	10	17	29
Nos	2	4	5	2	1	1	1	1
%	12	24	29	12	6	6	6	6

9.3.1.3. Individual Combined

Pt	autoxfused units	banked units
1	4	2
5	2	4
6	7	10
7	2	3
8	21	8
10	4	1
14	5	1
16	4	3
18	3	2
19	2	2
23	1	3
26	1	4
27	5	2
31	6	4
40	3	3
46	3	3
47	3	6

9.3.2. INJURIES, SURGERY, FINDINGS

Pt	chest	abdomen	other	operation	findings
1	blunt	0	0	lap	liver
5	stab	stab	# ribs	0	0
6	0	liver	0	lap	anastom
7	0	stab	hepatec	lap	liver
8	blunt	0	0	thoracot	ITArtery
10	0	stab	0	lap	spleen
14	0	stab	0	lap	spleen
16	blunt	0	0	thoracot	ITArtery
18	blunt	0	0	0	0
19	GSW	0	0	0	0
23	stab	0	# ribs	0	0
26	stab	0	0	lap	0
27	GSW	0	0	0	0
31	stab	0	0	thoracot	lung
40	0	stab	0	lap	spleen
46	0	blunt	0	lap	liver
47	blunt	blunt	0	lap	GIT,SCA+V

9.3.3. COMPLICATIONS

9.3.3.1. Complications, sepsis, death

Pt	sepsis	death	complic-1	complic-2
1	0	0	haematuria	0
5	0	0	0	0
6	0	0	atelectasis	encephalopathy
7	0	0	0	0
8	0	0	atelectasis	heparin-ooze
10	0	0	0	0
14	0	0	0	0
16	0	0	atelectasis	0
18	0	0	0	0
19	0	0	0	0
23	0	0	0	0
26	0	0	pulm-contuse	0
27	0	0	0	0
31	0	1	pulm-contuse	0
40	0	0	0	0
46	0	0	0	0
47	0	0	8	0

9.3.4. HAEMATOLOGY RESULTS

9.3.4.1. Haemoglobin, platelets, white cell count

Pt	Hb0	Hb1	Hb2	Plt0	Plt1	WCC0	WCC1
1	5	7	12	90	0	17	0
5	8	12	0	138	0	15	13
6	6	7	0	100	88	12	10
7	13	13	0	475	286	6	17
8	8	10	0	39	49	13	9
10	15	15	0	161	190	10	16
14	9	11	11	188	214	21	13
16	9	10	11	152	82	27	14
18	10	12	0	130	177	18	13
19	10	13	0	120	158	18	21
23	10	10	0	124	132	11	13
26	12	11	0	250	163	14	10
27	8	10	0	170	100	22	14
31	0	0	0	0	0	0	0
40	5	6	0	150	198	22	11
46	14	14	0	128	144	14	11
47	11	10	0	150	80	15	7

9.3.4.2. PI, PTT, FDP's

Pt	PI0	PI1	PI2	PTT0	PTT1	FDP
1	95	0	0	0	0	40-80
5	83	82	0	37	38	10-40
6	57	58	0	58	0	80-120
7	91	65	0	38	0	10-40
8	71	92	0	68	40	40-80
10	86	94	0	49	0	120-240
14	64	70	81	44	38	80-120
16	75	81	88	35	32	<10
18	68	86	97	40	33	120-240
19	77	86	95	34	33	<10
23	67	78	89	55	31	120-240
26	71	83	94	42	35	<10
27	98	65	84	48	37	120-240
31	0	0	0	0	0	0
40	56	82	95	46	37	120-240
46	69	81	93	45	34	80-120
47	90	79	85	42	37	<10

9.3.4.3. Haptoglobins, Haemopexins, Fibrinogen

Pt	HAPTOGLOB	HAEMOPEX	FIBRINO1	FIBRINO2
1	80	729	350	0
5	34	684	346	274
6	110	630	230	180
7	240	852	404	361
8	80	530	100	94
10	80	588	144	263
14	130	714	290	324
16	40	636	259	343
18	50	631	173	251
19	200	669	154	251
23	60	612	178	273
26	0	895	260	300
27	290	892	171	280
31	0	0	0	0
40	130	790	450	475
46	80	480	156	300
47	110	506	158	289

9.3.5. FIRST POST-TRANSFUSION PLATELET COUNT

9.3.5.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PLT0		16	0	999	0	0
*	LO	8	1	140		
*	N	7	141	400		
*	HI	1	401	999		

9.3.5.2. Platelet count

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PLT0	16	100	39	475	160.31	95.54	23.8849
* LO	8	50	39	138	108.62	32.359	11.4407
* N	7	43.75	150	250	174.43	36.036	13.6205
* HI	1	6.25	475	475	475		

9.3.5.3. Effect of transfusion volume on platelets

Name	Cases	%	Mean	Std.Error
Auto1	2	13	187	63
Auto2	3	19	244.3	115.45
Auto3	4	25	139.5	6.08
Auto4	3	19	134.3	22.32
Auto5	2	13	179	9
Auto6	0	0	0	0
Auto7	1	6	100	0
Auto21	1	6	39	0
Total	16	100	160.3	23.88

Significance of group effect on Plt = 1.148687

9.3.6. SECOND POST-TRANSFUSION PLATELET COUNT

9.3.6.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PLT1		16	0	999	0	0
*	LO	6	1	140		
*	N	8	141	400		
*	HI	0	401	999		

9.3.6.2. Platelet count

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PLT1	14	100	49	286	147.21	64.32	17.19
* LO	6	42.86	49	132	88.5	27.201	11.1048
* N	8	57.14	144	286	191.25	44.532	15.7443
* HI	0	0					

9.3.6.3. Effect of transfusion volume on platelets

Name	Cases	%	Mean	Std.Error
Auto1	2	14	147.5	15.5
Auto2	2	14	222	64
Auto3	4	29	149.7	25.77
Auto4	2	14	136	54
Auto5	2	14	157	57
Auto6	0	0	0	0
Auto7	1	7	88	0
Auto21	1	7	49	0
Total	14	100	147.2	17.19

Significance of group effect on PLT is 1.000499

9.3.7. ADMISSION WHITE CELL COUNT

9.3.7.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
WCC0		16	0	99	0	0
*	VLO	0	1	2		
*	LO	0	3	4		
*	N	3	5	11		
*	HI	9	12	20		
*	VHI	4	21	99		

9.3.7.2. White cell count

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
WCC0	16	100	6	27	15.94	5.31	1.3275
*VLO	0						
* LO	0						
* N	3	18.75	6	11	9	2.646	1.5275
* HI	9	56.25	12	18	15.11	2.147	0.7158
*VHI	4	25	21	27	23	2.708	1.354

9.3.7.3. Effect of transfusion volume on WCC

Name	Group	Cases	%	Min	Max	Mean
WCC0		2	100	11	18	14.5
	VLO	0	0			
	LO	0	0			
	N	1	50	11	11	
	HI	1	50	18	18	
	VHI	0	0			

9.3.8. FIRST POST-TRANSFUSION WHITE CELL COUNT

9.3.8.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
WCC1		16	0	99	0	0
*	VLO	0	1	2		
*	LO	0	3	4		
*	N	6	5	11		
*	HI	8	12	20		
*	VHI	1	21	99		

9.3 8.2. White cell count

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
WCC1	15	100	7	21	12.8	3.468	0.8955
*VLO	0	0					
* LO	0	0					
* N	6	40	7	11	9.67	1.506	0.6146
* HI	8	53.33	13	17	14.12	1.553	0.5489
*VHI	1	6.67	21	21	21		

9.3.8.3. Effect of transfusion volume on WCC

Name	Cases	%	Mean	Std.Error
Auto1	2	13	11.5	1.5
Auto2	3	20	17	2.31
Auto3	4	27	10.5	1.26
Auto4	2	13	15	1
Auto5	2	13	13.5	0.5
Auto6	0	0	0	0
Auto7	1	7	10	0
Auto21	1	7	9	0
Total	15	100	12.8	0.9

Significance of group effect on WCC is 0.11145

9.3.9. FIRST POST-TRANSFUSION PI

9.3.9.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI0		16	0	99	0	0
*	VLO	0	1	50		
*	LO	12	51	89		
*	N	4	90	99		

9.3.9.2. PI

Name	n	%	Lo	H	Mean	S.D.	S.E.M
PI0	16	100	56	98	76.12	13.079	3.2697
*VLO	0	0					
* LO	12	75	56	86	70.33	9.119	2.6324
* N	4	25	90	98	93.5	3.697	1.8484

9.3.9.3. Effect of transfusion volume on PI

Name	Cases	%	Mean	Std.Error
Auto1	2	13	69	2
Auto2	3	19	83.7	4.06
Auto3	4	25	70.7	7.06
Auto4	3	19	85.3	5.78
Auto5	2	13	81	17
Auto6	0	0	0	0
Auto7	1	6	57	0
Auto21	1	6	71	0
Total	16	100	76.1	3.27

Significance of group effect on PI is 0.43388

9.3.10. SECOND POST-TRANSFUSION PI

9.3.10.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI1		16	0	99	0	0
*	VLO	0	1	50		
*	LO	13	51	89		
*	N	2	90	99		

9.3.10.2. PI

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PI1	15	100	58	94	78.8	10.171	2.6262
*VLO	0						
* LO	13	86.67	58	86	76.62	9.042	2.5078
* N	2	13.33	92	94	93	1.414	1

9.3.10.3. Effect of transfusion volume on PI

Name	Cases	%	Mean	Std.Error
Auto1	2	13	80.5	2.5
Auto2	3	20	77.7	6.44
Auto3	4	27	82	1.47
Auto4	2	13	87.5	6.5
Auto5	2	13	67.5	2.5
Auto6	0	0	0	0
Auto7	1	7	58	0
Auto21	1	7	92	0
Total	15	100	78.8	2.63

Significance of group effect on PI is 0.046214 (Significant)

9.3.11. THIRD POST-TRANSFUSION PI

9.3.11.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI2		16	0	99	0	0
*	VLO	0	1	50		
*	LO	5	51	89		
*	N	5	90	99		

9.3.11.2. PI

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PI2	10	100	81	97	90.1	5.486	1.7349
*VLO	0	0					
* LO	5	50.00	81	89	85.4	3.209	1.4353
* N	5	50.00	93	97	94.8	1.483	0.6633

9.3.11.3. Effect of transfusion volume on PI

Name	Cases	%	Mean	Std.Error
Auto1	2	20	91.5	2.5
Auto2	1	10	95	0
Auto3	4	40	92.5	2.63
Auto4	1	10	88	0
Auto5	2	20	82.5	1.5
Auto6	0	0	0	0
Auto7	0	0	0	0
Auto21	0	0	0	0
Total	10	100	90.1	1.73

Significance of group effect on PI is 0.213362

9.3.12. FIRST POST-TRANSFUSION PTT

9.3.12.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT0		16	0	99	0	0
*	NORMAL	4	28	39		
*	ABN	11	40	99		

9.3.12.2. PTT

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PTT0	15	100	34	68	45.4	9.26	2.3901
*NORMAL	4	26.67	34	38	36	1.826	0.9129
* ABN	11	73.33	40	68	48.82	8.412	2.5363

9.3.12.3. Effect of transfusion volume on PTT

Name	Cases	%	Mean	Std.Error
Auto1	2	13	48.5	6.5
Auto2	3	20	36.3	1.2
Auto3	4	27	43.2	1.38
Auto4	2	13	42	7
Auto5	2	13	46	2
Auto6	0	0	0	0
Auto7	1	7	58	0
Auto21	1	7	68	0
Total	15	100	45.4	2.39

Significance of group effect on PTT is 0.012768 (Significant)

9.3.13. SECOND POST-TRANSFUSION PTT

9.3.13.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT1		16	0	99	0	0
*	NORMAL	11	28	39		
*	ABN	1	40	99		

9.3.13.2. PTT

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
PTT1	12	100	31	40	35.42	2.811	0.8115
*NORMAL	11	91.67	31	38	35	2.53	0.7628
* ABN	1	8.33	40	40	40		

9.3.13.3. Effect of transfusion volume on PTT

Name	Cases	%	Mean	Std.Error
Auto1	2	17	33	2
Auto2	2	17	35.5	2.5
Auto3	4	33	35.5	1.03
Auto4	1	8	32	0
Auto5	2	17	37.5	0.5
Auto6	0	0	0	0
Auto7	0	0	0	0
Auto21	1	8	40	0
Total	12	100	35.4	0.81

Significance of group effect on PTT is 0.230222

9.3.14. ADMISSION HB

9.3.14.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HB0		16	0	99	0	0
*	VLO	2	1	5		
*	LO	9	6	10		
*	N	4	11	14		
*	HI	1	15	20		

9.3.14.2. Hb

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HB0	16	100	5	15	9.56	2.966	0.7414
*VLO	2	12.5	5	5	5		
* LO	9	56.25	6	10	8.67	1.323	0.441
* N	4	25	11	14	12.5	1.291	0.6455
* HI	1	6.25	15	15	15		

9.3.15. FIRST POST-TRANSFUSION HB

9.3.15.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
HB1		16	0	99	0	0
*	VLO	0	1	5		
*	LO	8	6	10		
*	N	7	11	14		
*	HI	1	15	20		

9.3.15.2. Hb

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HB1	16	100	6	15	10.69	2.522	0.6306
*VLO	0	0					
* LO	8	50	6	10	8.75		0.6196
* N	7	43.75	11	14	12.29		0.4206
* HI	1	6.25	15	15	15		

9.3.15.3. Effect of transfusion volume on Hb

Name	Cases	%	Mean	Std.Error
Auto1	2	13	10.5	0.5
Auto2	3	19	12.7	0.33
Auto3	4	25	10.5	1.71
Auto4	3	19	10.7	2.33
Auto5	2	13	10.5	0.5
Auto6	Excluded, n=0			
Auto7	1	6	7	0
Auto21	1	6	10	0
Total	16	100	10.7	0.63

Significance of group effect on Hb is 1.269579

9.3.16. SECOND POST-TRANSFUSION HB

9.3.16.1. Variable Information

Name	Group	Cases	Min	Max	O/Range	Missing
HB2		16	0	99	0	0
*	VLO	0	1	5		
*	LO	0	6	10		
*	N	3	11	14		
*	HI	0	15	20		

9.3.16.2. Hb

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HB2	16	100	0	12	2.13	4.573	1.1434
*VLO	0	0					
* LO	0	0					
* N	3	18.75	11	12	11.33	0.577	0.3333
* HI	0	0					

9.3.16.3. Effect of transfusion volume on Hb

Name	Cases	%	Mean	Std.Error
Auto1		Excluded, n=0		
Auto2		Excluded, n=0		
Auto3		Excluded, n=0		
Auto4	2	67	11.5	0.5
Auto5	1	33	11	0
Auto6		Excluded, n=0		
Auto7		Excluded, n=0		
Auto2	1	Excluded, n=0		
Total	4	100	11.3	0.33

Significance of group effect on Hb is 1.15855

9.3.17. POST-TRANSFUSION FDP'S

9.3.17.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FDP		16	0	99	0	0
*	<10	4	1	1		
*	10-40	2	2	2		
*	40-80	2	4	4		
*	80-120	3	8	8		
*	120-240	5	16	16		
*	>240	0	32	32		

9.3.17.2. FDP's

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FDP	16	100	1	16	7.5	6.408	1.6021
*<10	4	25	1	1	1		
*10-40	2	12.5	2	2	2		
*40-80	2	12.5	4	4	4		
*80-120	3	18.75	8	8	8		
*120-240	5	31.25	16	16	16		
*>240	0	0					

9.3.18. POST-TRANSFUSION HAPTOGLOBINS

9.3.18.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HAPTOGLOB		16	0	999	0	0
*	VLO	3	1	50		
*	LO	5	51	99		
*	N	7	100	300		
*	HI	0	301	999		

9.3.18.2. Haptoglobins

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HAPTOGLOB	15	100	34	290	114.27	74.787	19.3099
* VLO	3	20	34	50	41.33	8.083	4.6667
* LO	5	33.33	60	80	76	8.944	4
* N	7	46.67	110	290	172.86	71.348	26.9668
* HI	0	0					

9.3.18.3. Effect of transfusion volume on Haptoglobins

Name	Cases	%	Mean	Std.Error
Auto1	1	7	60	
Auto2	3	20	63.07	
Auto3	4	27	17.5	
Auto4	3	20	13.33	
Auto5	2	13	80	
Auto6	Excluded, n=0			
Auto7	1	7	110	
Auto21	1	7	80	
Total	15	100		

Significance of group effect on Haptoglob is 0.399157

9.3.19. POST-TRANSFUSION HAEMOPEXINS

9.3.19.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HAEMOPEX		16	0	999	0	0
*	LO	1	1	500		
*	N	15	501	999		

9.3.19.2. Haemopexins

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
HAEMOPEX	16	100	480	895	677.37	128.877	32.2192
* LO	1	6.25	480	480	480		
* N	15	93.75	506	895	690.53	121.768	31.4403

9.3.19.3. Effect of transfusion volume on Haemopexins

Name	Cases	%	Mean	Std.Error
Auto1	2	13	753.5	141.5
Auto2	3	19	735	58.66
Auto3	4	25	601.7	70.88
Auto4	3	19	651	41.39
Auto5	2	13	803	89
Auto6	Excluded, n=0			
Auto7	1	6	630	0
Auto21	1	6	530	0
Total	16	100	677.4	32.22

Significance of group effect on Haemopex is 0.459308

9.3.20. FIRST POST-TRANSFUSION FIBRINOGEN

9.3.20.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO1		16	0	9999	0	0
*	LO	8	1	199		
*	N	6	200	400		
*	HI	2	401	999		

9.3.20.2. Fibrinogen

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FIBRINO1	16	100	100	450	238.94	103.525	25.8812
* LO	8	50	100	178	154.25	24.674	8.7234
* N	6	37.5	230	350	289.17	49.382	20.1601
* HI	2	12.5	404	450	427	32.527	23

9.3.20.3. Effect of transfusion volume on Fibrinogen

Name	Cases	%	Mean	Std.Error
Auto1	2	13	219	41
Auto2	3	19	301.3	75.55
Auto3	4	25	234.2	72.02
Auto4	3	19	251	59.6
Auto5	2	13	230.5	59.6
Auto6	Excluded, n=0			
Auto7	1	6	230	0
Auto21	1	6	100	0
Total	16	100	238.9	25.88

Significance of group effect on Fibrino1 is 1.394133

9.3.21. SECOND POST-TRANSFUSION FIBRINOGEN

9.3.21.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO2		16	0	9999	0	0
*	LO	2	1	199		
*	N	12	200	400		
*	HI	1	401	999		

9.3.21.2. Fibrinogen

Name	n	%	Lo	Hi	Mean	S.D.	S.E.M
FIBRINO2	15	100	94	475	284	83.683	21.6069
* LO	2	13.33	94	180	137	60.811	43
* N	12	80	251	361	292.58	35.367	10.2095
* HI	1	6.67	475	475	475		

9.3.21.3. Effect of transfusion volume on Fibrinogen

Name	Cases	%	Mean	Std.Error
Auto1	2	13	286.5	13.5
Auto2	3	20	295.3	33.5
Auto3	4	27	328.7	49.87
Auto4	2	13	304	41
Auto5	2	13	302	22
Auto6	Excluded, n=0			
Auto7	1	7	180	0
Auto21	1	7	94	0
Total	15	100	284	21.61

Significance of group effect on Fibrino2 is 0.209378

9.4. RUPTURED ECTOPIC PREGNANCIES

This group consisted of 12 patients.

9.4.1. TRANSFUSION VOLUME

9.4.1.1. Combined, Grouped

	Units	1-5	6-8
	Nos	11	1

9.4.1.2. Combined, Specific, and %

Total units	2	3	4	5	6	7	8
Nos	1	5	4	1	0	0	1
%	8	42	33	8	0	0	8

9.4.1.3. Individual Combined

Pt	autoxfuse	banked
43	4	4
55	5	0
56	3	0
57	4	0
58	3	0
59	3	0
60	3	0
61	4	0
62	4	0
63	4	0
64	3	0
65	2	0

9.4.2. HAEMATOLOGY RESULTS

9.4.2.1. Haemoglobin, platelets, white cell counts

Pt	HB0	HB1	HB2	WCC0	WCC1	PLT0	PLT1	AUTO
43	3	9	11	3	9	100	86	4
55	5	12	12	8	9	67	116	5
56	6	9	0	9	5	93	153	3
57	6	10	0	0	5	86	214	4
58	7	9	0	0	7	78	156	3
59	7	11	0	0	8	101	145	3
60	6	9	0	0	8	98	146	3
61	6	10	0	0	7	102	137	4
62	4	8	0	0	4	89	136	4
63	6	10	0	0	4	89	120	4
64	6	10	0	0	4	86	146	3
65	8	10	0	0	4	106	194	2

9.4.2.2. PI, FDP's

Pt	PI0	PI1	PI2	PTT0	PTT1	FDP	BANKED
43	89	80	94	34	33	2	4
55	79	88	95	0	0	2	0
56	82	86	96	0	0	2	0
57	88	97	0	0	0	8	0
58	84	96	0	0	0	2	0
59	86	94	0	0	0	2	0
60	81	89	95	0	0	2	0
61	79	88	0	0	0	2	0
62	74	86	0	0	0	8	0
63	83	91	0	0	0	8	0
64	76	81	0	0	0	2	0
65	85	94	0	0	0	2	0

9.4.2.3. Haptoglobins, haemopexins, fibrinogen

Pt	HAPTOGLOB	HAEMOPEX	FIBRINO1	FIBRINO2
43	60	460	284	315
55	38	280	0	0
56	43	370	0	0
57	45	340	228	314
58	60	380	262	0
59	50	400	0	0
60	47	372	346	0
61	64	390	480	0
62	30	274	231	0
63	47	430	462	0
64	60	362	240	0
65	168	400	265	0

9.4.3. FIRST POST-TRANSFUSION PLATELET COUNT

9.4.3.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PLTO		12	0	999	0	0
*	LO	12	1	140		
*	N	0	141	400		
*	HI	0	401	999		

9.4.3.2. Platelet count

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
PLTO	12	100	67	106	91.25	11.21	3.236
* LO	12	100	67	106	91.25	11.21	3.236
* N	0						
* HI	0						

9.4.3.3. Effect of transfusion volume on PLT

Group name	n	%	Mean	Std.Error
1	EXCLUDED - 0 Cases			
2	1	8	106	0
3	5	42	91.2	4.16
4	5	42	93.2	3.25
5	1	8	67	0
6	EXCLUDED - 0 Cases			
TOTAL	12	100	91.2	3.24

Significance of group effect on PLT is 0.05364

9.4.4. SECOND POST-TRANSFUSION PLATELET COUNT

9.4.4.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PLT1		12	0	999	0	0
*	LO	5	1	140		
*	N	7	141	400		
*	HI	0	401	999		

9.4.4.2. Platelet count

Name	n	%	Lo	Hi	Mean	S.D
PLT1	12	100	86	214	145.75	33.621
*LO	5	41.67	86	137	119	20.688
* N	7	58.33	145	214	164.86	27.655
*HI	0					

9.4.4.3. Effect of transfusion volume on PLT

Group name	n	%	Mean	Std.Error
1	EXCLUDED - 0 Cases			
2	1	8	194	0
3	5	42	149.2	2.22
4	5	42	138.6	20.98
5	1	8	116	0
6	EXCLUDED - 0 Cases			
TOTAL	12	100	145.7	9.71

Significance of group effect on PLT is 0.41964

9.4.5. ADMISSION WHITE CELL COUNT

9.4.5.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
WCC0		12	0	99	0	0
*	VLO	0	1	2		
*	LO	1	3	4		
*	N	2	5	11		
*	HI	0	12	20		
*	VHI	0	21	99		

9.4.5.2. White cell count

Name	n	%	Lo	Hi	Mean	S.D
WCC0	3	100	0	9	1.67	3.312
*VLO	0					
* LO	1	33.33	3	3	3	0
* N	2	66.67	8	9	8.5	0.707
* HI	0					
*VHI	0					

9.4.6. FIRST POST-TRANSFUSION WHITE CELL COUNT

9.4.6.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
WCC1		12	0	99	0	0
*	VLO	0	1	2		
*	LO	4	3	4		
*	N	8	5	11		
*	HI	0	12	20		
*	VHI	0	21	99		

9.4.6.2. White cell count

Name	n	%	Lo	Hi	Mean	S.D
WCC1	12	100	4	9	6.17	2.038
*VLO	0					
* LO	4	33.33	4	4	4	
* N	8	66.67	5	9	7.25	1.581
* HI	0					
*VHI	0					

9.4.6.3. Effect of transfusion volume on WCC

Name	n	%	Mean	Std.Error
1	Excluded, n=0			
2	1	8	4	0
3	5	42	6.4	0.81
4	5	42	5.8	0.97
5	1	8	9	0
6	Excluded, n=0			
Total	12	100	6.2	0.59

Significance of Autoxfuse group effect on WCC is 0.390312

9.4.7. FIRST POST-TRANSFUSION PI

9.4.7.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI1		12	0	99	0	0
	VLO	0	1	50		
	LO	12	51	89		
	N	0	90	99		

9.4.7.2. PI

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
PI0	12	100	74	89	82.17	4.609	1.3305
* VLO	0						
* LO	12	100	74	89	82.17	4.609	1.3305
* N	0						

9.4.7.3. Effect of transfusion volume on PI

Name	n	%	Mean	Std.Error
1	Excluded, n=0			
2	1	8	85	0
3	5	42	81.8	1.69
4	5	42	82.6	2.8
5	1	8	79	0
6	Excluded, n=0			
Total	12	100	82.2	1.33

Significance of Autoxfuse group effect on PI is 1.42151

9.4.8. SECOND POST-TRANSFUSION PI

9.4.8.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI1		12	0	99	0	0
*	VLO	0	1	50		
*	LO	7	51	89		
*	N	5	90	99		

9.4.8.2. PI

Name	n	%	Lo	Hi	Mean	S.D
PI1	12	100	80	97	89.17	5.491
*VLO	0	0				
* LO	7	58.33	80	89	85.43	3.552
* N	5	41.67	91	97	94.4	2.302

9.4.8.3. Effect of transfusion volume on PI

Name	n	%	Mean	Std.Error
1	Excluded, n=0			
2	1	8	94	0
3	5	42	89.2	2.71
4	5	42	88.4	2.8
5	1	8	88	0
6	Excluded, n=0			
Total	12	100	89.2	1.59

Significance of Autoxfuse group effect on PI is 1.42315

9.4.9. THIRD POST-TRANSFUSION PI

9.4.9.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PI2	All	12	0	99	0	0
*	VLO	0	1	50		
*	LO	0	51	89		
*	N	4	90	99		

9.4.9.2. PI

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
PI2	4	100	94	96	95	0.816	0.41
*VLO	0	0					
* LO	0	0					
* N	4	100	94	96	95	0.816	0.408

9.4.9.3. Effect of transfusion volume on PI

Name	n	%	Mean	Std.Error
1	Excluded, n=0			
2	Excluded, n=0			
3	2	50	95.5	0.5
4	1	25	94	0
5	1	25	95	0
6	Excluded, n=0			
Total	4	100	95	0.41

Significance of group effect on PI is 0.501667

9.4.10. FIRST POST-TRANSFUSION PTT

9.4.10.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT0		12	0	99	0	0
*	NORMAL	1	28	39		
*	ABN	0	40	99		

9.4.10.2. PTT

Name	n	%	Lo	Hi	Mean
PTT0	1	100	34	34	34
*NORMAL	1	100	34	34	34
* ABN	0	0			

9.4.11. SECOND POST-TRANSFUSION PTT

9.4.11.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
PTT1		12	0	99	0	0
*	NORMAL	1	28	39		
*	ABN	0	40	99		

9.4.11.2. PTT

Name	n	%	Lo	Hi	Mean
PTT1	1	100	33	33	33
*NORMAL	1	100	33	33	33
* ABN	0	0			

9.4.12. ADMISSION HB

9.4.12.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HB0		12	0	99	0	0
*	VLO	3	1	5		
*	LO	9	6	10		
*	N	0	11	14		
*	HI	0	15	20		

9.4.12.2. Hb

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
HB0	12	100	3	8	5.83	1.337	0.386
*VLO	3	25	3	5	4	1	0.5774
* LO	9	75	6	8	6.44	0.726	0.2422
* N	0	0					
* HI	0	0					

9.4.13. FIRST POST-TRANSFUSION HB

9.4.13.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HB1		12	0	99	0	0
*	VLO	0	1	5		
*	LO	10	6	10		
*	N	2	11	14		
*	HI	0	15	20		

9.4.13.2. Hb

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
HB1	12	100	8	12	9.75	1.055	0.3046
*VLO	0	0					
* LO	10	83.33	8	10	9.4	0.699	0.2211
* N	2	16.67	11	12	11.5	0.707	0.5
* HI	0	0					

9.4.13.3. Effect of transfusion volume on Hb

Group name	n	%	Mean	Std.Error
1	EXCLUDED - 0 Cases			
2	1	8	10	0
3	5	42	9.6	0.4
4	5	42	9.4	0.4
5	1	8	12	0
6	EXCLUDED - 0 Cases			
TOTAL	12	100	9.7	0.3

Significance of group effect on HB is 0.13959

9.4.14. SECOND POST-TRANSFUSION HB

9.4.14.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HB2		12	0	99	0	0
*	VLO	0	1	5		
*	LO	0	6	10		
*	N	2	11	14		
*	HI	0	15	20		

9.4.14.2. Hb

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
HB2	2	100	0	12	1.92	4.481	1.294
*VLO	0	0					
* LO	0	0					
* N	2	100	11	12	11.5	0.707	0.5
* HI	0	0					

9.4.15. POST-TRANSFUSION FDP'S

9.4.15.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FDP		12	0	99	0	0
*	+	9	2	2		
*	++	3	8	8		
*	+++	0	16	16		

9.4.15.2. FDP's

Name	n	%	Lo	Hi	Mean	S.D	SEM
FDP	12	100	2	8	3.5	2.714	0.7833
*10-40	9	75	2	2	2		
*80-120	3	25	8	8	8		
*120-240	0						

9.4.16. POST-TRANSFUSION HAPTOGLOBINS

9.4.16.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HAPTOGLOB		12	0	999	0	0
*	VLO	7	1	50		
*	LO	4	51	99		
*	N	1	100	300		
*	HI	0	301	999		

9.4.16.2. Haptoglobins

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
HAPTOGLOB	12	100	30	168	59.33	35.689	10.305
* VLO	7	58.33	30	50	42.86	6.817	2.577
* LO	4	33.33	60	64	61	21	
* N	1	8.33	168	168	168		
* HI	0						

9.4.16.3. Effect of transfusion volume on Haptoglobins

Group name	n	%	Mean	Std.Error
1	EXCLUDED - 0 Cases			
2	1	8	168	0
3	5	42	52	3.45
4	5	42	49.2	6.03
5	1	8	38	0
6	EXCLUDED - 0 Cases			
TOTAL	12	00	59.3	10.3

Significance of group effect on HAPTOGLOB is 0.000119 (Significant)

9.4.17. POST-TRANSFUSION HAEMOPEXINS

9.4.17.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
HAEMOPEX		12	0	999	0	0
*	LO	12	1	500		
*	N	0	501	999		

9.4.17.2. Haemopexins

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
HAEMOPEX	12	100	274	460	371.5	54.152	15.6324
* LO	12	100	274	460	371.5	54.152	15.6324
* N	0	0					

9.4.17.3. Effect of transfusion volume on Haemopexins

Name	n	%	Mean	Std.Error
1	EXCLUDED, n=0			
2	1	8	400	0
3	5	42	376.8	6.47
4	5	42	378.8	33.04
5	1	8	280	0
6	EXCLUDED, n=0			
TOTAL	12	100	371.5	15.63

Significance of group effect on HAEMOPEX is 0.393891

9.4.18. FIRST POST-TRANSFUSION FIBRINOGEN

9.4.18.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO1		12	0	9999	0	0
*	LO	0	1	199		
*	N	7	200	400		
*	HI	2	401	999		

9.4.18.2. Fibrinogen

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
FIB1	9	100	228	480	310.89	97.572	32.5241
* LO	0	0					
* N	7	77.78	228	346	265.14	40.99	15.4926
* HI	2	22.22	462	480	471	12.728	9

9.4.18.3. Effect of transfusion volume on Fibrinogen

Name	n	%	Mean	Std.Error
1	Excluded, n=0			
2	1	8	265	0
3	5	42	169.6	71.46
4	5	4	233.7	55.68
5	1	8	0	0
6	Excluded, n=0			
TOTAL	12	100	233.2	47.16

Significance of group effect on FIBRINO1 is 0.178156

9.4.19. SECOND POST-TRANSFUSION FIBRINOGEN

9.4.19.1. Variable information

Name	Group	Cases	Min	Max	O/Range	Missing
FIBRINO2		12	0	9999	0	0
*	LO	0	1	199		
*	N	2	200	400		
*	HI	0	401	999		

9.4.19.2. Fibrinogen

Name	n	%	Lo	Hi	Mean	S.D	S.E.M
FIBRINO2	2	100	314	315	314.5	0.707	0.5
* LO	0	0					
* N	2	100	314	315	314.5	0.707	0.5
* HI	0	0					

9.4.19.3. Effect of transfusion volume on Fibrinogen

Name	n	%	Mean	Std.Error
1	Excluded, n=0			
2	1	8	0	0
3	5	42	0	0
4	5	42	125.8	77.04
5	1	8	0	0
6	Excluded, n=0			
TOTAL	12	100	52.4	35.34

Significance of group effect on FIBRINO2 is 0.426905

9.5. COMPLICATIONS (ALL)

9.5.1. COMPLICATIONS, SEPSIS, DEATH

Pt	complic-1	complic-2	sepsis	death
1	Haematuria	0	0	0
6	Atelectasis	Encephalopathy	0	0
8	Atelectasis	Heparin ooze	0	0
16	Atelectasis	0	0	0
21	Pneumonia	0	0	0
26	(Pulm.contuse)	0	0	0
26	0	Thoracotomy/clot	0	0
29	Atelectasis	0	0	0
30	Atelectasis	0	0	0
31	(Pulm.contuse)	0	0	1
33	0	DVT/PE	0	0
38	DIC,ATN,ARDS		0	1
39	Pneumonia	0	0	0
43	Transient confusion		0	0
47	ARDS	0	0	0
49	Atelectasis	0	0	0
53	Atelectasis	0	0	0
58	Haematuria,atelectasis		0	0
60	Haematuria	0	0	0
61	Haematuria	0	0	0
62	Haematuria	0	0	0
63	Haematuria	0	0	0

Although 22 patients are in the above table, pulmonary contusions are not complications, so in effect there were only 20 complicated patients out of 65 trial patients (31%).

I do not consider post-cardiac arrest confusion and pneumonia following a pre-admission aspiration to be complications of management, so the above paragraph should be modified to 18 complicated patients out of 65 (28%).

The encephalopathy occurred in a patient with pre-existing alcoholic cirrhosis:

Haematuria:	6
Atelectasis:	8
Pneumonia:	1
ARDS:	2
DIC,ATN,ARDS:	1
Encephalopathy:	1
Transient confusion	*1*
Heparin coagulopathy:	1
Aspiration:	1
DVT/PE:	1
Thoracotomy/clot evacuation:	1
Overall complication:	24 in 18 patients.
Modified complications:	22 in 18 patients.

Death: 2 3%

9.5.1.1. Banked onl

Pt	complic1	complic2	sepsis	death
39	pneumonia	0	0	0
49	atelectasis	0	0	0
53	atelectasis	0	0	0
66	atelec,pneum	0	0	0
68	atelectasis	0	0	0
70	atelectasis	0	0	0
72	DIC,ATN,ARDS	intra/postop	0	YES
75	atelectasis	0	0	0

9.5.1.2. Autotransfusion only

Pt	COMPLIC-1	COMPLIC-2	SEPSIS	DEATH	UNITS
21	Pneumonia	Aspirate	0	0	3
28	0	Thoracotomy	0	0	2
29	Atelectasis	0	0	0	4
30	Atelectasis	0	0	0	2
33	0	DVT/PE	0	0	3
38	DIC,ATN,ARDS	Intra/postop	0	YES	7

9.5.1.3. Mixed

Pt	complic-1	complic-2	sepsis	death	units
1	Haematuria	0	0	0	6
6	Atelectasis	Encephalopathy	0	0	17
8	Atelectasis	Heparin	0	0	29
16	Atelectasis	0	0	0	7
26	(Pulm.contuse)	0	0	0	5
31	DIC	0	0	Yes	10
47	ARDS	0	0	0	9

9.5.1.4. Ectopics

Pt	complic1	complic2	sepsis	banked	autoxfuse
43	Transient confusion	0	0	4	4
58	Atelectasis/haematuria		0	0	3
60	Haematuria	0	0	0	3
61	Haematuria	0	0	0	4
62	Haematuria	0	0	0	4
63	Haematuria	0	0	0	4

9.5.2. SALVAGE TECHNIQUES

Pt	salvage	Pt	salvage
1	DPL	30	Sorensen
2	DPL+Swabs	31	Sorensen
3	ICD	32	Sorensen
4	ICD	33	Sorensen
5	ICD	34	Sorensen
6	Sorensen+Swabs	35	DPL
7	Sorensen	36	Sorensen
8	ICD	37	Sorensen
10	Sorensen	38	Sorensen+Swabs
11	Sorensen	40	Swabs
12	ICD	41	ICD
13	ICD	42	ICD
14	Sorensen	43	DPL
15	Sorensen	44	ICD
16	Sorensen	45	ICD
17	ICD	46	Swabs
18	ICD	47	ICD
19	ICD	55	Swabs
20	Sorensen+Swabs	56	Swabs
21	ICD	57	Swabs
22	ICD	58	Swabs
23	ICD	59	Swabs
24	DPL	60	Swabs
25	ICD	61	Swabs
26	ICD	62	Swabs
27	ICD	63	Swabs
28	ICD	64	Swabs
29	Sorensen+Swabs	65	Swabs

9.5.2.1. Equipment used

ICD only	21
Sorenson device only	13
Dialysis catheter only	4
Swab filtration only	13
Swabs & DPL	1
Sorensen & DPL	4

9.6. RESULTS OF SALVAGED BLOOD

The salvaged blood of 12 patients was analyzed to determine the cellular contents, the clotting profile, fibrinogen content, and the quantitative FDP content.

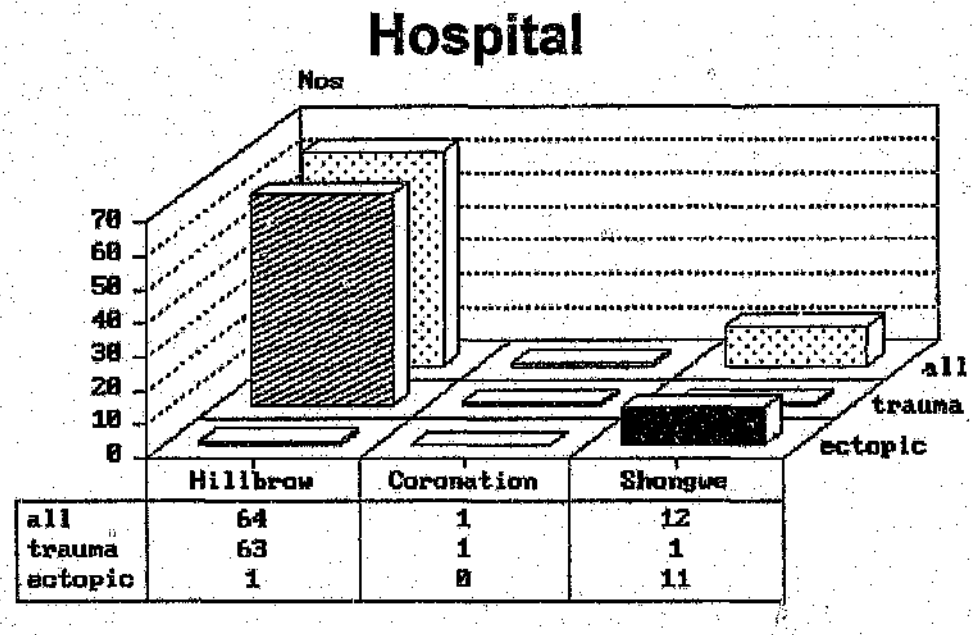
9.6.1. AUTOTRANSFUSED BLOOD

Hb	WCC	PLT	PI	PTT	FDP	Fibrinogen
10.5	4.1	11	>120	>120	>240	80
9.4	5.7	47	>120	>120	>240	0
14	8.3	83.2	>120	>120	>240	0
12.5	6.6	65	>120	>120	>240	100
14	8.1	67	>120	>120	>240	0
14.3	9.2	90	>120	>120	>240	0
5	5.2	35	>120	>120	>240	27
10.2	4.6	80	<10%	>120	120-240	0
11.9	5.6	88	>120	>120	>240	0
12.5	7.1	15	>120	>120	>240	0
8	8.9	120	NA	56%	120-240	0
10	17	315	>120	>120	>240	18

10. DISCUSSION

10.1. PATIENTS

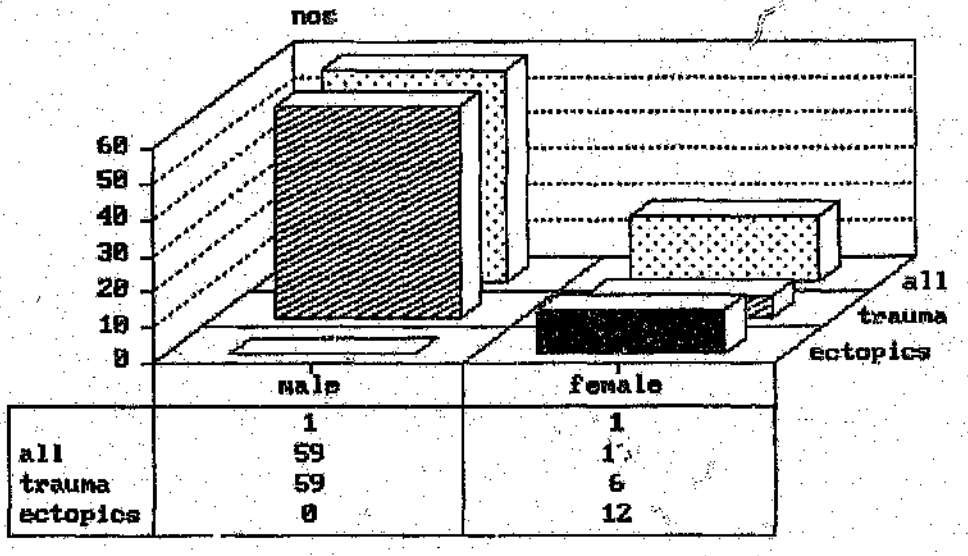
During the period June 1985 to December 1989, 77 patients were accumulated for the autotransfusion trial, 21 of which were control patients. These patients were managed at three institutions namely Hillbrow (64), Coronation (1) and Shongwe (12) Hospitals. 75 were of Bantu origin and 2 patients of mixed race. The graph **HOSPITAL**, below, shows the patient distribution within the 3 institutions.



	Hillbrow	Coronation	Shongwe
all	64	1	12
trauma	63	1	1
ectopic	1	0	11

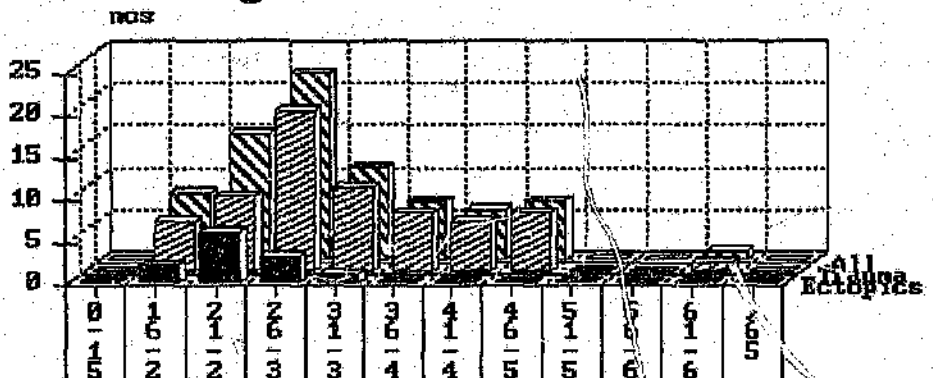
The graph **SEX DISTRIBUTION**, below, illustrates the sex distribution of the study patients. The patients comprised 59 males and 18 females. Of the female patients, 12 were ruptured ectopic pregnancies, thus reducing the trauma female numbers to 6. This male to female ratio of 59:6 is probably a fair indication of the usual ratio of male to female admissions to a trauma unit in the early to mid 1980's. With the onset of indiscriminate township violence, political inspired unrest and the taxi wars, the numbers of female patients, and children have risen.

Sex Distribution



	male	female
all	1 59	1 1
trauma	59	6
ectopics	0	12

Age Distribution



	0-15	16-20	21-25	26-30	31-35	36-40	41-45	46-50	51-55	56-60	61-65	>65
All	0	0	15	22	11	7	6	7	0	0	1	0
Trauma	0	6	9	19	10	7	6	7	0	0	1	0
Ectopics	0	2	6	3	1	0	0	0	0	0	0	0

Age Groups

The above graph **AGE DISTRIBUTION**, depicts the age distribution within the study. The ages of all patients ranged from 16 yrs to 65 yrs.

The ages were divided into 12 groups; 0-15, >65, and the intervening ages divided into half decile groups.

The trauma patients ages ranged from 16-65 years, with a mean of 33.06 years. The 26-30 group comprised the largest single group, making up 29.23% of the total. The 21-30 decile accounted for 43.08% of all trauma patients. This may well have been as a consequence of this age group being the most socially active within the community. Most of these patients were injured by interpersonal violence, largely alcohol related.

The older groups of over 40 years were often secondary to muggings by gangs of youths.

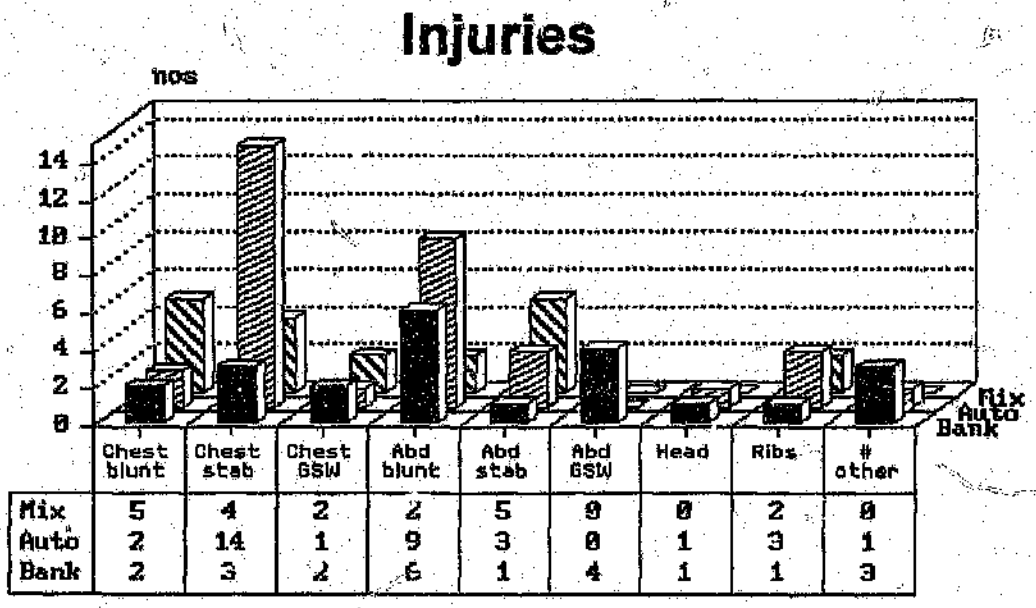
10.2. INJURIES

10.2.1. TRAUMA

The injuries comprised motor vehicle accidents with blunt chest and abdominal trauma, assault victims following either or both blunt or penetrating injuries of the chest, abdomen and head.

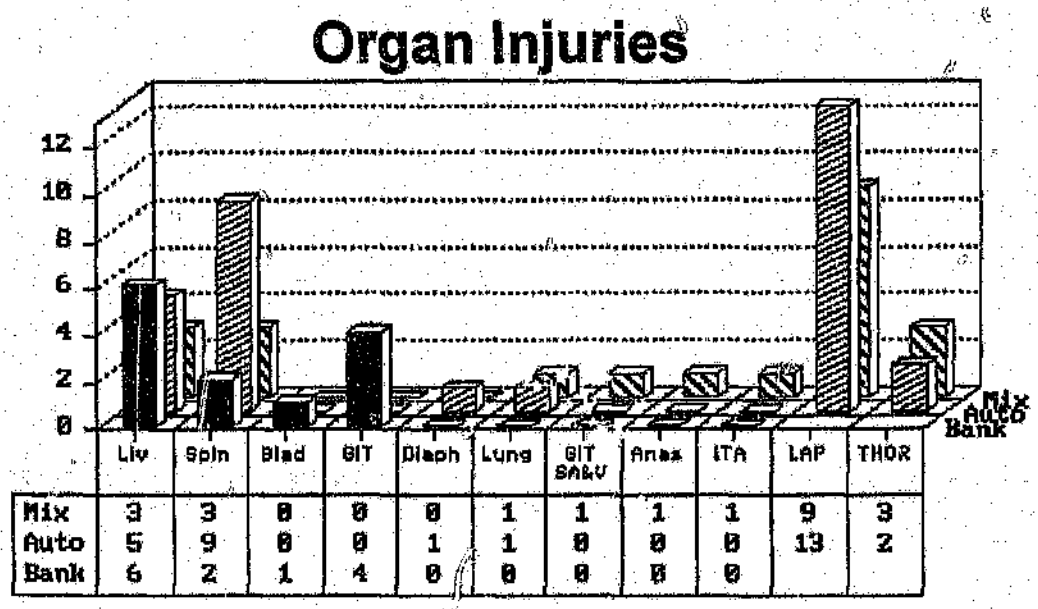
An attempt was made to keep the trauma distribution as similar as possible in the trauma groups.

The injury distribution is shown in graph **INJURIES**, below.



	Chest blunt	Chest stab	Chest GSW	Abd blunt	Abd stab	Abd GSW	Head	Ribs	# other
Mix	5	4	2	2	5	9	0	2	0
Auto	2	14	1	9	3	0	1	3	1
Bank	2	3	2	6	1	4	1	1	3

The organ injuries are depicted in graph **Organ Injuries**, below.



	Liv	Spln	Blad	GIT	Diaph	Lung	GIT SA&V	Anex	LTA	LAP	THOR
Mix	3	3	0	0	0	1	1	1	1	9	3
Auto	5	9	0	0	1	1	0	0	0	13	2
Bank	6	2	1	4	0	0	0	0	0		

10.2.2. ECTOPICS

Ruptured ectopic pregnancies comprised a fourth category, consisting of 12 patients. The ages ranged from 18-32 years, with a mean of 24.67 years. The 21-25 group accounted for 50% of these patients, while the 21-30 decile consisted of 75% of this group.

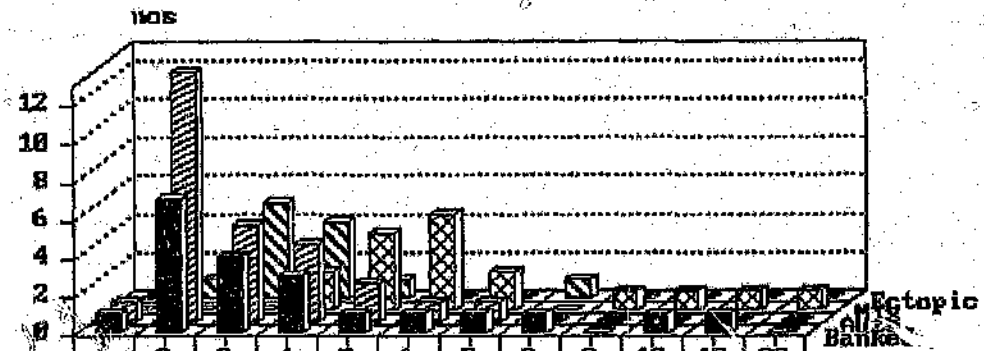
All 12 patients presented with haemoperitoneums which were diagnosed clinically, and confirmed by way of percutaneous DPL. Culdoscopy was not used as a diagnostic aid.

10.3. TRANSFUSIONS

The patients fell into 4 categories:

1) Banked blood only (**bank**, controls).
2) Autotransfused blood only (**auto**).
3) Both banked and autotransfused blood (**mix**).
4) Ruptured ectopic pregnancies (**ecto**).

Transfusions



	1	2	3	4	5	6	7	8	9	10	17	29
Ectopic	0	1	5	4	1	0	0	1	0	0	0	0
Mix	0	0	0	2	4	5	2	0	1	1	1	1
Auto	1	13	5	4	2	1	1	0	0	0	0	0
Banked	1	7	4	3	1	1	1	1	0	1	1	0

Units

As can be seen from the above graph **TRANSFUSIONS**, the banked only and the autotransfused only groups peaked at 2 unit transfusions, the mixed group at 6 units and the ectopic group at 3 unit transfusions.

52.38% of the banked group consisted of 2 or 3 unit transfusions.

66.67% of the autotransfused only group comprised 2 or 3 unit transfusions.

53% of the mixed group had 5 or 6 unit transfusions.

75% of the ectopics received 3 or 4 units.

10.3.1. BLOOD SAVING

10.3.1.1. Banked

In this control group of 21 patients, a total of 92 units of **banked** blood were transfused. This is equivalent to present costs (R150.00/unit), of **R13 800.00** .

10.3.1.2. Autotransfused

In this group of 27 patients, 81 units of blood were autotransfused. This represents a saving of 81 units of banked blood, which, at a present cost of R150.00 per unit, amounts to a saving of **R12 150.00**.

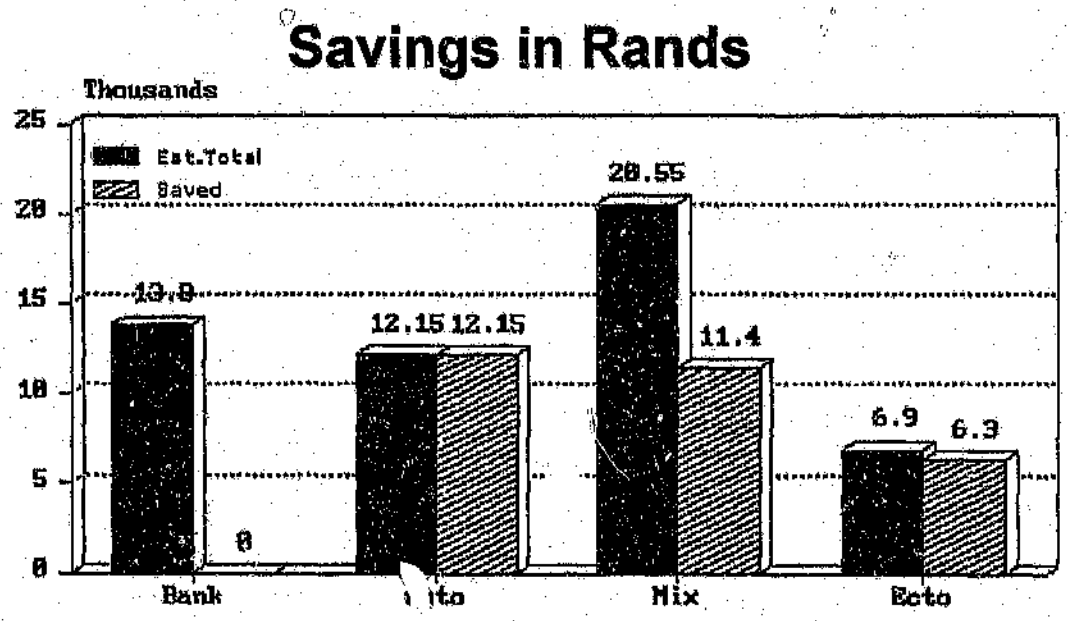
10.3.1.3. Mixed

In 17 patients, a total of 137 units were transfused. Out of these 137 units, 55% (76) were salvaged blood, and 45% (61) were banked blood. The 76 autotransfused units represent a financial saving of R11 400.00, out of a thecretical total cost of R20 550.00 (55%).

10.3.1.4. Ectopics

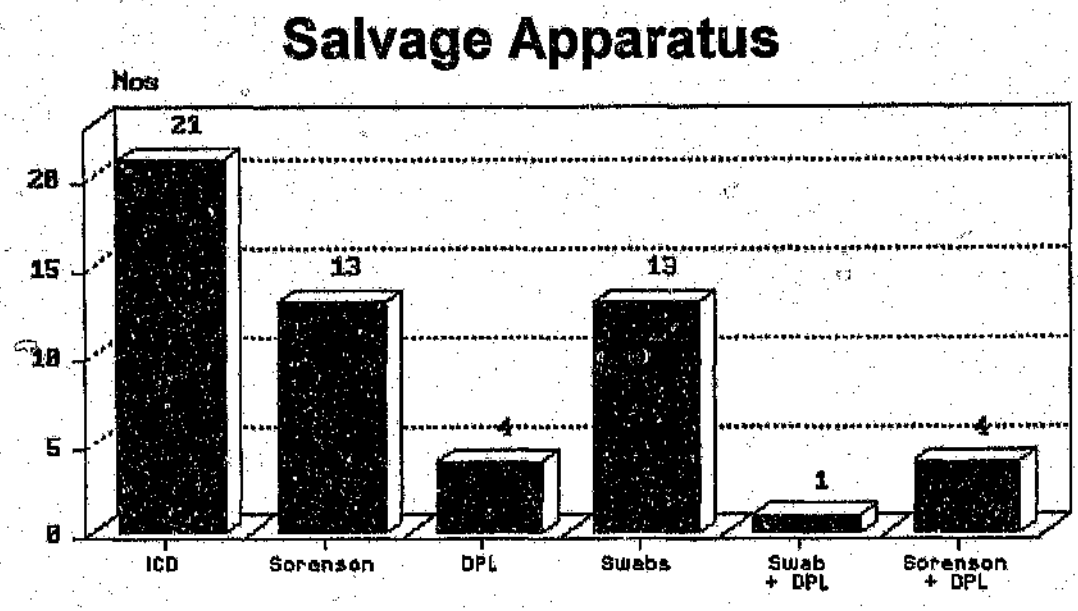
Of 12 patients with ruptured ectopic pregnancies, a total of 46 units were transfused. Of these 46 units, 91% were salvaged blood. This represents a monetary saving of R6 300.00 (91%), out of a theoretical R6 900.00 blood bill.

The graph **Savings in Rands**, below, illustrates an estimated total cost had all units been banked blood, compared to money saved according to salvaged volumes.



10.4. EQUIPMENT

10.4.1. INTRODUCTION



Of the 77 patients in this study, 56 received autotransfusions. Of the latter group, the graph **Salvage Apparatus** above, displays the equipment used to collect the blood.

Initially, only the Sorenson device was utilized. Although it is not a complicated apparatus, it does require about ten minutes to set up, and to prepare the heparin solution for anticoagulation. As trauma intakes can be fairly busy, and if an emergency case needed theatre urgently, it became apparent that other salvage techniques were as quick, more readily avail·ble, and probably more cost effective.

We often were confronted with a situation where the blood supply exceeded the available blood collection bags and resulted in the loss of a valuable resource.

Although it proved to be an effective device, it was found that for trauma blood salvage, simpler and cheaper apparatus was equally effective.

The alternative methods were adopted in cases where body cavity blood was available for salvage, where the autotransfusor had not been set up, or, was unavailable, and blood was required for transfusion.

In several patients, combined collecting methods were used.

This blood salvage utilized on site materials and did not add to the cost of patient management.

10.4.2. SORENSON CELL SAVER

This device has the advantage of simplicity when compared to more sophisticated cell savers. The disposables are also much cheaper; the 2 000ml inner liner costs about R35.00, so as little as 1 unit of blood is cost effective. This is not the case with cell-washing cell savers, where several units are required to cover the cost of the disposables

As simple as this device may be, it still required setting up, and that was where problems arose.

Unless trauma staff are encouraged to utilize it, and serious autotransfusion is the official policy of a trauma centre, it becomes the hobby-horse of only one person.

This cell saver does not wash and compact red cells, it only filters and simultaneously anticoagulates the salvaged blood. The use of such blood results in the transfusion of activated leucocytes, platelets, clotting factors and other vasoactive agents.

As demonstrated above, virtually all blood collected from mesothelial lined cavities is incapable of clotting, and thus raises the question of whether blood salvage really needs anticoagulation, and if so, how much? Not a single specimen of uncoagulated blood from salvaged units clotted in this trial.

10.4.3. INTERCOSTAL DRAIN SALVAGE

Blood collected in the conventional intercostal drain bottles would be transferred into an emptied 1L transfusovac bottle, anticoagulated with 500u of heparin and subsequently transfused as any other bottled blood unit.

It was found to be easier to use emptied 1l transfusovac bottles than the cell saver. Theoretically less cell trauma occurred with this method. Minimal extra expense was incurred as all the material was on site. Each bottle could be used more than once. This method

was more accepted than the cell saver, which required more time to set up, and utilized unfamiliar components.

10.4.4. DIALYSIS CATHETER SALVAGE

The use of the peritoneal lavage catheter stems from the time of the young woman who presented with severe shock from a ruptured ectopic pregnancy. She had a hypovolaemic cardiac arrest in casualty. No blood was available at the time and we collected sufficient blood into emptied 1L vacolitre bags to successfully resuscitate her.

In the trauma setting, large haemoperitoneums diagnosed via DPL were collected in vacolitre bags and autotransfused intraoperatively when bowel injuries were excluded. Heparin, 500u, was added to each bag.

This proved to be a useful method of blood salvage in large haemoperitoneums. In those patients where DPL drained frank blood at insertion, no saline was introduced into the peritoneal cavity, but the blood was allowed to drain into an emptied plastic vacolitre bag. This proved to be a very cheap method of blood salvage.

10.4.5. SWAB FILTRATION METHOD

Haemoperitoneums were filtered through abdominal swabs into sterile bowls and the blood was subsequently transferred into glass transfusovac bottles. The same dosage of heparin was used.

This technique required little input from others. Blood that was filtered through abdominal swabs was poured from the sterile basin into an empty transfusovac bottle, which was then hung up as any other glass blood bottle. This was a very cost effective method of blood salvage, where the only extra requirement was the transfusovac bottle.

10.5. HAEMATOLOGICAL CONSEQUENCES OF TRANSFUSIONS

10.5.1. HAEMATOLOGY OF AUTOTRANSFUSED BLOOD

It is obvious from the results in the Table in 9.6.1 in that there are gross haematological alterations of blood in contact with mesothelial surfaces.

These tests were performed on haemothoraces before anticoagulation and autotransfusion.

Hb>=10:	9	WCC<4	0
Hb<8 :	1	WCC>11	1
8=<Hb<10:	2	4=<WCC=<11	11

75% of the Hb's were greater than or equal to 10, 17% were less than 10, but greater than or equal to 8. Only one (8.3%) had a haemoglobin level of less than 8.

The WCC was normal in 92% and raised in 1 (8.3%).

Plt>=140	1
Plt<50	4
50<Plt<100	6
100<Plt<140	1

The platelet count was normal in only 1 (8.3%), the other 92% were all reduced to a greater or lesser extent. Ten had counts of less than 100.

PI	10 failed to clot	PTT	11 failed to clot,
PI	1=10%	PTT	1=56

All PI and PTT's were grossly deranged. 91% of PI's and 92% of PTT's failed to clot.

Fibrinogen=0	8	FDP 120-240	2
=negligible	3	>240	10
=low	1		

All FDP's were in the extremes of elevation indicating that fibrinolysis and fibrin degradation had occurred.

Fibrinogen levels were zero in 67%, negligible in 25%, and low in 8.3%.

The above results are indicative of a marked coagulation defect resulting from contact with mesothelial cells, however, abnormalities may arise from varying degrees of clotting before the mesothelial effects.

Many patients have clots present at laparotomy or thoracotomy in the presence of blood lacking the ability to clot. This may explain the

25% incidence of Hb less than 10g% in blood salvaged for autotrarsfusion.

It is evident that in addition to the lack of clotting factors and platelets, large amounts of activated clotting factors and cells will be present.

Elevated FDP levels are significant inhibitors of platelet function, contributing to the bleeding tendency. As mentioned earlier in the literature review, platelets in autotransfused blood are probably largely inactive or have a very depressed response to activation.

The activated clotting factors will also tend to activate in vivo, the normal clotting cascade, thus leading to a DIC tendency. The activated platelets and white cells will release substances which have an effect on white cells in vitro. If they are released from microaggregates filtered in the pulmonary microvasculature, activated white cells lead to local and regional inflammation and thrombosis.

The findings in this study show a normal to elevated white cell count indicating that leucocyte numbers are probably not much affected in such blood.

Normal or near normal haemoglobin levels, in the presence of normal 2,3 DPG levels and normal erythrocyte survival means that oxygen carrying ability of autotransfused blood should be close to that of normal blood.

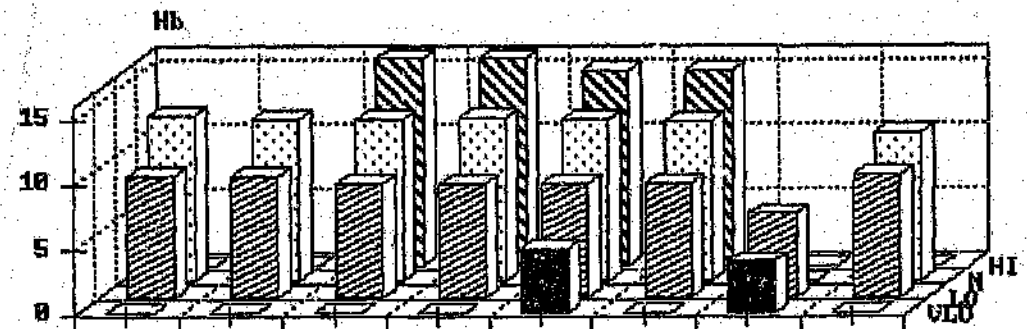
The haematological effects of autotransfused blood upon the host, will be con idered in the following sections.

10.6. COMPARATIVE ANALYSIS

10.6.1. HAEMOGLOBIN

VLO= 1-5	LO= 6-10	N= >10

Mean Hb

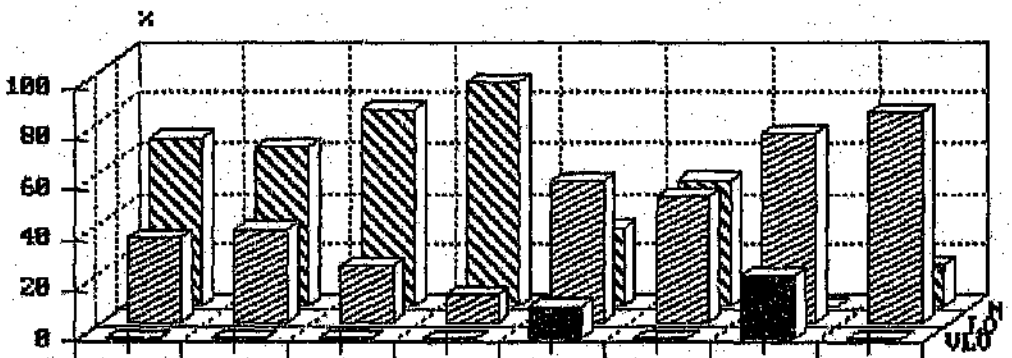


	Bank Adm	Bank Post	Auto Adm	Auto Post	Mix Adm	Mix Post	Ecto Adm	Ecto Post
HI	0	0	16	16	15	15	0	0
N	12.79	12.42	12.47	12.64	12.5	12.29	0	11.5
LO	9.29	9.29	8.67	8.67	8.67	8.75	6.44	9.4
VLO	0	0	0	0	5	0	4	0

Groups

Graph **Mean Hb**, above, compares the haemoglobin transfusion responses between the 4 groups. The **Banked, Auto,** and the **Mixed** groups had remarkably similar mean pre and post-transfusion Hb values, without noticeable rises in Hb levels. The **Ecto** group differs in that there were no normal pre-transfusion Hb levels; the mean Hb levels rose after transfusion.

% and Hb



	Bank Adm	Bank Post	Auto Adm	Auto Post	Mix Adm	Mix Post	Ecto Adm	Ecto Post
N	66.67	63.16	77.78	88.89	31.25	50	0	16.67
LO	33.33	36.84	22.22	11.11	56.25	50	75	83.33
VLO	0	0	0	0	12.5	0	25	0

Groups

The above graph, % and Hb, depicts the patient percentages in the groups and Hb subgroups.

The **banked** transfusion group did not exhibit an Hb rise following transfusion, in fact the normal Hb dropped by 3.51%.

The **autotransfusion** group increased the Hb by 11.11%.

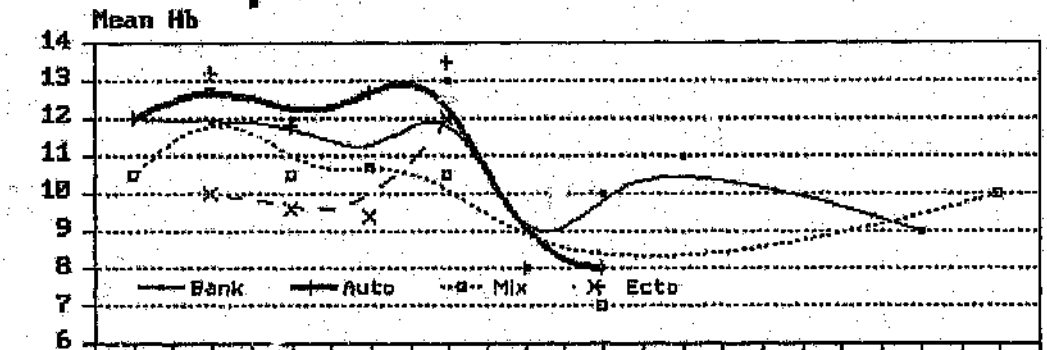
The **mixed** group had a low/ very low Hb of 68.75%, and a normal Hb of 31.25% on admission. This improved to 50% low and 50% normal after transfusion.

All **ectopic** patients had a low/ very low Hb on admission. After transfusion, the Hb shifted to a low/ normal of 83.33%/ 16.67% respectively.

The **ectopic** group had the largest proportion of low Hb's, but it must be borne in mind that these patients come from a population subset having low *"normal"* Hb's because of poor dietary iron intakes superimposed on pregnancy.

If one considers the three groups with autotransfusions, the majority had adequate Hb increases, and all had normal circulations restored.

Hb Response to Transfused Units



	1	2	3	4	5	6	7	8	9	10	17	21
Bank	12	11.9	12	10.7	13	8	10	11			9	
Auto	12	13.2	11.8	12.7	13.5	9	8					
Mix	10.5	12.7	10.5	10.7	10.5		7					10
Ecto		10	9.6	9.4	12							

Units Transfused

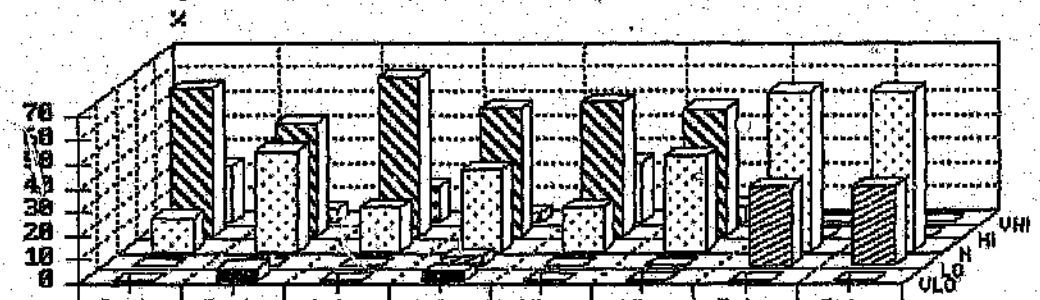
The above trend curve graph **Hb Response to Transfused Units**, shows the post transfusion mean Hb values related to the number of transfused units.

In all 4 groups, Hb levels remained steady until 5 units; thereafter levels tended to drop. Accuracy is probably true for up to and including 5 units, as the numbers of patients for higher volumes are too small for accuracy. The **mixed** group curve was represented by autotransfused units only.

10.6.2. WCC

VLO=1-2	LO=3-4	N=5-11	HI=12-20	VHI=21-99

% and White Cell Counts



	Bank Adm	Bank Post	Auto Adm	Auto Post	Mix Adm	Mix Post	Ecto Adm	Ecto Post
VHI	23.81	5.26	14.81	3.85	25	6.67	0	0
HI	61.9	47.37	66.67	53.85	56.25	53.33	0	0
N	14.29	42.11	18.52	34.62	18.75	40	66.67	66.67
LO	0	0	0	3.85	0	0	33.33	33.33
VLO	0	5.26	0	3.85	0	0	0	0

Groups

At least 81.25% (81.25%-85.71%) of the trauma patients had an elevated admission WCC on admission. This contrasts sharply with the **ectopic** patients where no elevations of the WCC were seen.

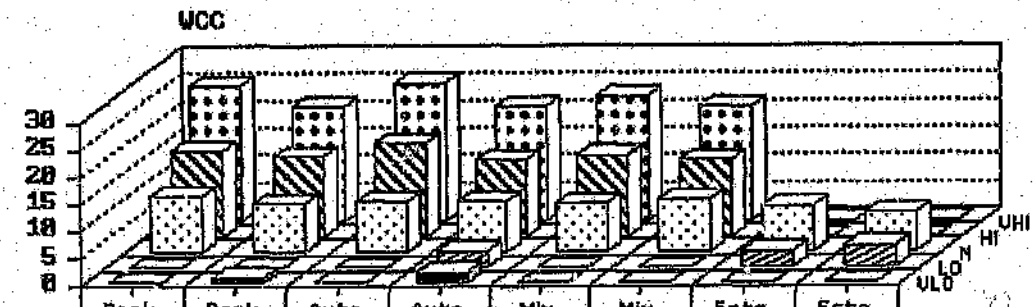
The second post-transfusion WCC's showed reductions towards normality; only between 52.63% and 60% had elevated WCC's.

The **ectopic** group's WCC remained unchanged after transfusion.

There were no post-transfusion leucopaenias in the **mixed** group, while the **banked** and **autotransfused** groups had minor reductions in the post-transfusion WCC's. Only the **Bank** and **auto** groups exhibited leucopaenia, 5.26% and 7.7% respectively. It is evident that transfusions of any type had little effect in causing leucopaenia.

The Graph **Mean WCC**, below, shows the mean values of the admission and the first post-transfusion WCC's in each group, and cell count subgroup.

Mean White Cell Counts



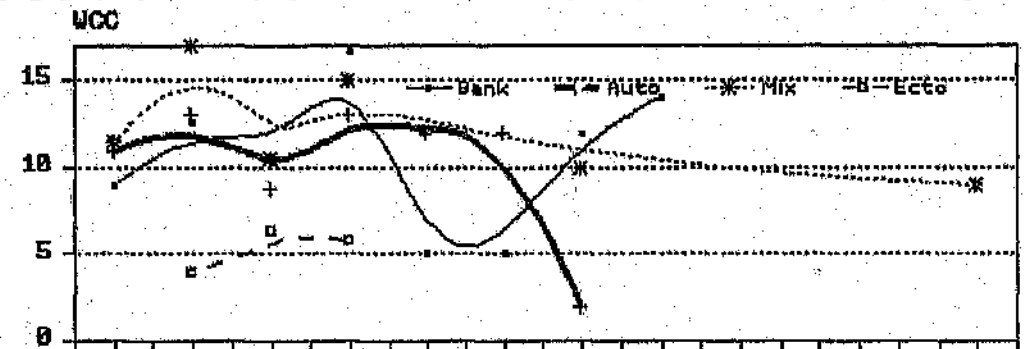
	Bank Adm	Bank Post	Auto Adm	Auto Post	Mix Adm	Mix Post	Ecto Adm	Ecto Post
VHI	25	21	25.5	21	23	21	0	0
HI	16	14.89	17.39	14.21	15.11	14.12	0	0
N	10.33	9.12	9.4	9.44	9	9.67	8.5	7.25
LO	0	0	0	3	0	0	3	4
VLO	0	1	0	2	0	0	0	0

Groups

The trend curve graph **Post-Transfusion Mean WCC** below, shows the mean WCC in relation to transfusion volumes.

Cell counts remained relatively stable for all groups, except for the drop in the higher Auto volumes. Low patient numbers probably skewed the curves at higher volumes.

Post-transfusion Mean White Cell Count



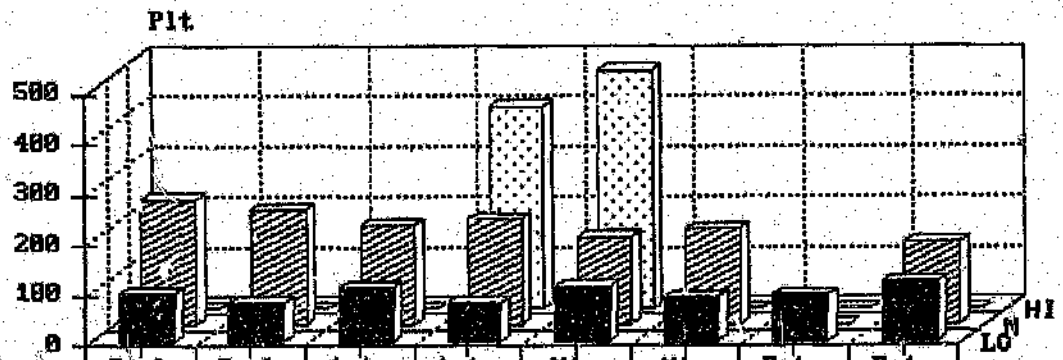
	1	2	3	4	5	6	7	8	9	10	17	21
Bank	9	12.6	10.3	16.7	5	5	12	14				
Auto	11	13	8.8	13	12	12	2					
Mix	11.5	17	10.5	15			10					9
Ecto		4	6.4	5.8								

Transfused Units

10.6.3. PLATELETS

LO=1-140	N=141-400	HI=401-999

Mean Platelet Counts

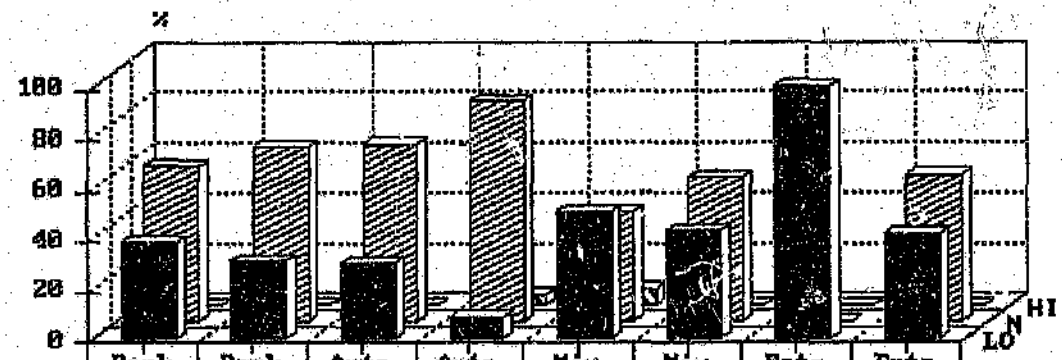


	Bank 1	Bank 2	Auto 1	Auto 2	Mix 1	Mix 2	Ecto 1	Ecto 2
HI	0	0	0	403	475	0	0	0
N	250.15	230	198.26	289.71	174.43	191.25	0	164.86
LO	95.12	76.75	107.75	76.5	108.62	88.5	91.25	119

Groups

The above graph, Mean Platelet Counts, displays the mean platelet counts in the major groups and subgroups.

% and Platelets



	Bank 1	Bank 2	Auto 1	Auto 2	Mix 1	Mix 2	Ecto 1	Ecto 2
HI	0	0	0	4.17	6.25	0	0	0
N	61.9	69.23	70.37	87.5	43.75	57.14	0	58.33
LO	38.1	30.77	29.63	8.33	50	42.86	100	41.67

Groups

Assuming that pre-transfusion platelet counts were normal in the trauma group, all patients had noticeable reductions in platelet counts after transfusion.

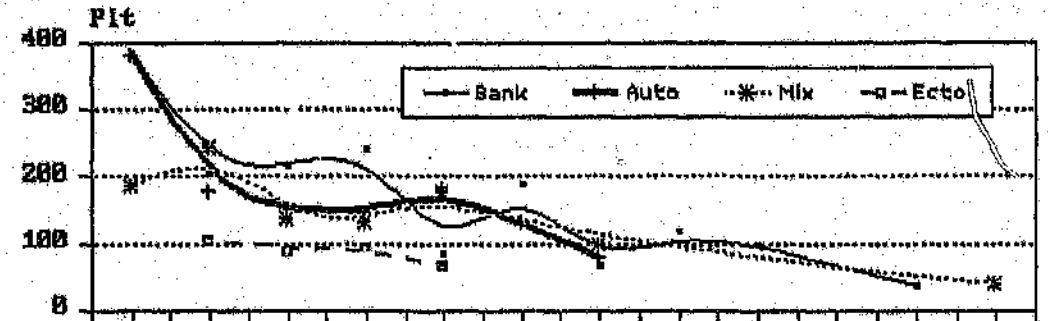
On the first post-transfusion day, of the trauma patients, the **mixed** group had the greatest reduction (50% low), followed by the **banked** group (38.15%), while the **autotransfused** group had the lowest value of low counts, (29.63%). The greater drop in counts in the mixed group may be due to the fact that these patients had, on average, a larger volume of transfused blood.

The autotransfused (only) group improved to 91.67% normal counts by day two, however, the banked and mixed groups were little changed compared to the first post-transfusion counts.

All **ectopic** patients had a low post-transfusion count, but this had improved to 41.47% by day two. The low counts may have been contributed to by hormonal effects of pregnancy on platelet counts.

Transfusions vs Mean Platelet Counts

First



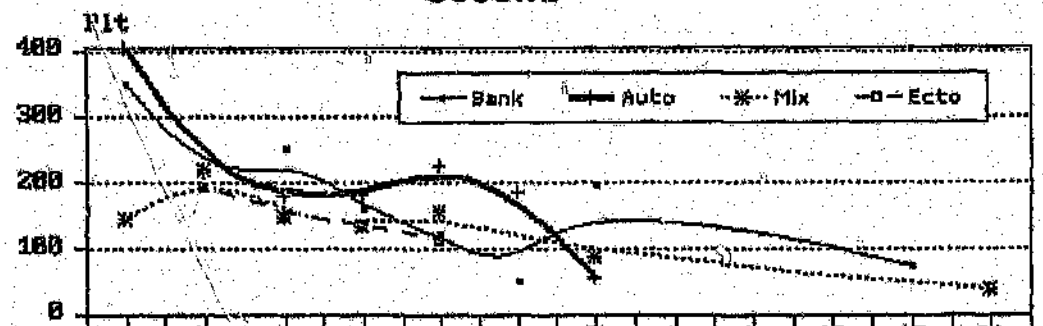
	1	2	3	4	5	6	7	8	9	10	17	21
Bank	385	284.6	218	241.3	85	191	67	120	100		37	
Auto	383	177.8	153.2	146.5	185	132	81					
Mix	187	244.3	139.5	134.3	179		100					39
Ecto		106	91.2	93.2	67							

Transfused Units

The above trend curve graph **Transfusions vs Mean PLT Counts, First,** representing mean platelet counts on the first day after transfusion, shows an obvious reduction of counts with rising transfusion volumes. All groups had similar responses; the **ectopic** group had the lowest counts for equivalent volumes. Graph **Transfusions vs Mean PLT Counts, Second,** below, representing the mean platelet counts on day 2, shows remarkably similar distributions to day 1. All 4 groups were similar, and the **ectopic** curve was now closely associated with the other groups.

Transfusions vs Mean Platelet Counts

Second



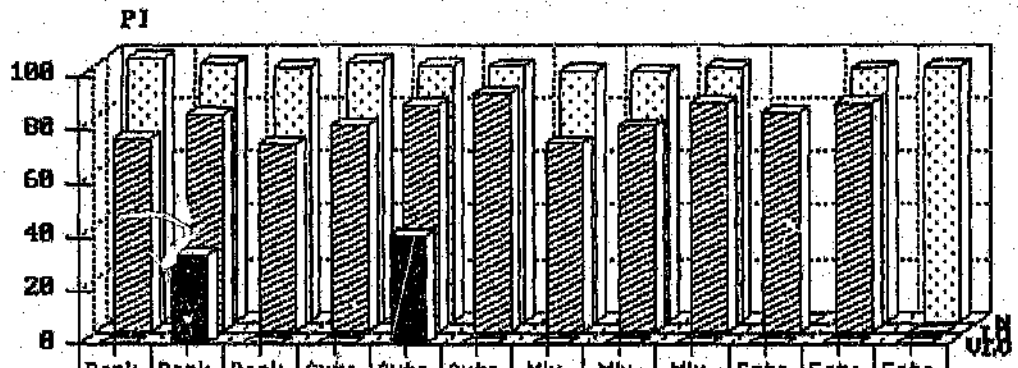
	1	2	3	4	5	6	7	8	9	10	17	21
Bank	350	194.7	252	160.3	123	53	197				74	
Auto	403	218.9	161.8	182.5	227.5	187	57					
Mix	147.5	222	143.7	136.3	157		88					41
Ecto		194	149.7	138.6	116							

Transfused Units

10.6.4. PI

Very Low=1-50	Low=51-89	Normal=90-99

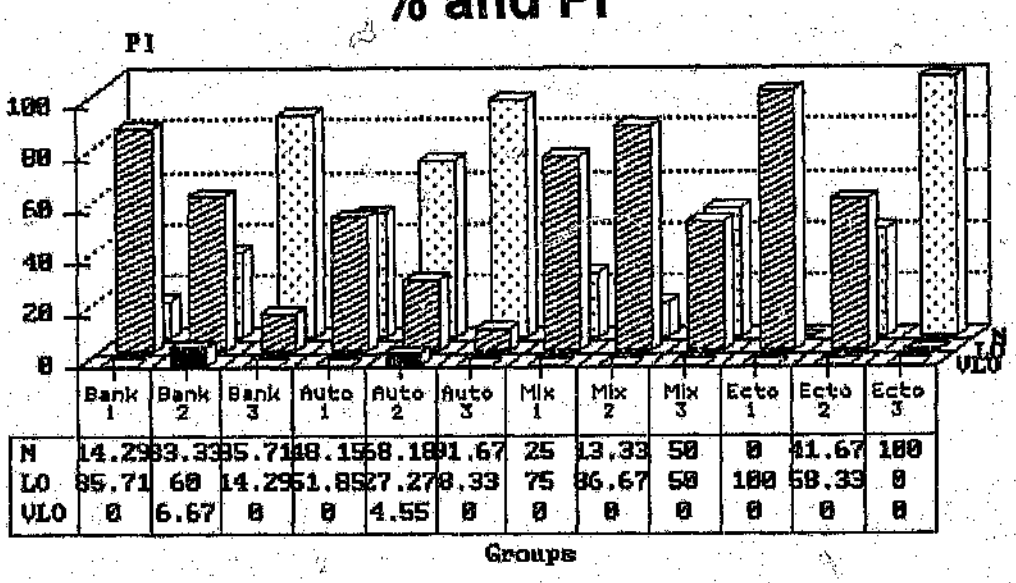
Mean PI



	Bank 1	Bank 2	Bank 3	Auto 1	Auto 2	Auto 3	Mix 1	Mix 2	Mix 3	Ecto 1	Ecto 2	Ecto 3
N	98.33	96.2	95	96.85	95.6	95.18	93.5	93	94.8	0	94.4	95
LO	72	81.33	70	77.87	84.83	89	78.33	76.62	85.4	92.17	85.43	0
VLO	0	33	0	0	40	0	0	0	0	0	0	0

Groups

The Graph Mean PI above, indicates the mean PI values in each group and count subgroups. It is noted that the initial post-transfusion PI had no normal values. This rapidly improved as seen with subsequent PI's.

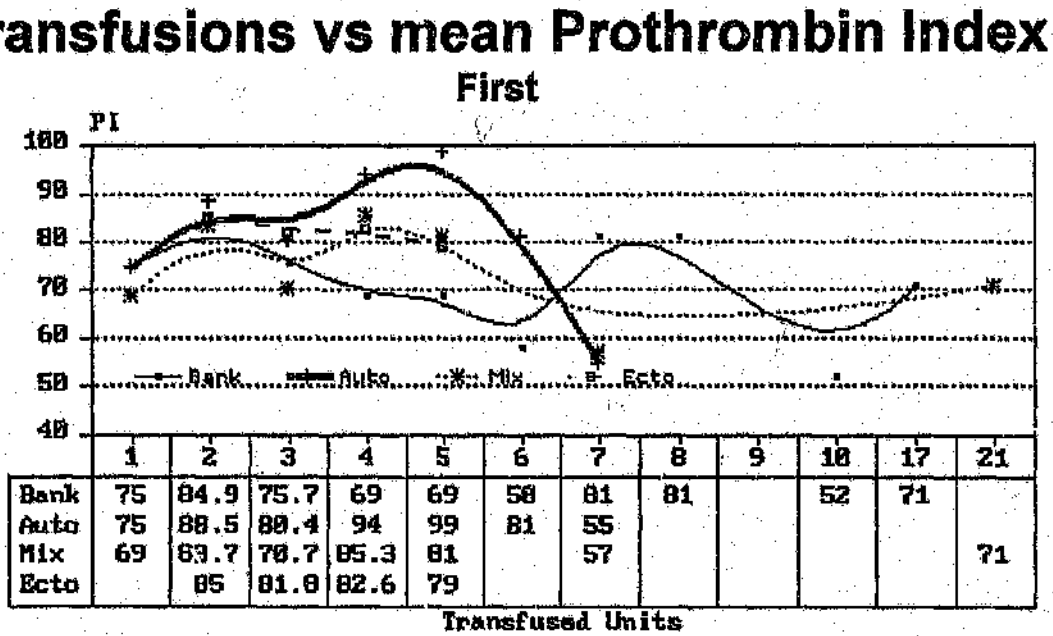


Graph **% and PI** above, demonstrates the % of patients within the groups and PI subgroups.

All 4 groups demonstrated substantial reductions of the first post-transfusion PI's, all except the **mixed** group improved each day and had virtually returned to normal values by the 3rd day.

The **autotransfused** group showed the least declines in PI's, while the **ectopic** group had no normal values initially, all of the latter had returned to normal by the third day.

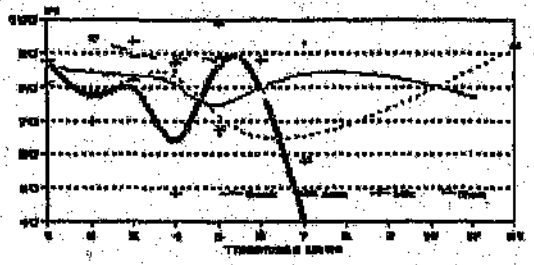
In the case of the **mixed** group, 75% were initially low, and had deteriorated further to 86.67% by the second day. The PI values were normal in only 50% of patients by the third post-transfusion day.



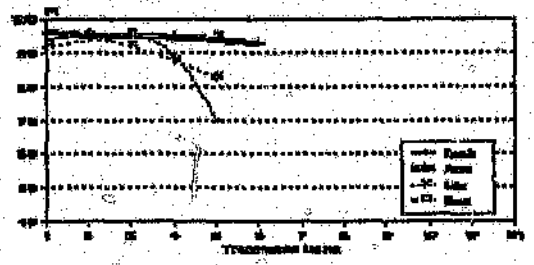
Graphs **TRANSFUSIONS vs PI 1, 2, and 3** demonstrate the mean PI values as a function of transfusion volume. The day 1 PI's were virtually all low, and fairly similar between groups. Day 2's PI curves were much flatter, but still below normal, while the curves of Day 3 were much more normal, with deterioration towards volumes above 4 units.

Transfusions vs Mean Prothrombin Index

Second



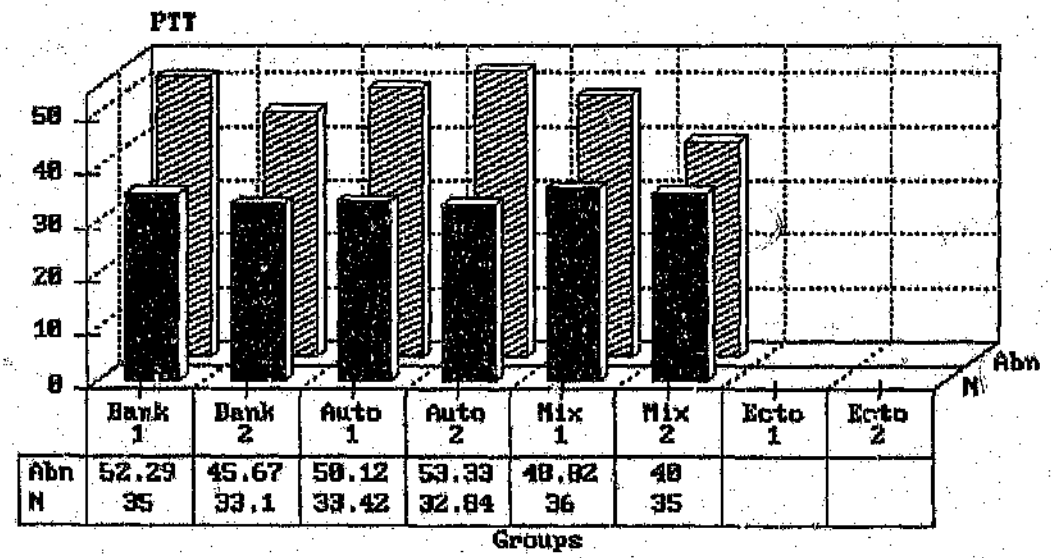
Third



10.6.5. PTT

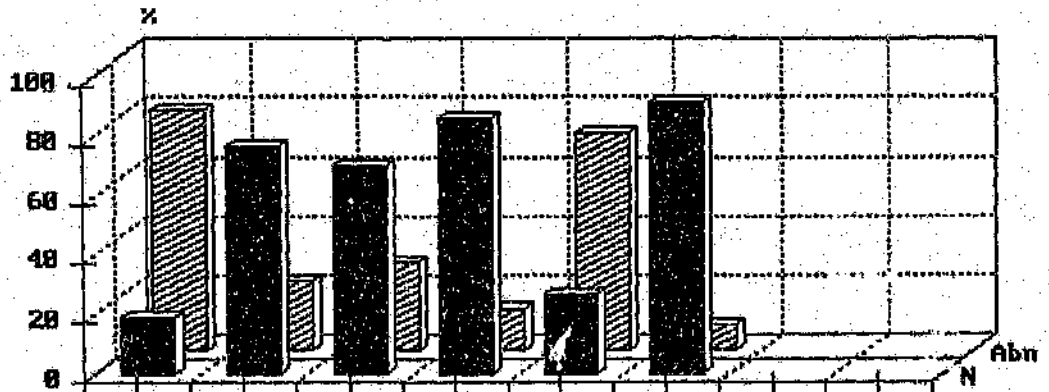
NORMAL=28-39 ABN=40-99

Mean PTT



The above graph Mean PTT indicates mean PTT values in the groups and PTT subgroups.

% and PTT



	Bank 1	Bank 2	Auto 1	Auto 2	Mix 1	Mix 2	Ecto 1	Ecto 2
Abn	80.95	23.08	29.63	13.64	73.33	8.33		
N	19.05	76.92	70.37	86.36	26.67	91.67		

Groups

Graph % and PTT, above, demonstrates graphically the patient distribution in groups and PTT subgroups.

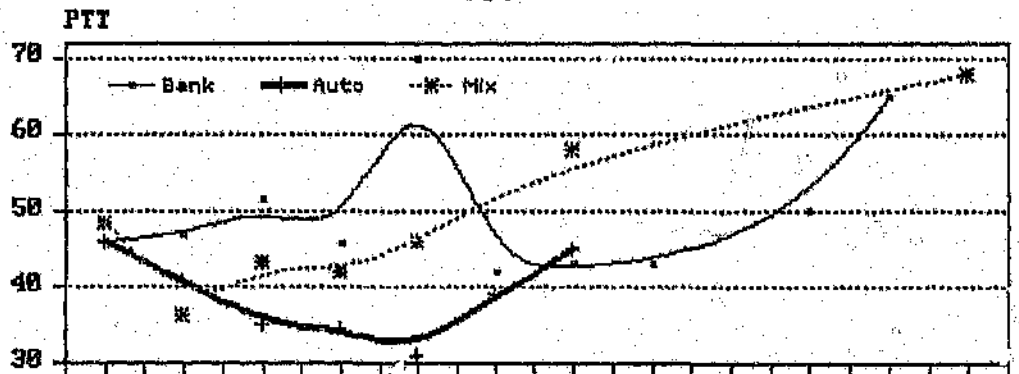
The **banked** and **mixed** groups had similar trends. The **banked** were 80.95% abnormal on day 1, and had improved to 76.92% normal at day 2. The **mixed** group had figures of 73.33% and 91.67% respectively.

In contrast, the **autotransfused** group had 70.37% of PTT's normal on day 1, and improved further to 86.36% on day 2.

Both groups who had received banked blood had initial normal values of at least 2.5 fold less than the groups who were only autotransfused.

Transfusions vs Mean PTT

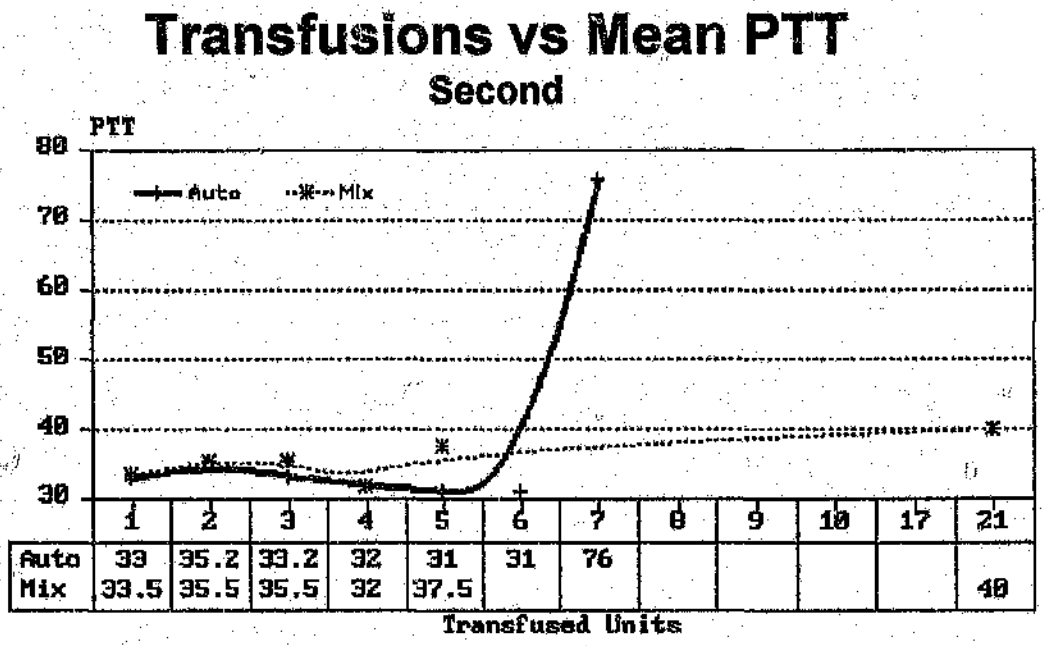
First



	1	2	3	4	5	6	7	8	9	10	17	21
Bank	46	46.7	51.5	45.7	70	42	43	43		50	65	
Auto	46	40.8	35	34.7	31	39	45					
Mix	40.5	36.3	43.2	42	46		58					68

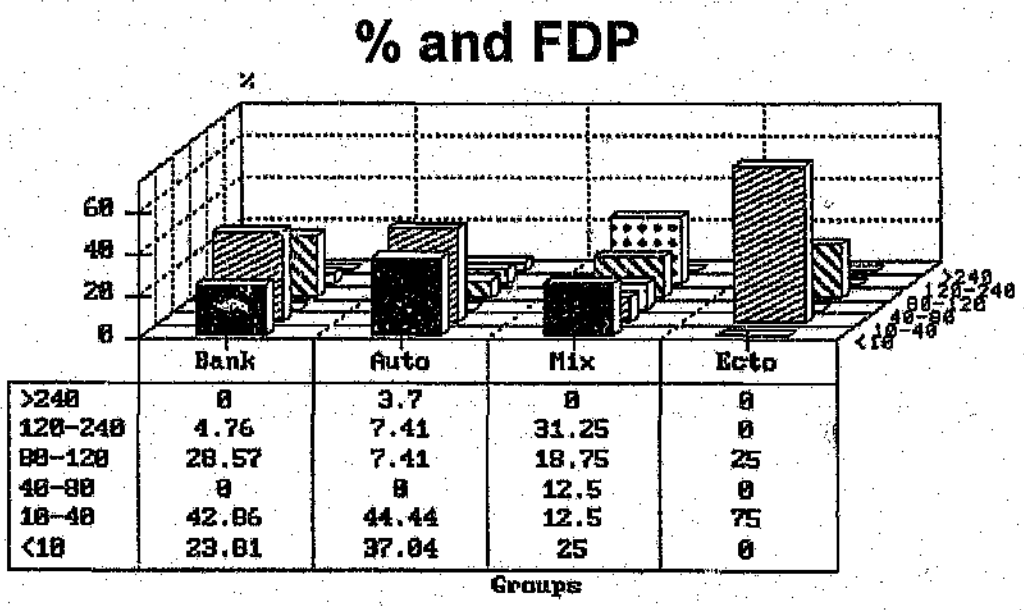
Transfused Units

Graph **Transfusions vs Mean PTT, First**, above, depicts the mean PTT values on day 1. The groups **bank** and **mix** had rising curves in the abnormal range, while the curve of the **auto** group was mostly normal, and falling. The day 2 curves had returned to normal levels, as seen below, in graph **Transfusions vs MEAN PTT, Second.**

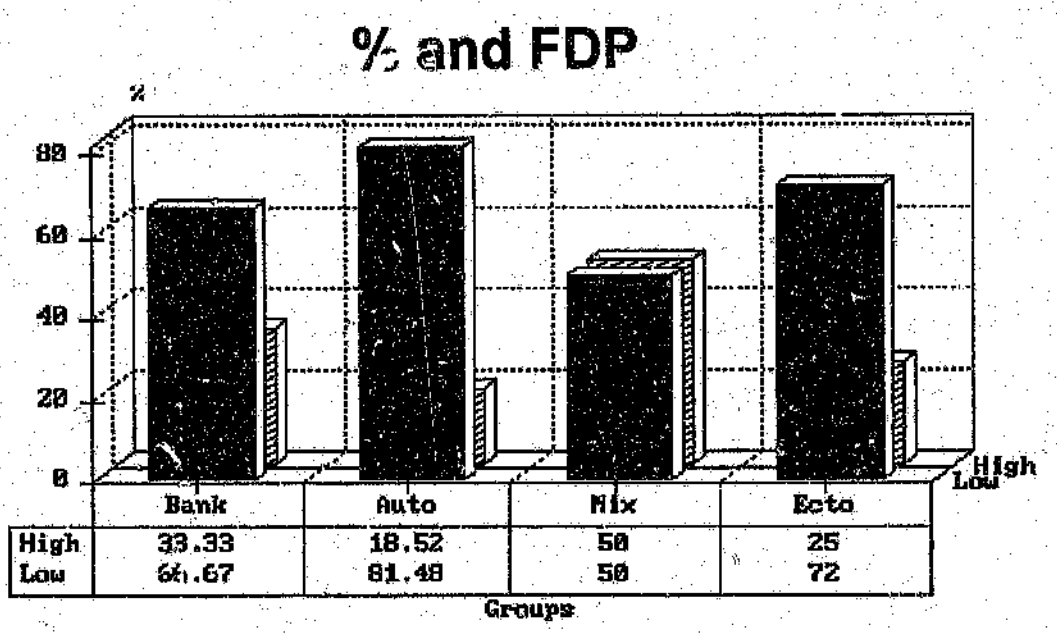


10.6.6. FDP'S

Graph % and FDP illustrates the patient distribution within groups and FDP subgroups.

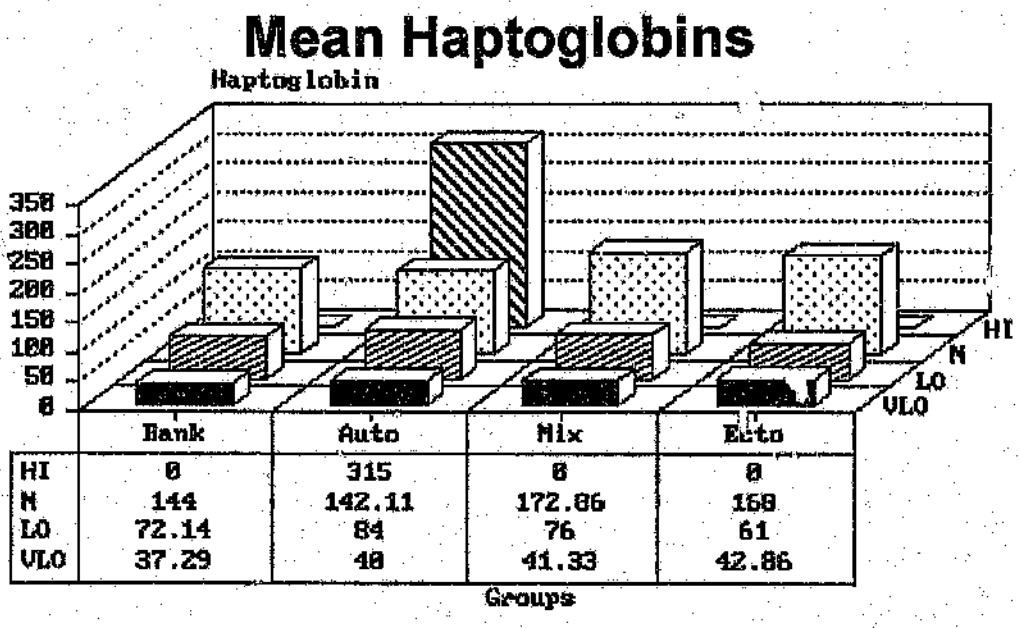


If one divides the FDP results into 2 categories, one of minimal to mild increases (<10 & 10-40), and the other of moderate to severe increases (40-80 - >240), the following results are obtained.



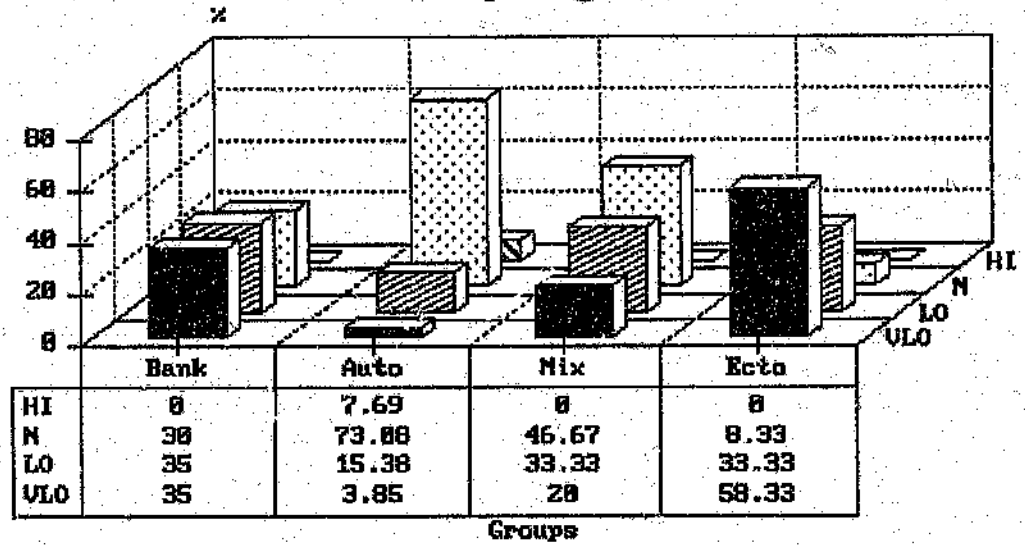
The **autotransfused** group had 81.48% in the more normal category, the **ectopics** 75%, and the **banked** group 66.67%. The **mixed** group was dissimilar in that only 37.5% were found in the more normal category. This is graphically illustrated in the simplified graph **%FDP**.

10.6.7. HAPTOGLOBINS



Haptoglobins are acute phase reactant proteins that bind free circulating haemoglobin. Graph **Mean Haptoglobins** shows the mean values within groups and subgroups. The means within the VLO, LO, and N subgroups are fairly similar, however, the **auto** group was the only group with haptoglobin levels above the upper limits of normal.

% and Haptoglobins



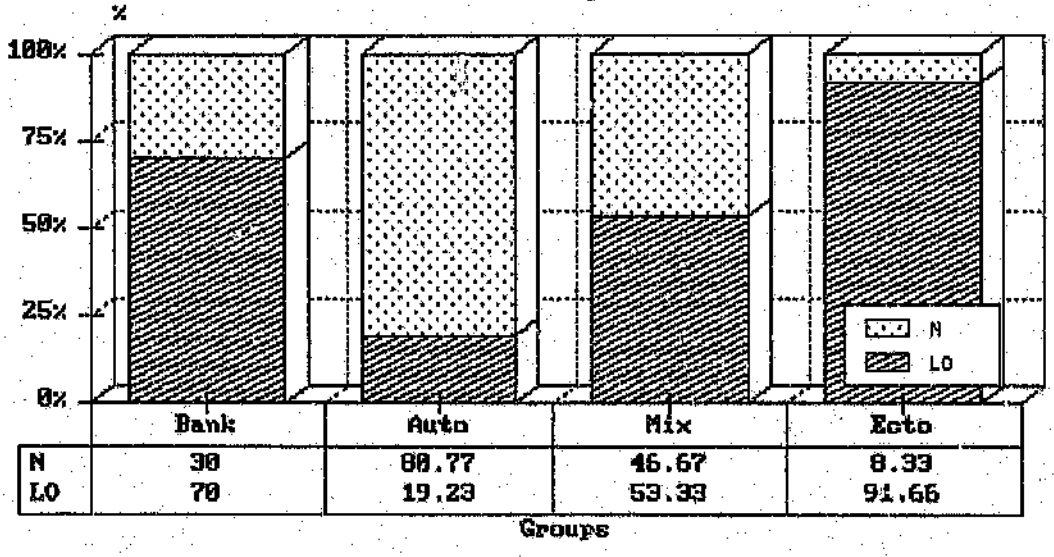
	Bank	Auto	Mix	Ecto
HI	0	7.69	0	0
N	30	73.08	46.67	8.33
LO	35	15.38	33.33	33.33
VLO	35	3.85	20	58.33

VLO=1-50 LO=51-99 N=100-300 HI=301-999

The graph **% and Haptoglobins** below, shows the patient distribution within major groups and haptoglobin subgroups.

If these results are divided into 2 categories, one with very low or low haptoglobin levels, the other with normal or high levels, the following distributions are noted.

% and Haptoglobins



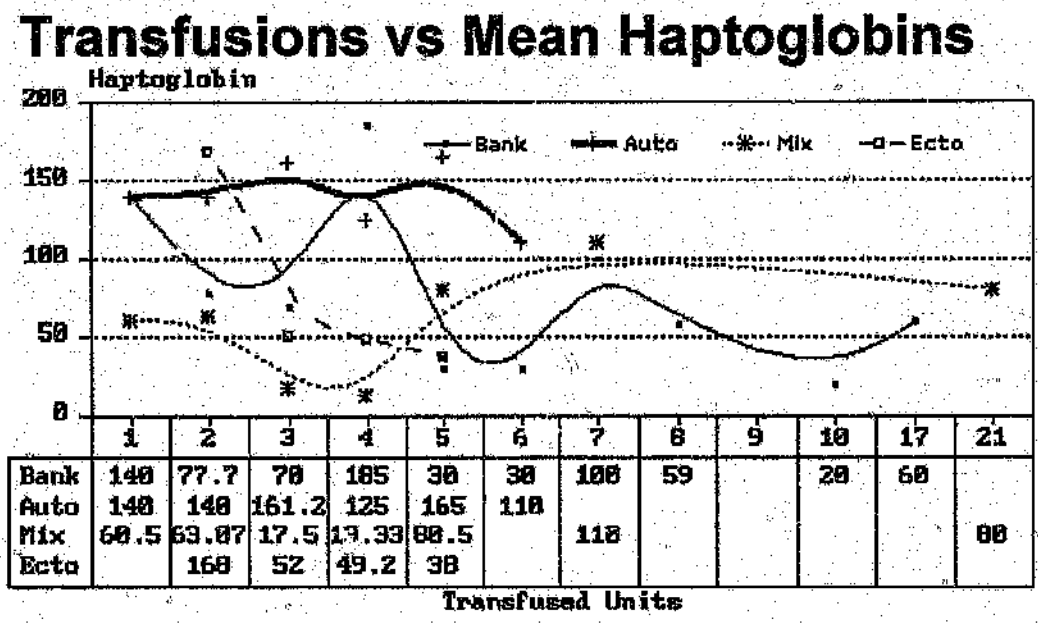
	Bank	Auto	Mix	Ecto
N	30	80.77	46.67	8.33
LO	70	19.23	53.33	91.66

The **autotransfusion** group was found to have 80.77% within the HI/N category as opposed to only 8.33% with the **ectopic** group. This large difference within 2 autotransfusion populations is difficult to explain. It is obvious that a greater degree of haemolysis had occurred in the **ectopic** patients, as evidenced by reduced levels of

haptoglobins, and the observed increase in macroscopic haematuria in the ectopic group.

Only 30% of the **banked** group were found in the N/ HI category, and the mixed group was similar with 46.67%.

The **autotransfused**-only trauma patients again demonstrated lesser abnormalities than the recipients of banked blood.

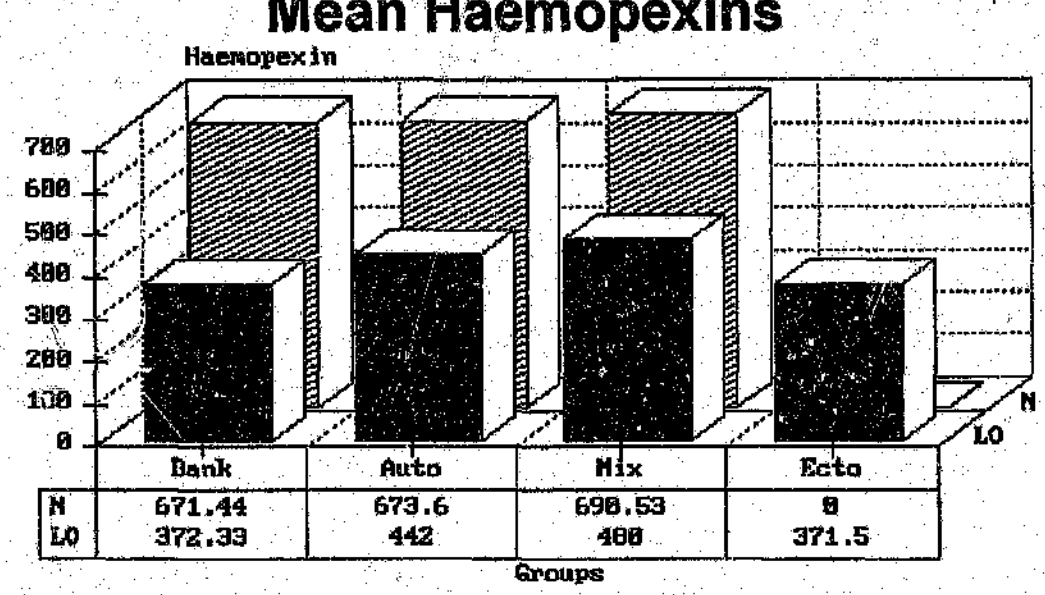


	1	2	3	4	5	6	7	8	9	10	17	21
Bank	140	77.7	70	105	30	30	100	59		20	60	
Auto	140	140	161.2	125	165	110						
Mix	60.5	63.07	17.5	19.33	80.5		110					80
Ecto		160	52	49.2	30							

Graph **Transfusions vs Mean Haptoglobins** above, depicts the mean haptoglobin responses to transfusion volumes. It shows that the **auto** curve fell within normal levels, while the **mixed** group was subnormal at all times. The remaining groups started with normal values, and fell with rising volumes.

10.6.8. HAEMOPEXINS

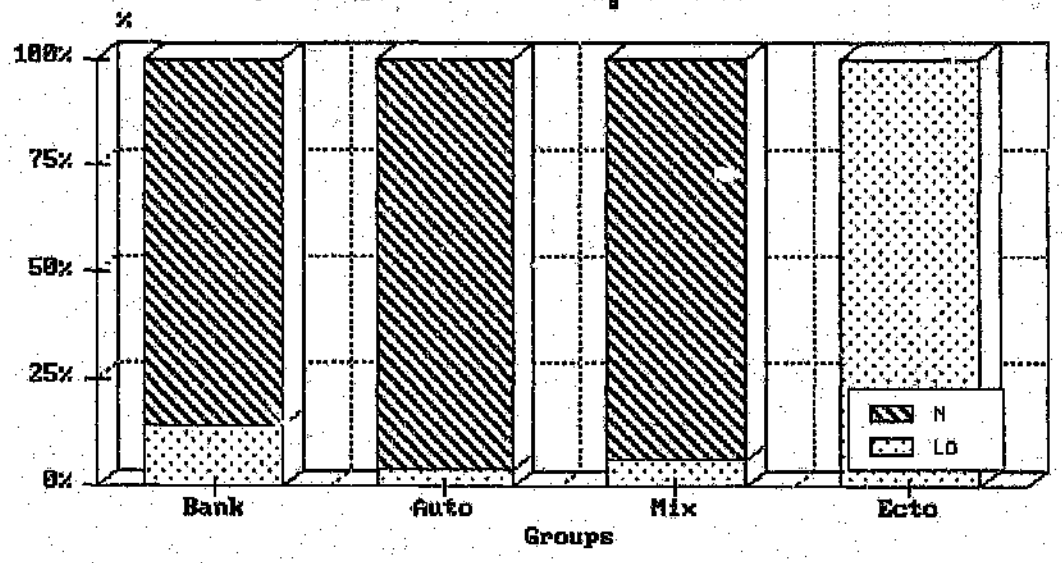
Haemopexins are also haemoglobin scavengers, functioning as a backup when haptoglobin levels are low.



LO=1-500 N=501-999

The above graph **Mean Haemopexins**, illustrates the mean haemopexin levels of the major groups and haemopexin subgroups.

In the case of haemopexins, the results are divided into two categories; a group with low levels, and a group with normal levels. The graph **% and Haemopexins** below, illustrates these values.



Three of the four transfusion groups were found to have haemopexin levels falling within the "normal" category; autotransfusions 96.15%, mixed 93.75%, and the banked group with 85.71%.

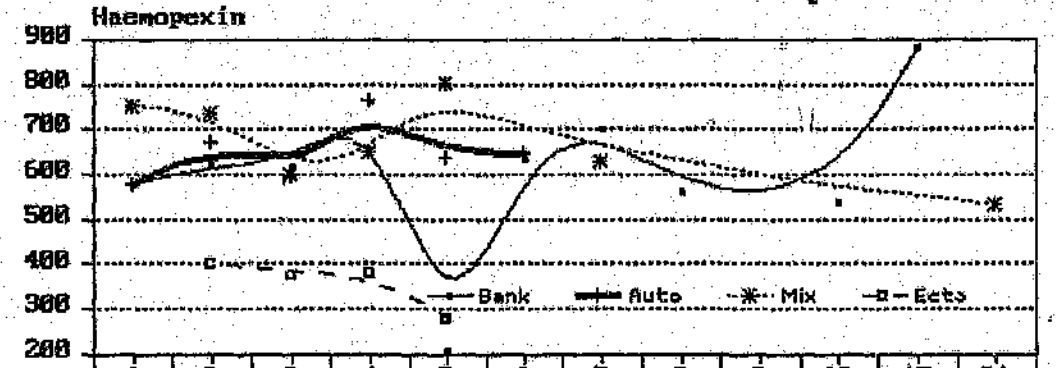
In contrast to these results, all patients in the ectopic group had low haemopexin levels. This is indicative of haemolysis in the

pretransfusion period, as the collection methods, and delays were no different to the trauma groups.

The results of haemopexin levels parallel those of the preceding haptoglobin levels.

As in the preceding section, it is apparent that trauma patients in the autotransfusion-only group demonstrate lesser deviations from normal than do groups receiving banked blood or combinations of banked and salvaged blood.

Transfusions vs Mean Haemopexins

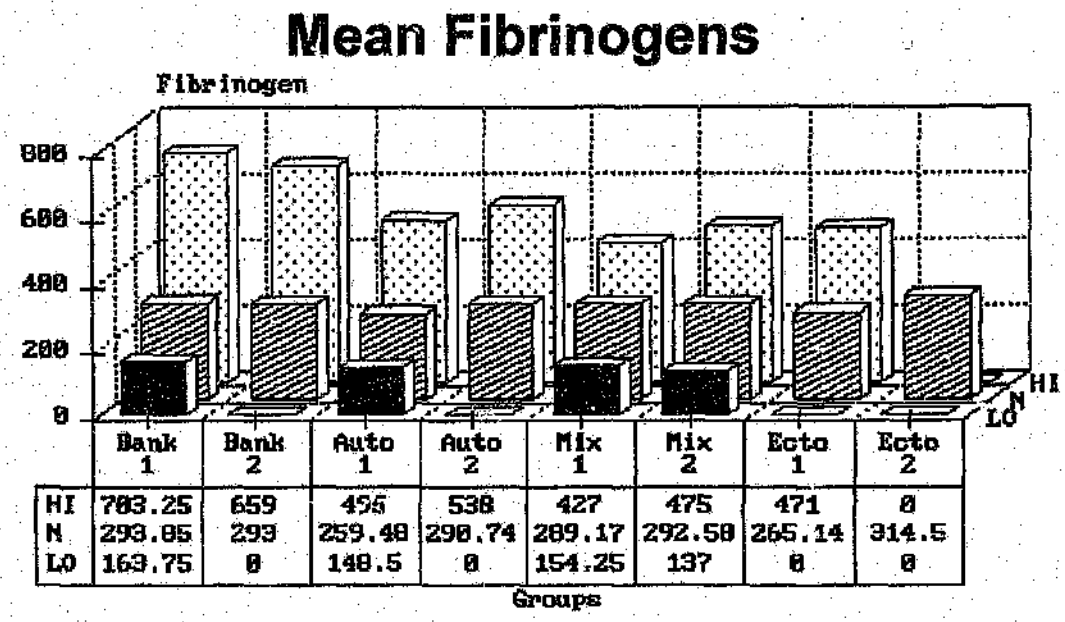


	1	2	3	4	5	6	7	8	9	10	17	21
Bank	580	620.9	618.5	759.7	207	634	701	563		535	884	
Auto	580	672.4	595.5	765.2	638.5	646						
Mix	753.5	735	601	651	803		630					530
Ecto		400	376.8	378.8	200							

Transfused Units

Graph **Transfusions vs Haemopexins** above, demonstrates the mean haemopexin levels as a function of transfusion volume. The **ectopic** group curve was well below normal, while the remaining groups were within normal limits.

10.6.9. FIBRINOGEN

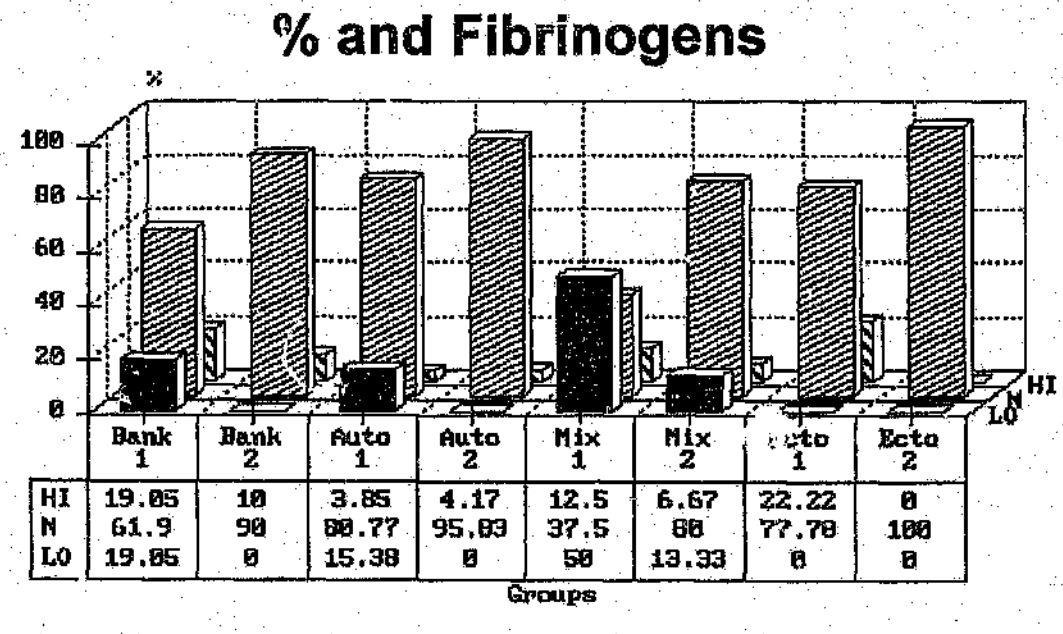


	Bank 1	Bank 2	Auto 1	Auto 2	Mix 1	Mix 2	Ecto 1	Ecto 2
HI	703.25	659	455	538	427	475	471	0
N	293.85	293	259.48	290.74	289.17	292.58	265.14	314.5
LO	163.75	0	148.5	0	154.25	137	0	0

LO=1-199	N=200-400	HI=401-999

Graph **Mean Fibrinogens** above, demonstrates the mean post-transfusion fibrinogen levels in the major groups and fibrinogen subgroups. The **banked** group was noted to have higher mean levels in the HI range than other groups.

Fibrinogen levels were tested on the first and second mornings after transfusion.



	Bank 1	Bank 2	Auto 1	Auto 2	Mix 1	Mix 2	Ecto 1	Ecto 2
HI	19.05	10	3.85	4.17	12.5	6.67	22.22	0
N	61.9	90	80.77	95.83	37.5	80	77.78	100
LO	19.05	0	15.38	0	50	13.33	0	0

Graph **% and Fibrinogens** above, illustrates the patient distribution within groups and subgroups.

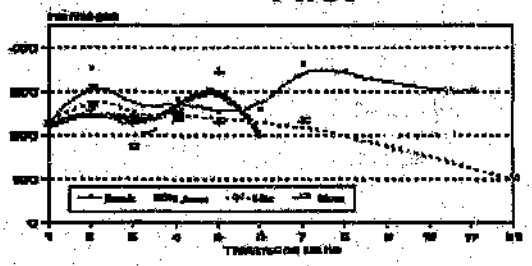
On the first day, three of the four groups had levels below normal, **mixed** 50%, **banked** 19.05%, **autotransfused** 15.38%, but by the second day, all four transfusion groups had at least 86,67% in the normal or elevated range of fibrinogen levels.

The **mixed** group was the only one still with reduced levels (13.33%).

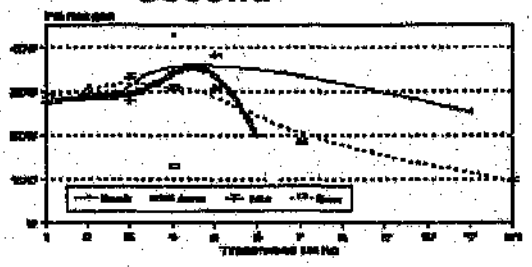
Up to 22.22% of patients in any one group had elevated fibrinogen levels within 24 hrs of transfusion. This is in spite of the above evidence of coagulation activation and clot lysis. The **auto** group had the smallest increase in blood levels above the upper limits of normal of fibrinogen, in both post-transfusion tests. Fibrinogen, being an acute phase reactant, does increase in level following stress.

Transfusions vs Mean Fibrinogen

First



Second



Graphs **Transfusions vs Fibrinogen First and Second** depict the mean fibrinogen responses to transfused blood volume on days 1 and 2. Both graphs had similar trends, and the group curves were mostly normal.

10.6.10. COMPLICATIONS

10.6.10.1. Deaths

There were two deaths in this study. Both patients sustained multiple injuries as pedestrians in motor vehicle accidents.

The first patient was a 39 year old female admitted severely shocked with blunt abdominal and chest trauma. Emergency room radiography revealed a haemothorax for which an intercostal drain was inserted.

The salvaged blood amounted to 6 units of blood in total. This was autotransfused; in addition a further 4 units of banked emergency Rh -ve blood was transfused intraoperatively. No intra-abominal injury was found. A right pulmonary hilar injury was discovered during

emergency thoracotomy requiring a lobectomy. Severe intraoperative hypotension culminated in an intraoperative cardiac arrest, which, despite intensive efforts, did not respond to resuscitation.

No haematological tests were performed on this patient owing to the urgent nature of her condition, however, a coagulopathy was noted intraoperatively, the cause of which was probably secondary to extensive blunt soft tissue trauma with release of tissue thromboplastins, although the autotransfusion may have contributed to the development of her DIC.

The second patient was a 35 yr male admitted following a motor vehicle accident involving the victim as a pedestrian. He was found to be shocked, semi-confused, and with fractures of the pelvis, femur, tibia, fibula and ribs. Emergency room radiography excluded a thoracic cause for the shock.

A slightly distended abdomen with fractured ribs overlying the spleen led to a DPL being performed which revealed frank blood. While awaiting theatre, 3 units of blood were collected by this means.

A lacerated spleen was found at laparotomy; without bowel injury. The salvaged blood, collected pre-operatively, was then transfused, followed by a further 4 unit autotransfusion salvaged via the sorenson autotransfusion device. By this stage a stable circulation had been achieved after autotransfusion. Towards the end of the procedure however, it was noted that a coagulation defect had developed, resulting in diffuse wound oozing

Postoperatively he was admitted to the ICU where he was cardiovascularly stable. The DIC worsened and he developed pulmonary oedema and acute renal failure, culminating in a refractory cardiac arrest.

Once again, autotransfusion established a normal circulation and avoided the use of banked blood.

The admission haemoglobin before resuscitation was 11g%, with a WCC of 10 000mm^{-3} and a reduced platelet count of 81 000mm^{-3}.

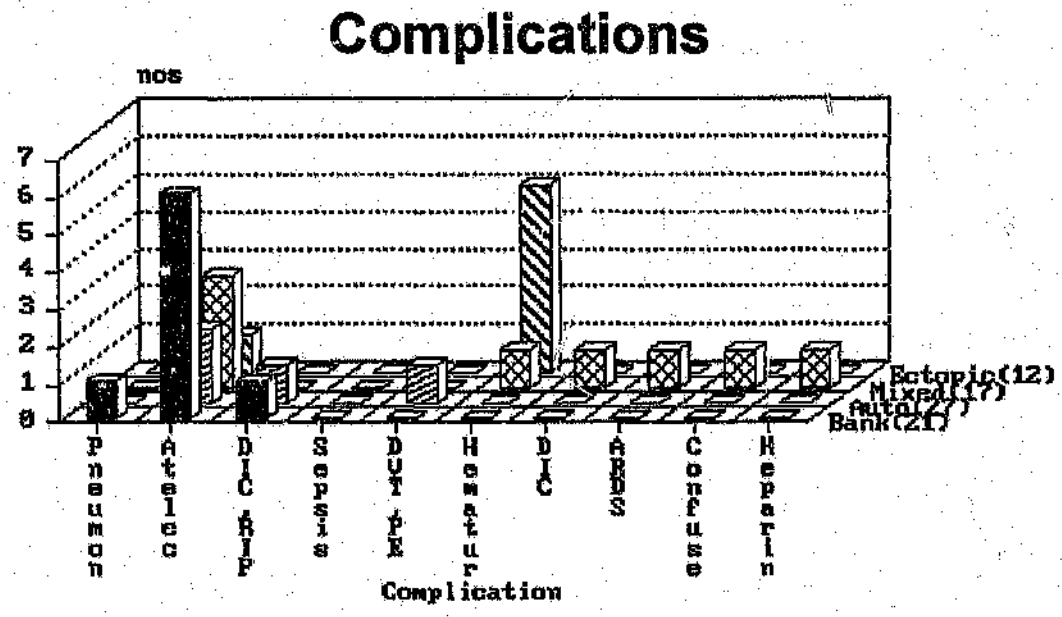
The following morning in ICU, his FBC results showed a Hb=8, a leucopaenia of 2 and a thrombocytopaenia of 56 000mm^{-3}. The PI was 45% and PTT was significantly prolonged. The fibrinogen degradation products was markedly elevated.

The DIC became apparent intraoperatively, probably triggered by the hypotension and the multiple soft tissue trauma, but was probably contributed to by the large volume of autotransfused blood.

10.6.10.2. Complications

		bank	auto	mix	ectopic
pneumonia:		1	0	0	0
atelectasis:		6	2	3	1
DIC,ATN,ARDS	intraop Death	1	1	0	0
Sepsis:		0	0	0	0
DVT/PE:		0	1	0	0
Haematuria:		0	0	1	5
DIC:		0	0	1	0
ARDS:		0	0	1	0
Encephalopathy:		0	0	1	0
Heparin coagulopathy:		0	0	1	0

The encephalopathy occurred in a patient with pre-existing alcoholic cirrhosis, was transient, and reversed with appropriate therapy.



The graph **Complications** above, shows graphically the complications seen in this trial. Of note, not a single septic complication occurred.

Of the 21 patients who received banked blood only, 8 (38%) developed complications, all respiratory related.

Of the 27 patients who were transfused with salvaged blood only, 4 developed complications (31%). The case of aspiration pneumonia should not be considered a complication, in that the event occurred before admission.

Of the 17 patients who were transfused with both banked and salvaged blood, 7 developed complications. Three were atelectasis, 1 haematuria, and 1 a heparin coagulopathy.

The latter patient was a 20yr male who presented with a large haemothorax after being stabbed in the right chest. The intercostal drain continued to drain blood and while awaiting thoracotomy, 21 units of blood were salvaged and autotransfused. Intraoperatively a further 8 units of banked blood were transfused.

A bleeding tendency was noted intraoperatively, which was suspected to be partly due to the heparin in the salvaged blood (500u per litre, 10 500u total). Intravenous protamine, a specific heparin antagonist was administered, and the oozing appeared to stop. The patient made an uneventful postoperative recovery.

Again, the use of blood salvage and autotransfusion significantly reduced banked blood usage and maintained a good circulation. His pre-operative Hb was 8,5 and postoperatively was 9,5 after 29 units in total. Four units of FFP were added during the course of his transfusions.

The admission platelet count was 39 000, which rose to 49 000 the morning after his surgery.

The postoperative PI was 71% while the PTT was mildly elevated. Haptoglobin and haemopexin levels dropped to below normal, indicating that some degree of haemolysis was associated with his transfusions.

The fibrinogen levels dropped markedly, this can be explained on the basis of two mechanisms.

1 The transfusion of defibrinated blood.
2 The activation of the clotting system with consequent fibrinogen consumption.

Of the 12 patients with ruptured ectopic pregnancies, 5 developed complications:

Five patients were noted to have transient haematuria, probably from haemolysed autotransfused blood. No renal or electrolyte abnormalities were noted in any patients. One of these patients developed a basal atelectasis.

Another patient, not to be considered as a complication, had transient confusion following a hypovolaemic cardiac arrest.

She was a 30 year female with an exsanguinating haemorrhage from a ruptured tubal ectopic pregnancy. She had a hypovolaemic cardiac arrest on arrival in casualty. At assessment, no obvious evidence of trauma was apparent, but a significantly distended abdomen associated with vaginal blood led us to suspect a ruptured ectopic pregnancy. The patient was obviously bled out and extremely pale. While the patient was undergoing cardiopulmonary resuscitation, an DPL by the author confirmed the presence of a large haemoperitoneum. Insertion of a peritoneal lavage catheter, allowed the collection of four units of blood which were immediately used to replenish blood volume. Successful resuscitation with cardiovascular stability was followed by an emergency laparotomy where a tubal ectopic pregnancy was discovered and a right tubal resection performed. Four units of banked blood were transfused intraoperatively.

She had a haemoglobin of 2,2g% at admission, and a postoperative HB of 11g%. A transient period of disorientation was evident postoperatively which lasted for less than 24 hours.

The patient was discharged completely well within a week of admission. The emergency pre-operative use of autotransfusion undoubtedly contributed to a successful outcome. An admission platelet count of 100 000/mm3 dropped to 86 000/mm3 by the next morning and had returned to normal levels within 36 hours of admission. No clinical coagulation defects were noted, although a mild coagulopathy was present as shown by the haematological tests.

11.CONCLUSION

Many findings arising from this study parallel trends seen in other trauma centres with regards to sex ratio, age distribution, and types of pathology.

There were 77 patients in this study. Of these 77 patients, 65 were involved in penetrating or blunt injuries, and 12 were ruptured ectopic pregnancies.

There were 48 males and 17 females. The trauma group comprised 48 males and 5 females. The ectopic group obviously consisted of 12 females.

The ages ranged from 16 years to 65 years, with an average age of 29.91 years. The 26-30 year group comprised the largest number of patients.

11.1. COST

It is obvious from the previous chapter that autotransfusion, where possible, using non cell-washing techniques, is highly cost effective. The money saved by the administration of even one unit of blood more than covers the cost of the apparatus disposables.

11.2. TRAUMA EPIDEMIC

South Africa is experiencing an unprecedented wave of civil violence. Much stems from disadvantaged social backgrounds, however, the political changes occurring in this country are responsible for a major proportion of violence and unrest.

Associated with the political events appears to be an inability, or unwillingness on the part of security forces to control even lesser causes of injury and death, for example, road traffic behavior.

The violence is not only responsible for much unnecessary misery, but the costs involved in the treatment of victims, and the loss of working hours from injury, death, and unrest, threatens to harm, if not destroy the economy of this country.

The violence of the 1990's has also altered the age and sex distributions of trauma. Where this study reflects the pre-violence findings, trauma centres now see increasing numbers of females and children, resulting from indiscriminate violence.

11.3. COMPLICATIONS

No septic complications occurred in this trial, indicating that sepsis in patients with autotransfused blood is no higher than in patients having been transfused with banked blood.

No allergic reactions occurred in any group in this study.

The two deaths that occurred in the patients studied cannot be attributed to the use of autotransfusion, but mainly as a consequenc of the severe trauma sustained. There were few complications within this study, certainly none can be blamed entirely on the use of autotransfusion except the cases of haematuria which occurred in the autotransfused groups. These were probably as a result of the salvage techniques.

In the use of autotransfusion, disease substitution occurs. In return for an asymptomatic coagulopathy, one avoids the transmission of potentially serious diseases eg AIDS, hepatitis etc.

11.4. HAEMATOLOGY

Remarkably few clinical effects were noted from the use of autotransfusion, although the deviation from normal haematological values are startling. This once again confirms the adage that one must treat the patient and not the blood results. To routinely replace blood components during autotransfusions would escalate the cost of patient management and subject the patients to an added risk of transfusion reactions and disease transmission.

A notable and common finding in the study is the degree of leucocytosis in the trauma patients, and the surprising absence of leucocytosis in the autotransfused ectopic patients. This is worth further research.

11.5. APPARATUS

The use of autotransfusion does not require sophisticated and expensive equipment as this study indicates.

This is particularly important in the South African context where financial constraints are concerned. Furthermore, the frequent lack of blood supplies on hospital premises, and not infrequent shortages of blood in blood banks, makes this technique very attractive, both in the trauma situation and in the elective situation. Where economy is concerned, autotransfusion is obviously cost effective.

11.6. ANTICOAGULANT

The use of heparin as an anticoagulant may, in the light of the above results, not be required in the case of an existing haemothorax or haemoperitoneum.

In the case of ongoing bleeding and blood salvage, anticoagulation is certainly required, as in the case of the patient with a 21 unit autotransfusion. In these cases, blood is not in contact with mesothelial surfaces for a sufficient time to anticoagulate the blood naturally.

The use of heparin in the above patient led to a bleeding state which was easily reversed with intravenous protamine.

This study cannot demonstrate whether heparin is better or worse that citrate, although other individuals attempting autotransfusion in other SA centres, using citrate, had no success in this technique. (personal admission from several delegates at the Medunsa SRS meeting in 1985).

The advantage of heparin is reversibility, minimal volume, and a possible anti-DIC effect. In view of recent publications, heparin may also have a protective effect on the microvasculature in shock states. In the case of citrate anticoagulation, the added volume with reduced haematocrits is a disadvantage in situations with large autotransfused volumes.

It was obvious that during the course of this study, that the technique of blood salvage and autotransfusion should be a unit

policy, and not merely the pet subject of one individual as was the case in this study.

11.7. CONCLUSION

The use of autotransfusion is of significant benefit to patients and should become standard practice, particularly in the acute surgical units of all major institutions in this country.

The blood that was salvaged from body cavities often had macroscopic fat globules. No effects were noted in any patient.

We found there to have been no difference between stabs, blunt chest trauma, and Gunshot wounds.

Autotransfusion Indications:

Body cavity blood less than 12 hours old; this includes ruptured ectopic pregnancies.

The contraindications were as follows:

>12 hrs after onset,
Left lower chest penetration,
High velocity wounds,
Pre-existing DIC.

The results of this study confirm the question posed in the study, in that autotransfusion is safe, efficacious, and cost effective.

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