HISTOLOGICAL STUDY ON THE EFFECT OF ACUTE HAEMORRHAGE ON SOME ENDOCRINE GLANDS OF ALBINO RAT

THESIS

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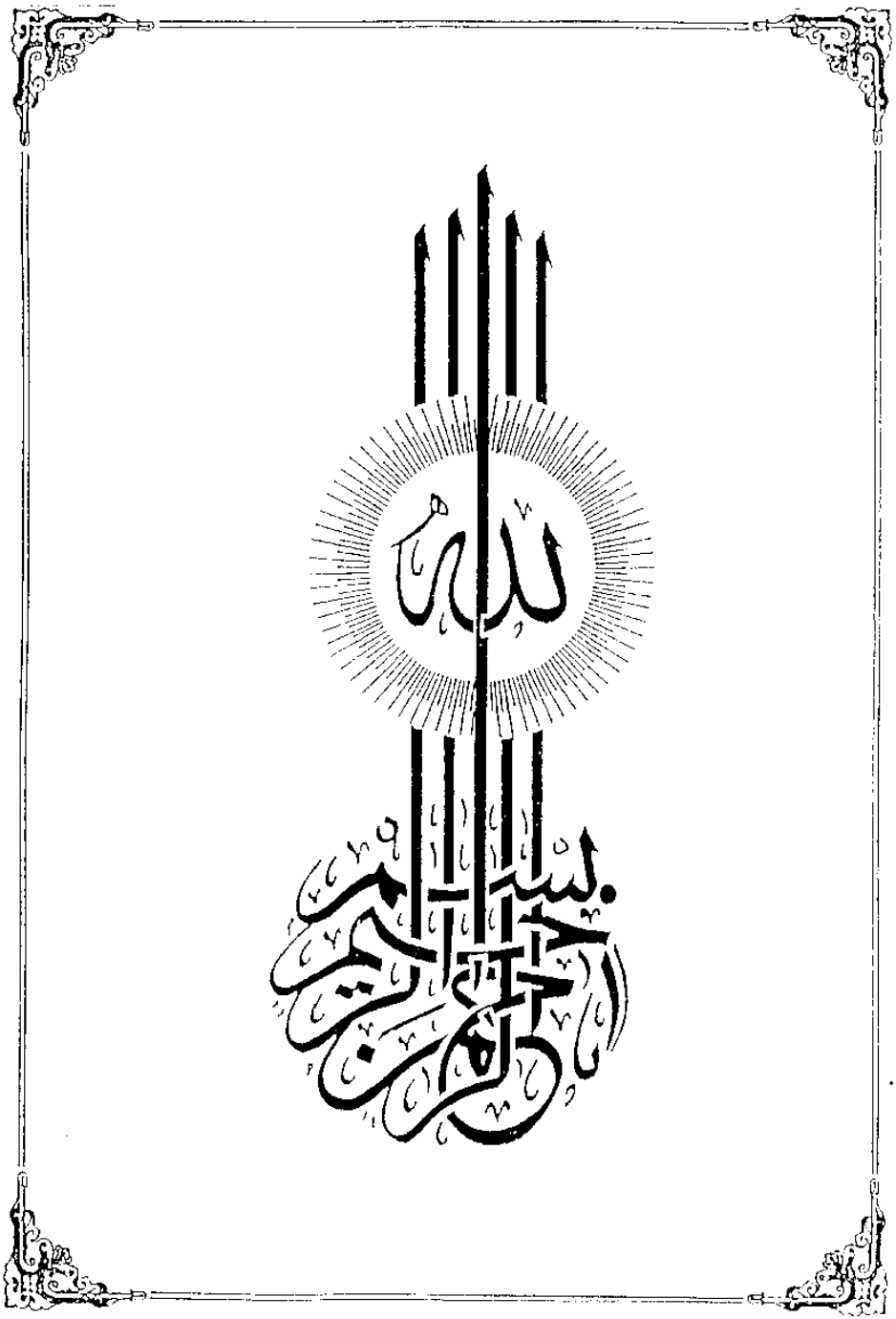

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INTRODUCTION
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Acute bleeding caused by accidents, bleeding oesophageal varices and post partum haemorrhage may be severe and results in a shock state which indicated acute circulatory failure *[Friedberg, 1969]*. In relation to haemorrhage, the endocrine system has been a subject of research work.

Reviewing the available literature, it was evident that special attention was paid by many authors to the physiology and hormonal responses of endocrine glands to acute haemorrhage. *Carey et al. (1970)* followed by *Hiebert et al. (1973)* noticed that haemorrhage was a powerful stimulus to increased glucagon release and decreased insulin release from the pancreas. *Moreover, Briand et al. (1989)* recorded that during haemorrhage pancreatic glucagon may be involved in the hyperglycaemic response. *Sheehan (1937)* observed anterior pituitary necrosis in delivaries complicated by severe post-partum haemorrhage. While *McCormick et al. (1966)* claimed that there was increase ingrowth hormone release in humans during haemorrhage. *Ryan (1979)* stated that varying degree of hypothyroidism occured secondary to severe post partum haemorrhage.

However, the histological changes of endocrine glands after haemorrhage did not recieve much attention, so the present work aims to study the effect of different grades of acute haemorrhage on the histological picture of islets of Langerhans, pituitary and thyroid glands.

REVIEW OF LITERATURE

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Simeone, (1963) defined the haemorrhagic shock as a clinical state caused by an absolute deficit of blood such that the circulating blood volume was insufficient to permit normal perfusion of the vascular beds. The subliminal perfusion of living tissues with blood caused a progressive metabolic acidosis which if not corrected, resulted in death.

Hamilton, (1965) recorded that haemorrhagic shock was a type of hypovolaemic shock caused by loss of one third or more of the total blood volume. The loss included plasma as well as blood cells.

Clinical pictures occured in hypovolaemic shock were observed by ***Ganong, (1981)***. He noticed the decrease in blood pressure, rapid thready pulse, cold pale skin, intense thirst and rapid respiration.

These clinical sings were found to be accompanied by changes in the tissues of some organs.

I. ISLETS OF LANGERHANS

Engel; Winton and Long (1943) observed that during haemorrhagic shock in rats, there was a progressive rise in the blood levels of aminonitrogen, ketoacids as pyruvate and lactate, while the blood sugar level fell, provided epinephrine hyperglycaemia was prevented either by previous removal of suprarenal medulla or by reduction of liver glycogen level by fasting.

Russell, Long and Engel (1944) compared the changes in the blood levels of amino nitrogen, glucose, lactate and pyruvate in liverless rats and liverless rats subjected to haemorrhage in order to establish the role of the peripheral tissue in the blood changes during shock. He found that blood sugar fell more rapidly in the liverless rats after haemorrhage, both in animals with suprarenal gland and those with enucleated suprarenal medullae.

The effect of ischaemia on the islets of Langerhans of albino rats was observed by ***Adams and Harrison, (1953)*** by ligation of the main blood supply of the pancreas. They noticed fibrosis of the necrotic zone, but reorganization occured within seven days. In these areas, there were only remnants of islet parenchyma showing vacuolation or degranulation of the alpha and beta cells, with no evidence of islet regeneration.

Allison, Hinton and Chamberlain (1968) suggested that the acute insulin secretory response to glucose was inhibited after shock despite persistantly elevated peripheral insulin concentration. They suggested that insulin resistance was present in addition to inhibition of insulin secretion.

Bauer, Vigas, Haist, and Drucker (1969) mentioned that when well-fed dogs subjected to haemorrhagic shock, there was a characteristic rise in blood glucose in early haemorrhagic shock associated with a significant rise in plasma insulin. As shock contineoued, the level of both insulin and glucose declined.

Carey, Lowery and Cloutier (1970) noticed that humans in hypovolaemic shock developed severe hyperglycaemia within minutes which was related to the severity of shock. Serum insulin in man was irresponsive to hyperglycaemia of shock and it did not resolve after resuscitation had been accomplished.

Hiebert, Celik, Soeldner and Egdahl (1973) recorded that haemorrhage was a powerful stimulus for decreased insulin release from pancreas, and that catecholamines from the adrenal medulla were the principal agents causing suppression of insulin secretion in haemorrhagic shock. So,

peripheral and portal blood insulin concentration did not reflect insulin secretion rates during haemorrhagic shock.

Lindsey, Faloona and Unger (1975) confirmed that pancreatic glucagon was considered as one of the stress hormones since its plasma concentration was markedly increased during different types of stress such as haemorrhage and haemorrhagic shock.

Andersson, Holst and Jarhult, (1979) recorded glucose, insulin and glucagon concentrations in the blood before, during and after haemorrhagic shock. They concluded that the prompt hyperglycaemic and hypoinsulinaemic response to haemorrhage in cats were caused by adrenergic, non medullary mechanism, whereas the marked rise in pancreatic glucagon release seemed to be due to factors unrelated to the sympathoadrenal system.

Seemayer, Osborne and Jean Pierre de Chadarevian, (1985) described a pancreatic lesion restricted to the islets of Langerhans in infants who died from hypovolaemic shock. The islets injury was manifested by nuclear pyknosis, cytoplasmic acidophilia and reduction in the cell size. Some cases showed coagulative necrosis of islet cells such that the islets were represented by aggregates of necrotic cellular remnants. Meanwhile, they noticed the absence, in

all cases, of cellular infiltrate, fibrin thrombi and evidence of antecedent haemorrhage.

Mcleod, Carlson and Gann, (1986) noticed that when dogs were haemorrhaged 10, 20, 30 percent of the estimated blood volume, peripheral glucose did not change after 10 percent haemorrhage, but increased after 20 and 30 percent haemorrhage from ten minutes to two hours. In contrast, the portal venous delivery of immunoreactive glucagon did not increase until at least 1 hour after any magnitude of haemorrhage. Portal venous delivery of immunoreactive insulin decreased from 20 minutes to 6 hours after 10 percent haemorrhage. The authors assumed that an increase in release of glucagon occured too late to account for the early hyperglycaemia. Likewise, the decrease in release of insulin, that occured after a 10 percent haemorrhage, cannot be accounted for the increase in glucose. However, these changes might contribute to the hyperglycaemia observed after 20 and 30 percent haemorrhage.

Recently, ***Briand, Gagne and Yamaguchi, (1989)*** studied the role of pancreatic glucagon in haemorrhage induced hyperglycaemia in dogs. They found that the increase in plasma glucagon was of pancreatic origin, and it might be involved in the hyperglycaemic response to haemorrhage through glucose mobilization by the liver during the early phase of haemorrhagic hypotension.

II PITUITARY GLAND

Sheehan, (1937) described massive necrosis of anterior pituitary as post mortum finding in patients who died during puerperium after a delivery complicated by severe haemorrhage. In some cases, there was almost complete necrosis of the anterior pituitary. The cells were rounded off, their nuclei were all small and pyknotic, their cytoplasm was acidophilic. The acidophil cells contained granules in normal amount. The anterior lobe was congested throughout. Many of the sinuses in the necrosed area were dilated and contained some blood, but the blood cells were degenerating. The endothelium lining the sinuses appeared intact, but its nuclei were unusually dark. In other cases, there were only small areas of necrosis, where the parenchyma cells showed coagulative necrosis with very faint ghosts of nuclei still remaining and acidophil cells retained their granular staining. The sinuses were lined by swollen endothelium with healthy nuclei, and occasional polymorphs were scattered throughout the area. The author noticed that necrosis centered usually in the antero inferior part of the anterior lobe in the middle line and spread out to involve most of the lobe. The parts which normally escape were the postero-superior angle beneath and in front of the stalk and a very thin layer on the surface; the middle and posterior lobes were unaffected.