PERIOPERATIVE BLOOD CONSERVATION

An Essay

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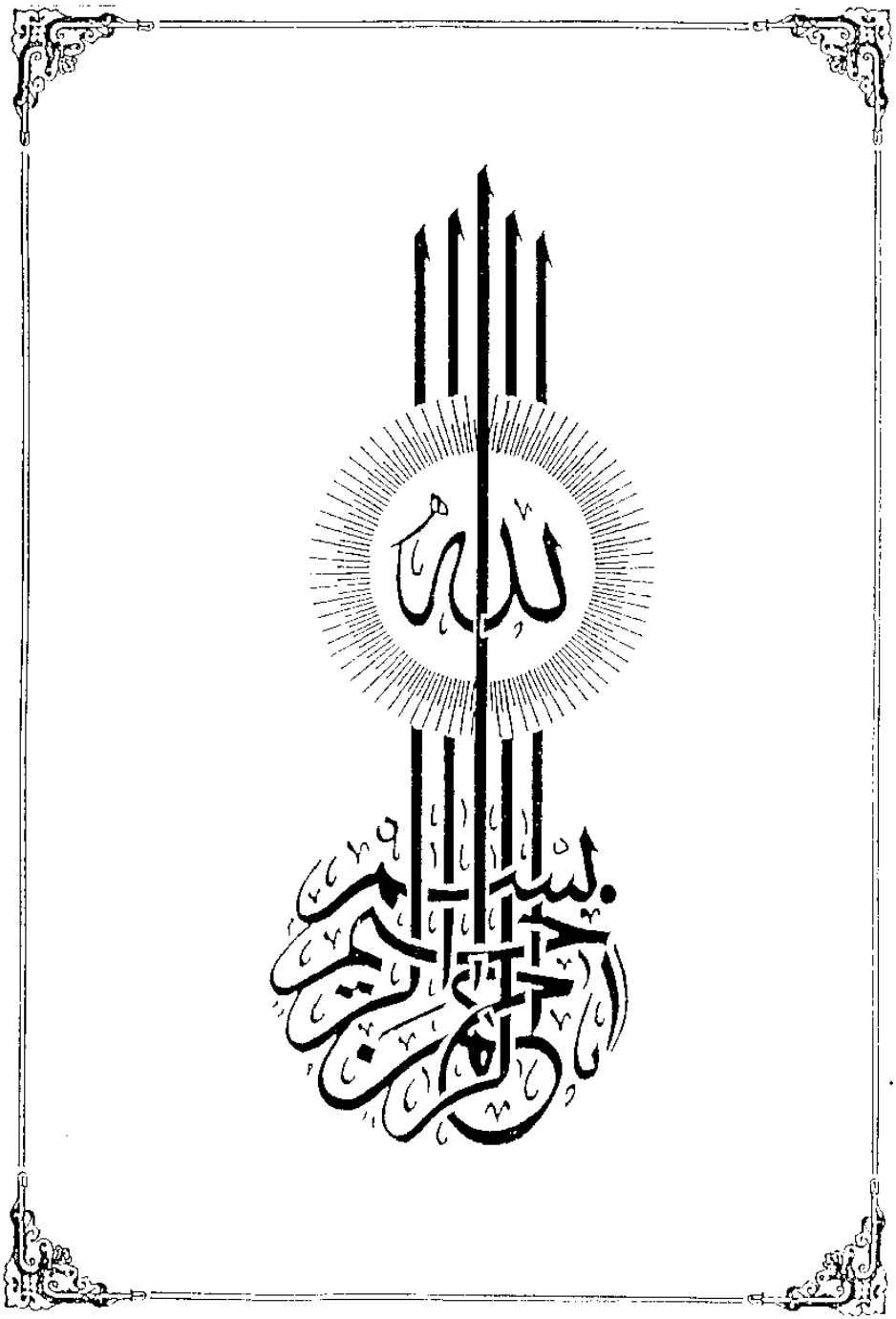

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INTRODUCTION

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With the increase in complexity of operations done nowadays, blood conservation during elective surgery will be of great benefit to the patients by reducing their exposure to the adverse effects of homologous blood transfusion, on top of which is transmission of diseases especially the acquired immune deficiency syndrome (AIDS), the horrible ghost in our universe.

Blood conservation during elective surgery will assist the chronic imbalance between the supply of homologous blood and its demand for elective surgery. The techniques are particularly important in patients who have religious objections to homologous blood transfusion and in patients with rare blood groups.

Blood conservation depends on close co-operation between anaesthesiologists, haematologists and surgeons. The anaesthesiologist can make a considerable contribution by using techniques that reduce the amount of blood loss during surgery or techniques to reduce the amount of homologous blood used to maintain normal blood volume.

The aim of this essay is to consider the hazards of liberal use of homologous blood during surgery and to consider the important aspects of special techniques, the preoperative

acute limited normovolaemic haemodilution and intraoperative autotransfusion as a new method for blood conservation during elective surgery.

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PATHOPHYSIOLOGY OF HAEMORRHAGIC SHOCK

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The modern understanding of shock dates from field studies carried out behind the lines in the First World War by workers such as Cannon, Bayliss and McLean. As a result of these studies, it became generally accepted that shock was caused by tissue hypoxia due to inadequate oxygen delivery as a result of failure of tissue perfusion, with the resultant lactic acidosis from anaerobic metabolism producing the characteristic compensatory hyperventilation. (Thal, et al., 1972)

In haemorrhagic shock, of course, both hypovolaemia and reduced blood oxygen carrying capacity contribute to the reduction in tissue oxygen delivery. It is now recognized that this inadequate tissue perfusion is accompanied very early by an abnormal cellular metabolism which contributes to the complex physiological and biochemical features of shock. (Wilson, 1980)

* HOMEOSTATIC RESPONSES IN HAEMORRHAGIC SHOCK *

The body responds to blood loss by several mechanisms, these include the following :-

Sympathetic activity :- This is mediated by baro and chemo-receptors, pain and anxiety and results in an increase in rate and force of myocardial contraction and widespread arteriolar and veno-constriction diverting available blood flow to heart and brain. (Heymans and Neil, 1958)

Circulating catecholamines :- There may be ten to forty fold increase in the concentration of circulating adrenaline and noradrenaline (up to 3 and 7 ng/ml respectively) in haemorrhagic shock. Noradrenaline represents spill-over from synaptic clefts, whereas adrenaline is released from the adrenal medulla. (Runciman, 1980)

Renin-Angiotensin :- In haemorrhagic shock there is an increase in renin secretion by the renal juxtaglomerular apparatus due to reduced glomerular blood flow, decreased sodium delivery to the macula densa of the distal tubules, reduced renal afferent arteriolar pressure and increased sympathetic activity. The resultant increased circulating angiotensin causes vasoconstriction and plays a role in maintaining the blood pressure and also causes aldosterone release. (Barnes and McDowell, 1982)

Anti-Diuretic hormone [ADH] :- ADH secretion by the posterior pituitary is normally mediated via osmoreceptors in the hypothalamus in response to increased body fluid

osmolality. In shock, this may be augmented by decreased discharge of arterial and atrial baroreceptors. ADH has been shown to cause splanchnic vasoconstriction and to permit distal renal tubular and collecting duct reabsorption of water. (Ganong, 1983)

Aldosterone :- There is increased release of aldosterone due to the increased release of renin, leading to increase in renal distal tubular reabsorption of sodium and water and excretion of potassium, which is released from damaged cells. (Ganong, 1983)

Adrenocorticotrophic hormone [ACTH] and Beta-Endorphins :- The endogenous opiate beta-endorphin is stored and concomitantly regulated with pituitary ACTH and both are released from sudden blood loss, presumably by activation of hypothalamic releasing factors. The ACTH causes increased release of glucocorticoids, whereas the endorphins may reduce pain perception and may play a role in perpetuating hypotension, as naloxone administration may reduce the fall in blood pressure. (Holaday and Faden, 1978)

Glucocorticoids :- Circulating glucocorticoid concentrations increase in haemorrhagic shock. These high concentrations of cortisol may be necessary for a normal homeostatic response in shock. (Shatney, 1982)

Glucagon :- Glucagon is secreted by alpha cells of the pancreatic islets. Its main site of action is the liver, where it stimulates gluconeogenesis. (Chaudry and Baue, 1982)

Erythropoeitin :- After significant blood loss there is an increase in circulating erythropoeitin, with an increase in reticulocytes in peripheral blood. Normal red blood cell volume is restored within 4-8 weeks. (Ganong, 1983)

2,3 Diphosphoglycerate :- After acute blood loss there is an increase in 2,3 diphosphoglycerate (2,3 DPG) in the red blood cells. It is a product of glycolysis via the Embden-Meyerhoff pathway, and is very plentiful in red blood cells. It is highly charged anion, which binds to deoxygenated haemoglobin, but not to oxyhaemoglobin; thus an increase in 2,3 DPG concentration increases liberation of oxygen. Acidosis inhibits red cell glycolysis leading to decreased 2,3 DPG, while hypoxia, exercise and shock cause 2,3 DPG concentrations to increase. (Runciman and Skowronski, 1984)

Prostaglandins :- The prostaglandins comprise several carboxylic acid compounds synthesized from essential fatty acids in cellular microsomes. PGE2 and PG12 are vasodilators locally produced by organs such as liver and kidney, and may

have some protective influence early in shock. PG12 is also a potent inhibitor of platelet aggregation; however its production by endothelial cells may fail in shock states. (George and Tinker, 1983)

Prostaglandins PGF2-alpha and PGA2 are both vasoconstrictors. PGF2-alpha concentrations rise in haemorrhagic shock and may contribute to both the pulmonary and systemic vasoconstrictions. (Rinaldo and Rogers, 1982)

PGA2 also causes platelet aggregation and may play a role in the production of disseminated intravascular coagulopathy DIC. (Chaudry and Baue, 1982)

Complement activation :- The complement system consists of a cascading series of plasma enzymes similar to the coagulation pathway. complement is involved in the cell lysis, leucocyte chemotaxis and histamine release which normally follows antigen-antibody binding. (Ganong, 1983)

Complement activation can also occur in the absence of antibodies, via the alternative or properdin pathway. In shock, excessive complement activation via this pathway results in disordered leucocytic function which is especially important in the production of adult respiratory distress syndrome ARDS. (Rinaldo, 1982)

The immediate effect of sudden blood loss is the activation of the above variety of homeostatic mechanisms. This is accompanied or very soon followed by the local release of wide variety of substances, many of which constitute appropriate responses to local injury, but which may cause deleterious effects when released in large quantities into the blood stream. If blood loss is substantial and remains untreated, this is followed by the release of the contents of hypoxic or dying cells, many of which cause both local and systemic damage.

* MEDIATORS IMPLICATED IN HAEMORRHAGIC SHOCK *

Lysozomal enzymes :- They are released from cells in the presence of hypoxia and acidosis, especially in the liver, kidney, spleen and pancreas and increased plasma concentrations are found in haemorrhagic shock. (George and Tinker, 1983). These enzymes cleave proteins, causing cellular damage, and also cause myocardial depression and coronary vasoconstriction. (Haglund and Lundgre, 1978)

Kinins :- They are normally formed by the action of proteolytic enzymes called kallikreins or kininogens. In shock states some are produced as a result of lysozomal enzyme action on alpha-2 globin. (Ganong, 1983)

They may play a major role in microcirculatory failure as they cause vasodilation, increase capillary permeability,

and precipitate disseminated intravascular coagulation. They are also myocardial depressants. (George and Tinker, 1983)

Histamine :- Histamine may normally play a role as a co-transmitter in the regulation of the microcirculation under hypoxic or ischaemic conditions. (Ganong, 1983; Shumer and Nyhus, 1974)

Histamine release from damaged cells causes increased capillary permeability. (Wilson, 1980)

Serotonin :- Serotonin released from platelets exerts a strong local vasoconstrictor effect on arteries and arterioles. (Ganong, 1983)

Fibronectin :- Fibronectin, an important adhesive glycoprotein organizer of tissue topography in the interstitium, is also an essential serum opsonin for the effective phagocytic function of the reticuloendothelial system. Fibronectin is depleted in patients with haemorrhagic shock, trauma, sepsis, and DIC, thus allowing particulate matter such as cellular debris and fibrinogen degradation products (FDPs) to circulate for prolonged periods in these states. (Rinaldo and Rogers, 1982)