ANAESTHETIC COMPLICATIONS OF LIMB SALVAGE SURGERIES

A 4 year retrospective study

by

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

ACD	Acid Citrate Dextrose
ASA	American Society of Anesthetist
CC	Closing Capacity
CO2	Carbon Dioxide
COAD	Chronic Obstructive Airway Disease
CRE	Critical Respiratory Event
CT	Computed Tomography scan
DIVC	Disseminated Intravascular Coagulation
EACA	Epsilon Amino Caproic Acid
FFP	Fresh Frozen Plasma
FiO2	Fractional inspired oxygen concentration

FRC	Functional Residual Capacity
HLA-DR	Human Leukocyte Antigen
IL-2	Interleukin - 2
MRI	Magnetic Resonance Imaging
O2	Oxygen
ODC	Oxygen Dissociation Curve
OORU	Orthopaedic Oncologic Reconstructive Unit
PaCO2	Partial pressure of arterial carbon dioxide
PaO2	Partial pressure of arterial oxygen
REM	Rapid Eye Movement
SpO2	Arterial oxygen saturation

ABSTRAK

Objektif :

Penyelidikan ini bertujuan mengkaji komplikasi berkaitan bius yang berlaku dalam pembedahan menyelamatkan anggota dan juga setakat mana sumbangan keadaan am pesakit dan tempoh pembedahan terhadap kejadian komplikasi tersebut.Kami juga bertujuan mengkaji takat dan mutu penjagaan pesakit setelah pembedahan tersebut dilakukan.

Tatacara :

Rekod perubatan 120 pesakit yang telah menjalani pembedahan menyelamatkan anggota dikenalpasti dan dikaji untuk kejadian komplikasi dalam 4 kategori : hematology, suhu, kardiovaskular dan pulmonary. Maklumat berkenaan diisi dalam borang pungutan data.

Keputusan :

Tiada maklumat rumusan didapati dari data demografi. Penurunan suhu badan semasa pembedahan merupakan komplikasi bius tunggal yang ketara. Kami dapat membuktikan hubungan yang ketara diantara tempoh pembedahan/pembiusan dengan kejadian penurunan suhu badan .Hampir setengah pesakit diberi bantuan penafasan ventilasi yang dilakukan di unit rawatan rapi.

Kesimpulan :

Penurunan suhu badan semasa pembedahan terbukti merupakan komplikasi bius semasa pembedahan menyelamatkan anggota. Kejadian penurunan suhu badan mempunyai hubungan rapat dengan tempoh pembedahan dan pembiusan. Bantuan penafasan ventilasi di unit rawatan rapi adalah rawatan selepas pembedahan yang paling kerap dilakukan.

ABSTRACT

Objectives:

Our purpose was to study the incidence of perioperative anaesthetic complications and morbidity following limb salvage surgeries as well as to determine the extent of contribution of preoperative status and duration of surgery towards incidences of these perioperative complications. Apart from that we aimed to study the extent and quality of postoperative care in limb salvage surgeries

Study Design :

The medical records of 120 patients who underwent limb salvage surgery were traced and studied for incidences of perioperative anaesthetic complications in 4 categories : hematological, temperature, cardiovascular and pulmonary. Necessary information were tabulated into individual data collection sheets (Appendix A) .

Results :

There were no significant findings in the demographic data. Intraoperative hypothermia appeared to be the only significant perioperative anaesthetic complication. We were able to demonstrate a significant relationship between the duration of surgery / anaesthesia and the development of intraoperative hypothermia. Almost half the patients were ventilated postoperatively whereby majority of care was instituted in the intensive care unit.

Conclusion :

Intraoperative hypothermia is an proven perioperative anaesthetic complication of limb salvage surgery. Incidences of hypothermia in limb salvage surgery are directly proportional to the duration of surgery and anaesthesia. Ventilation in the intensive care unit appears to be the most common postoperative care.

1.0 INTRODUCTION

The prognosis of patients with musculoskeletal tumors has improved markedly in recent years because of the advent of new chemotherapeutic drugs and regimes, advancements in imaging methods (computed tomography [CT scan] and magnetic resonance imaging [MRI]) and finally improvements in surgical technique, both resection and reconstruction.

Limb-salvage surgeries can currently be performed with better outcomes, while in the past, limbs with tumors were treated with only amputation. Today, it is the exception that a patient loses a limb as part of cancer treatment.

The conduct of limb salvage is such that it is performed in three stages: removal of the tumour and a margin of healthy tissue, implant of the prosthesis or bone graft (when necessary), and closure of the wound by transferring soft tissue and muscle from other parts of the patient's body to the surgical site.

It is therefore without doubt, limb salvage surgery is complexed and more often than not prolonged in duration. It is a general understanding that the more complexed the surgical procedure, the higher is the morbidity .Consequently, it is also a long held belief that prolonged surgery carries a higher risk than expected complication rate.

With this in mind, this retrospective study was carried out in an attempt to describe the perioperative complications associated with limb salvage surgeries. Since it is a pioneer

study of this nature in this region, six commonly encountered complications of interest to the anaesthetist were assessed, namely haematological, hypothermia, cardiovascular, pulmonary and deep vein thrombosis.

Following the establishment of the Orthopaedic Oncologic Reconstructive Unit (OORU) in Hospital Universiti Sains Malaysia (HUSM) in 1997, limb salvage surgeries are a routine event where in an average 1 limb salvage surgery is performed in a week. It is the hope of the investigators that, the results of this study would contribute to further understanding and management of anaesthesia for such a surgery.

2.0 LITERATURE REVIEW

2.1 ANAESTHESIA and LIMB SALVAGE SURGERY

2.1.1. Terms and definitions

Limb salvage surgery is defined as a non ablative limb sparing surgery involving musculoskeletal tumour of the extremities (Dee, 1998). In other words, it is not only resection but also reconstruction of the affected limb which is a far cry from what the orthopaedic oncologic treatment of yesteryears could offer i.e. amputation. The two principle aims are to achieve local clearance of tumour, equal or superior to the margin of amputation and retainment of involved limb function. Techniques of bony reconstruction include resection-arthrodesis, vascularised autograft, massive allograft, prosthetic-allograft composite, custom endoprosthetic replacement and modular endoprosthetic replacement (Simon, 1996). Soft tissue defects may require either local or distant flaps of which the latter will involve microvascular surgery.

2.1.2. The problems in limb salvage surgery

Increased knowledge and improved surgical techniques cannot overcome the basic surgical problems of limb salvage surgery namely haemorrhage and ischaemia. Resection of a limb involves transection through vascular bundles and richly vascular skeletal muscle which may lead to blood losses of varying degrees. The administration of limb

tourniquets in allowable situations serves to alleviate these problems but the complications and sequelae of haemorrhage still remain.

The reconstruction aspects of limb salvage surgery most often involve microvascular surgery. In this aspect hypoperfusion and subsequent necrosis poses a major challenge (Furnas, 1991). Like other surgeries, reconstructive microvascular surgery is an injury that activates pro-coagulatory processes, favours sequestration of platelets and other formed blood particles, setting the stage for stagnation of blood flow (Banic, 1995). Ischaemic necrosis of distal segment of the flap follows primary and then secondary ischaemia (Kerrigan, 1983). Primary ischaemia occurs between the time the original vascular supply of the flap has been cut off until arterial and venous anastomoses have been established at the recipient site. This phenomenon renders the flap more sensitive to secondary ischaemia which is due to delayed phase of hypoperfusion that commences some time after the re-establishment of vascular supply.

2.1.3. Anaesthesia for Limb Salvage Surgery

The lack of studies on anaesthetic aspects of limb salvage surgery probably reflects the general attitude of anaesthetists towards it. Limb salvage surgery may be regarded as straightforward as most patients are young and otherwise healthy with surgery seldom involving vital organs. However poor anaesthetic management would result in poor surgical outcome particularly if the surgical factors themselves are less than optimal.

Anaesthetic management directly influences the viability of the salvaged limb mostly through the changes in central haemodynamics and regional blood flow (Sigurdsson, 1995). These are brought about in many general ways in addition to the specific agents used for anaesthesia itself.

1. The positioning and pre-surgical preparation of the patient on the operating table is time consuming, during which the patient may lose considerable amounts of heat from vasodilation and poor insulation from cold operating theatre environment. Large areas of exposed body surface are washed with cold antiseptic solutions which are then allowed to evaporate.

2. Blood and fluid loss can be extensive due to large areas of dissection and prolonged surgery. Thus hypovolaemic peripheral vasoconstriction is added with hypothermia to further threaten the survival of the salvaged limb (MacDonald, 1985).

3. Limb salvage surgery is often prolonged. Durations of 8-12 hours or longer are not unusual (Cochrane, 1987). Instituting prophylaxis against thromboembolism is justified even in young and fit adults either by giving low molecular weight heparin or dextran (Bergqvist, 1992).

2.2 PATHOPHYSIOLOGY OF PERIOPERATIVE COMPLICATIONS AND MORBIDITY

All patients undergoing an operative surgery will undergo surgical *stress response* which involves changes in organ function believed to be mediated trauma induced endocrine metabolic changes and activation of several biological cascade systems (cytokines, complement, arachidonic acid metabolites, nitric oxide, free oxygen radicals etc). These responses although confer an advantage for survival, may also cause erosion of body cell mass and physiological reserve capacity if amplified and prolonged.

2.2.1. Perioperative risk factors

2.2.1.a. Preoperative Factors:

1. Pre-existing disease.

Concomitant disease and organ dysfunction are determining factors of post operative complication rates and duration of hospital stay.

2. Malnutrition

Malnutrition is a well established perioperative risk factor. Adequate nutrition is required for normal cellular and therefore normal organ function. The initial surgical stress response of catabolism which normally well tolerated in adequately nourished individuals

will definitely not occur in malnourished patients. This will not only affect intraoperative events but postoperative recovery as well.

3. Alcohol abuse

Preoperative alcohol abuse even without alcohol related organ dysfunction is a significant risk factor. The mechanisms of action include alcohol induced immunosuppression, subclinical cardiac dysfunction and amplified hormonal response to surgery.

4. Preoperative chemotherapy / radiotherapy

Chemotherapy and to a lesser extent radiotherapy or both are an integral part of the preoperative treatment in many patients planned for limb salvage surgery. Consideration of their side effects is mandatory as some patients will be suffering from acute systemic upset related to these agents preoperatively while some may even be suffering from long term toxicity

Side Effects of Oncology

Haematological

Anaemia

Leucopaenia

Thrombocytopaenia

Hypercoagulability

Cardiac

Dysrhythmias

Cardiomyopathy

Pericarditis

Respiratory fibrosis

Nephrotoxicity

Hepatotoxicity

Neurotoxicity

Poor nutritional state

Poor venous access

2.2.1.b. Intraoperative Factors:

1. Surgical stress response

During and after surgical injury, the body responds with profound changes in neural, endocrine and metabolic systems in addition to alterations in organ functions. These changes are physiological adaptations designed to return the patient to normal function (Bone, 1992). The pattern reflects the adequacy of host defense system and arises out of the interorgan fuel metabolism and immunologic response mechanism. Haemodynamically, heart rate, contractility, cardiac index and work are increased due to autonomic system activation with augmentation of oxygen consumption. Metabolically in

the first few days of surgical 'trauma', the normal balance between catabolic and anabolic processes is altered with the predominance of catabolic metabolism. This is characterized by increased secretion of catabolic hormones: noradrenaline, adrenaline, glucagons and cortisol with decreased effects of anabolic hormones resulting in hypermetabolism.

Glucose turnover increases by at least 2 fold. The rate of glucose production can be matched by an increased rate of utilization that euglycaemia is maintained. Besides these, there is impaired pulmonary function, pain, gastrointestinal side effects with nausea and ileus, hypercoagulopathy and thrombosis, loss of muscle tissue and immunosuppression. Within days, this sympathetic stress response settles with restoration of lean muscle mass. This pattern of response would be the normal reaction to post traumatic stress. The failure to achieve this state or to deviate from the normal response indicates an abnormal response in a post surgical patient (Miller, 2004).

Surgical stress response, although a cellular defense mechanism may also cause stress induced changes on postoperative organ function leading to postoperative complications. In 'clean' elective surgeries, the main mechanism of stress response is the afferent neural stimuli from the surgical area. In addition, humoral substances such as cytokines, arachidonic acid cascade metabolites, nitric oxide, endotoxins and other biological cascade systems are involved. The magnitude of surgical stress response is related to extent of surgical injury whereby lower morbidity rates are observed in minor and minimally invasive surgery (Vishvanathan, 2000).

2. Hypothermia

Intraoperative heat loss is a significant risk factor leading to increased stress response, cardiovascular, metabolic and hematological complications. Prevention of hypothermia by conservation of body heat should be done to reduce the stress responses to rewarming i.e. increased oxygen consumption, catabolic hormone secretion and nitrogen loss.

3. Blood Transfusion

Massive blood loss and intraoperative blood transfusions correlate with increased risk of perioperative infective complications. The risk is due to the content of white cells and non cellular transfusion components within the transfused blood products. Even autologous blood transfusion with prolonged storage time has detrimental effects as toxic mediators (histamine, PAI-1, myeloperoxidase etc) are released from leucocytes and platelets during storage of blood products for more than 2 weeks

2.2.1.c Postoperative factors

1. Pain

All surgical procedures result in pain which then amplify endocrine metabolic responses, autonomic reflexes, nausea, ileus and muscle spasm thereby delaying restoration of normal function. Optimal treatment of postoperative pain is mandatory in enhancing recovery and reducing morbidity.

2. Immunosuppression

Major surgery causes immunosuppression by several mechanisms: reduced delayed hypersensitivity response, T cell dependant antibody response, IL-2 production, HLA-DR antigen expression and T- cell blastogenesis. In contrast neutrophil and macrophage functions are activated with increased release of oxygen radicals and TNF as well as chemotaxis. The clinical consequences of pre and post operative immunological alterations are increased susceptibility to infective complications (Wheatley, 1997) and even increased risk of recurrence after oncologic surgeries. The most effective technique to reduce immunosuppression and risk of infection appears to be reduction in the extent of trauma by performing minimally invasive surgery (Vishvanathan, 2000).

3. Postoperative hypoxaemia

The mechanism of constant intraoperative hypoxaemia is primarily an intrapulmonary shunt caused by reduction in functional residual capacity. Postoperative episodic hypoxaemia may be due to ventilatory arrhythmias (hypoventilation and apnoeas) related to rebound rapid eye movement (REM) sleep on the second and third nights after operation. Late postoperative hypoxaemia may be involved in cardiac, cerebral and wound complications. Postoperative impairment of cognitive function and delirium may be a result of postoperative hypoxaemia as well.

4. Immobilization

Traditional perioperative care is said to be bed rest although immobilization is an all too well known risk for thromboembolic and pulmonary complications. Bed rest predisposes to orthostatic intolerances well as increased loss of muscle bulk and function leading to instability during standing. Postoperative hypoxaemia is more pronounced in the supine position with potential detrimental effects on cardiac, cerebral and wound function. In contrast, early ambulation may contribute to improved wound healing.

5. Catabolism and muscle wasting

Post operative catabolism and muscle wasting lead to postoperative fatigue thereby affecting the process of recovery. Catabolism is mediated by surgical stress response, postoperative immobilization and semi starvation. These 'destructive' albeit

physiological changes may persist for up to several months in patients who undergo major surgery.

6. Postoperative morbid sequelae

Surgical procedures may be followed by undesirable sequelae namely cardiac, pulmonary, thromboembolic and infective complications among a few. These may not be related directly to surgical nor anaesthetic techniques, rather to perioperative risk factors and pathophysiological responses.

2.3 Intraoperative Complications

2.3.1 Haematological Complications

Intraoperative and postoperative blood transfusions are given to increase oxygen carrying capacity and intravascular volume. Logically, increasing oxygen carrying capacity is the only real indication for blood transfusions as volume can be resuscitated with crystalloids or colloids.

Controversy remains on how much emphasis to be given on hemoglobin and haematocrit values. These values are limited to extreme variability from one patient to the other. A young, healthy, cardiorespiratory fit patient can easily compensate for anaemia

of acute or chronic haemorrhage. On the other hand, an elderly patient with cardiac problems having similar hemoglobin and haematocrit values may face serious problems perioperatively. Hence many medical organizations recommend emphasis on overall clinical judgment than on specific laboratory value (ASA Task Force, 1996) (Royal College of Physicians Edinburgh, 1994).

Healthy patients with a haematocrit value greater than 30 % rarely require perioperative blood transfusion, whereas patients with acute anaemia (e.g intraoperative blood loss) and haematocrit of less than 21% most often do. Patients with chronic anaemia (e.g. renal failure) may tolerate haemoglobin concentration of less than 7 g/dL. The final determination as to the haemoglobin or haematocrit value at which blood should be given is the clinical judgment based on factors such as cardiovascular status, age, anticipated additional blood loss, arterial oxygenation, mixed venous oxygen tension, cardiac output and blood volume (National Institutes of Health Consensus Development Conference Statement,1988).

Massive haemorrhage in elective surgery can be either anticipated (e.g. organ transplantation) or unexpected. Management requires early recognition, securing haemostasis and maintenance of normovolaemia. Transfusion management involves the transfusion of packed red cells, platelet concentrates and plasma (fresh frozen plasma and cryoprecipitate). Blood product support should be based on clinical judgment and be guided by repeated laboratory tests of coagulation. Although coagulation tests may not

provide a true representation of in vivo haemostasis, they do assist in management of haemostatic factors. When below critical levels (prothrombin time or activated partial thromboplastin time >1.8; fibrinogen <1.0 g/l; platelet count < 80,000/ml) it is difficult to achieve haemostasis. Despite seemingly adequate blood component therapy there remain situations where haemorrhage is uncontrollable. In this setting, alternative approaches must be considered. These include the use of other blood products (e.g. prothrombin complex concentrates; fresh whole blood; fibrin glue) and pharmacological agents like aprotinin and tranexamic acid (Erber, 2002). Although there are no trials on the use of activated Factor VIIa in this type of surgeries, consideration could be given to the possibility of the role of this drug in the reduction of blood loss.

Complications of massive transfusion result in significant morbidity and mortality. These may be secondary to the storage lesion of the transfused blood products, disseminated intravascular coagulation, hypothermia or hypovolaemic shock. The use of fresh blood products and leucocyte-reduced packed red cells and platelets, may minimise some of the adverse clinical sequelae.

The need for perioperative blood transfusion should be well justified and criteria for transfusion clearly stated so that it outweighs the many complications of blood transfusion as stated below.

Complications of Blood Transfusion

1. Changes in Oxygen Transport
2. Coagulopathies

- Dilutional thrombocytopaenia
- Low Factors V and VIII
- Disseminated Intravascular Coagulation
- Haemolytic transfusion reactions

3. Citrate intoxication and hyperkalaemia
4. Temperature alterations
5. Acid-Base abnormalities
6. Infusion of microaggregates

7. Transfusion reactions
   - Haemolytic transfusion reactions
   - Immune extravascular reaction (Delayed haemolytic transfusion reaction)
   - Nonhaemolytic transfusion reactions
8. Infectivity of Blood
   - Hepatitis
   - Acquired Immunodefiency Syndrome
   - Cytomegalovirus
9. Transfusion-Associated-Graft-Versus-Host Disease
10. Transfusion- Related Acute Lung Injury
11. Transfusion- Induced Immunodepression

Although complications of blood transfusions are many, the knowledge of those causing perioperative complications, particularly of intraoperative and immediate postoperative is of special interest in this review.

Changes in Oxygen Transport

Increased circulating red blood cell mass from increased red cell transfusion produces an increased oxygen uptake in the lungs and therefore increased oxygen delivery to tissues. The respiratory function of red cells may be impaired during preservation, making it difficult for them to release oxygen to the tissues immediately after transfusion.

Following transfusion of 7-day or older acid citrate dextrose blood (ACD), oxygen dissociation curves (ODC) of all patients shift to the left. The magnitude of the left shift is related to the volume and storage time of the infused ACD blood. The shift of the curve may remain to the left for as long as 24 hours after transfusion.

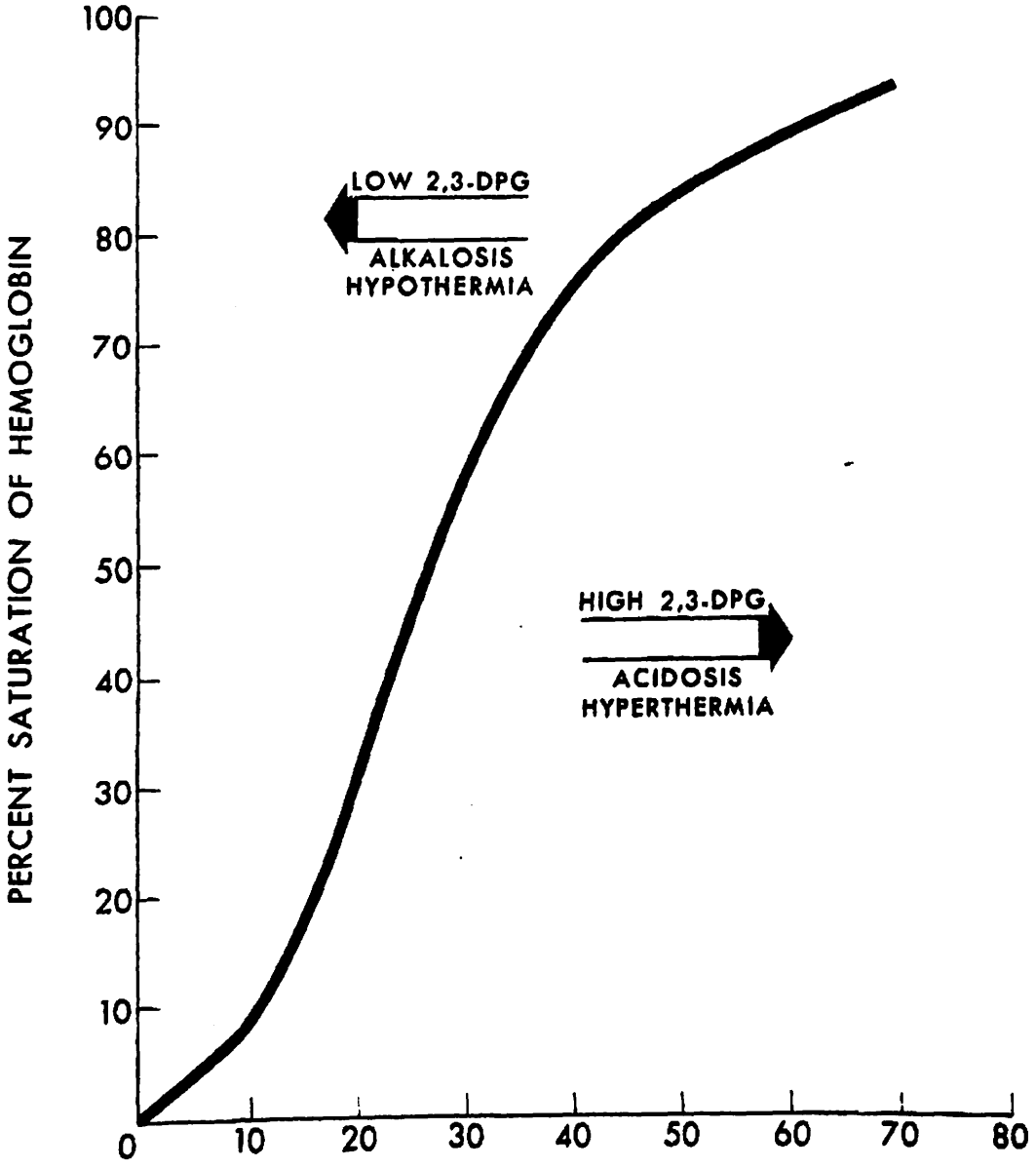

Figure 2.1 Factors that shift the oxygen dissociation curve

Theoretically, the left shift and increased affinity for oxygen may increase cardiac output and work of the heart (Valeri, 1979). In a patient with marginal cardiac reserve and inability to increase cardiac output, tissue hypoxia may occur. However, another study showed that although oxyhaemoglobin affinity increases after transfusion of stored blood, arterio-venous oxygen extraction by organs or tissue may not be altered by changes in oxyhaemoglobin affinity, particularly if a compensatory flow mechanism takes place at the capillary level. Such mechanisms may open capillaries permitting increased blood flow to tissues, thereby increasing cardiac output and reducing the capillary tissue oxygen gradient to maintain the rate of tissue oxygen extraction (Bowen, 1974)

Coagulopathy

Bleeding tendencies in massively transfused patients are due to a combination of factors most importantly being volume of blood given followed by duration of hypotension or hypoperfusion (Collins, 1987).

Dilutional thrombocytopaenia

Platelet viability and activity is known to reduce as storage time progresses. Storage at a temperature of 4°C causes platelets to be so damaged that they are fit to be sequestered by the reticuloendothelial system as soon as transfused. Platelets retain only 50-70 % of in vivo activity after 6 hours of storage in blood bank at 4°C. Activity further reduces to 5-10 % after 24-48 hours of storage. Infusion of such platelets only dilutes the available platelet pool.

Low factors V and VIII

Most factors are stable in stored blood with the exception of Factors V and VIII. These factors gradually decrease to 15 and 50% of normal respectively after 21 days of storage. Transfusion of packed cells exacerbates this condition as it contains fewer coagulation factors. Administration of fresh frozen plasma (FFP) containing all factors except platelets should be done with caution as only 5-20 % factor V and 30% factor VIII are needed for adequate haemostasis during surgery.

Disseminated Intravascular Coagulation

The coagulation system consists of clotting mechanism which prevents excessive blood loss and fibrinolytic mechanism which ensures circulation within the vasculature. In DIVC, the clotting system is deranged leading to disseminated fibrin deposition rendering blood unclottable. Deposited fibrin also affects microcirculation leading to ischaemic necrosis of various organs namely the kidney.

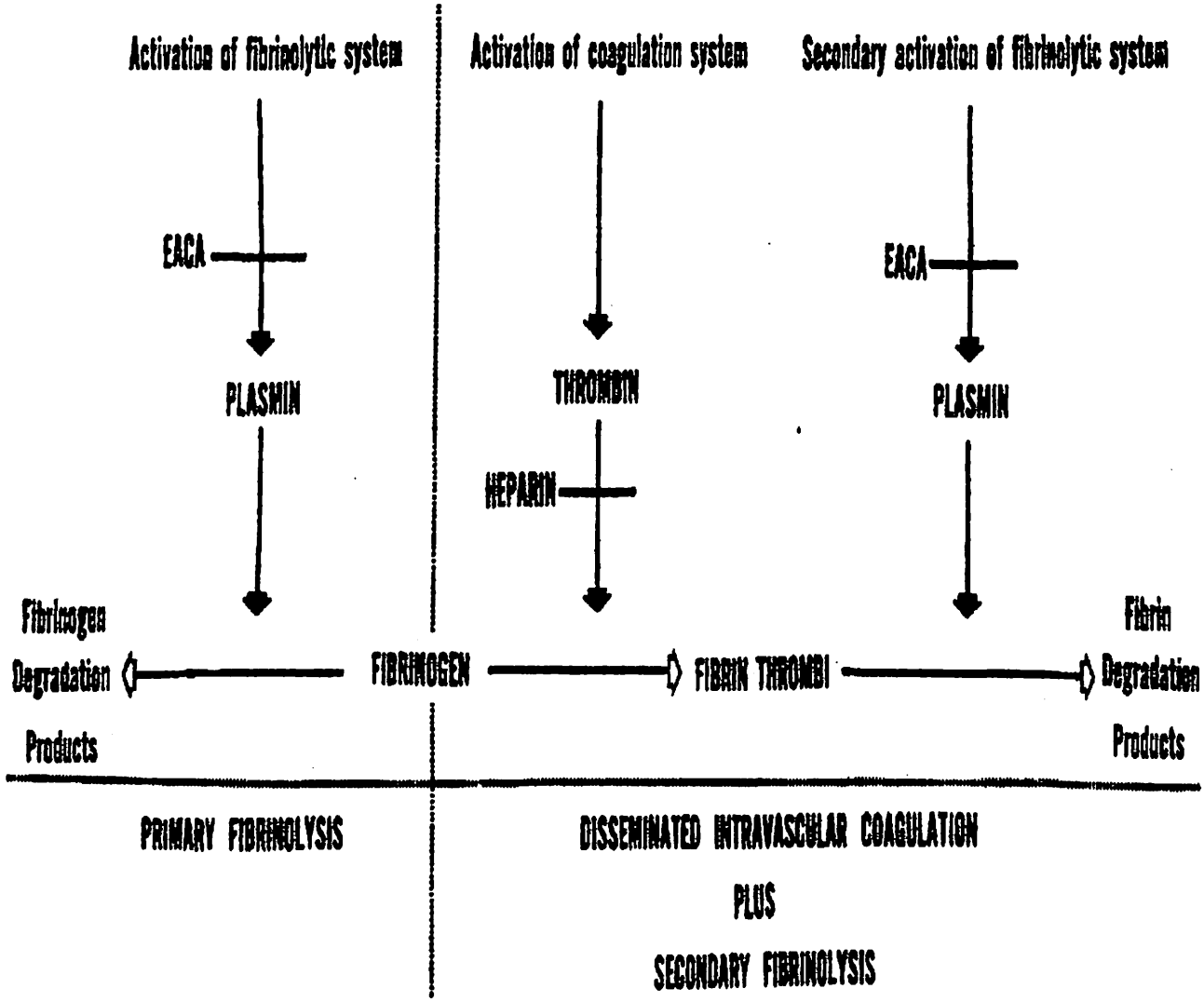

Figure 2.2 Schematic representation of primary fibrinolysis and fibrinolysis secondary to DIVC

Hypoxic, acidotic tissues with stagnant blood flow release tissue thromboplastin either directly or via liberation of toxins. In sepsis with eventual organ failure, the extrinsic coagulation pathway is activated by endotoxins and tumour necrotising factor. Although the intrinsic coagulation pathway does not induce DIVC, it contributes to hypotension. Triggering of the coagulation pathway results in the consumption of factors I, II, V, VIII and platelets. In an attempt to counteract the hypercoagulable state, the fibrinolytic system is activated to lyse the excessive fibrin; secondary fibrinolysis. Plasmin is rapidly formed from plasminogen which in turn digests fibrin causing further reduction in fibrinogen level. While fibrinolysis is a protective mechanism actively trying to counteract DIVC, plasminogen activator activity and plasmin generation rapidly decline leaving DIVC to progress unopposed (Levi, 1993). At this stage, severe morbidity and eventual mortality are likely to occur.

Harvey et al. (1995) in a retrospective study involving 43 patients who underwent massive blood transfusion found no significant correlation between the severity of coagulopathy or thrombocytopenia and total units transfused, or between the age of the units of blood and development of coagulopathy or microvascular bleeding. He concluded that severe coagulopathy is common after massive transfusions although there was absence of clear correlation with the number of units transfused. Duration of tissue hypoperfusion and platelet consumption are likely to be more important than simple haemodilution of coagulation factors.

Temperature alterations

Administration of inadequately warmed blood which is less than 30°C will decrease patient's temperature causing ventricular irritability and even cardiac arrest. Low temperatures of the operating theatre result in decreased body temperature of patients in particularly those undergoing extensive and prolonged surgeries. Administration of cold blood further decreases body temperature. A decline in body temperature as little as 0.5-1°C may induce shivering postoperatively, increasing oxygen consumption by 400%.

Acid- Base Abnormalities

The addition of acidic storage media (e.g citrate phosphate dextrose, pH 5.5) to freshly drawn blood immediately decreases pH to approximately 7.0-7.1. Accumulation of lactic and pyruvic acids by RBC metabolism and glycolysis further decreases pH to about 6.9 after 21 days of storage. Acidosis is also contributed by PCO_2 of 150-220 mmHg. This high PCO_2 value in the stored blood is due to inability of CO_2 to escape when stored in the plastic blood container.

Metabolic acid-base response to blood transfusion is actually variable. Blood transfusion provides citrate in large quantities for endogenous generation of bicarbonate. This accounts for the significant incidence of metabolic acidosis after blood transfusions.

2.3.2 Intraoperative Hypothermia

Body temperature between 33-36°C is considered mild hypothermia whereas that below 33°C and 23°C, severe and profound hypothermia respectively. Patients undergoing surgical procedures under anaesthesia are at increased risk of hypothermia due to (1) age impaired thermoregulatory responses, (2) anaesthesia impaired thermoregulatory responses, (3) ambient factors.

Age impaired thermoregulatory responses include decreased perception of cold, decreased ability to prevent heat loss (blunted vasoconstrictor response), and diminished ability to increase heat production (reduced muscle mass). Microvascular atheroma and diabetic autonomic neuropathy could also contribute to inappropriate thermoregulatory vasoconstriction to cold.

Both general and regional anaesthesia disrupt normal thermoregulatory responses (Sessler, 1997). Dose-dependent suppression (2-4°C) of the hypothermic vasoconstriction threshold (about 37°C) occurs with volatile or opioid anaesthesia, with the greatest suppression occurring in the elderly. In regional anaesthesia, sympathetic blockade prevents vasoconstriction in affected dermatomes. Heat generation from muscle activity is reduced in proportion to segmental motor blockade. Spinal thermoregulatory centres may be depressed with central neuraxial anaesthesia or analgesia. Shivering is abolished with anaesthetic agents and neuromuscular relaxants. Warm response (vasodilatation,