PATHOPHYSIOLOGY & MANAGEMENT OF SHOCK

Essay

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By

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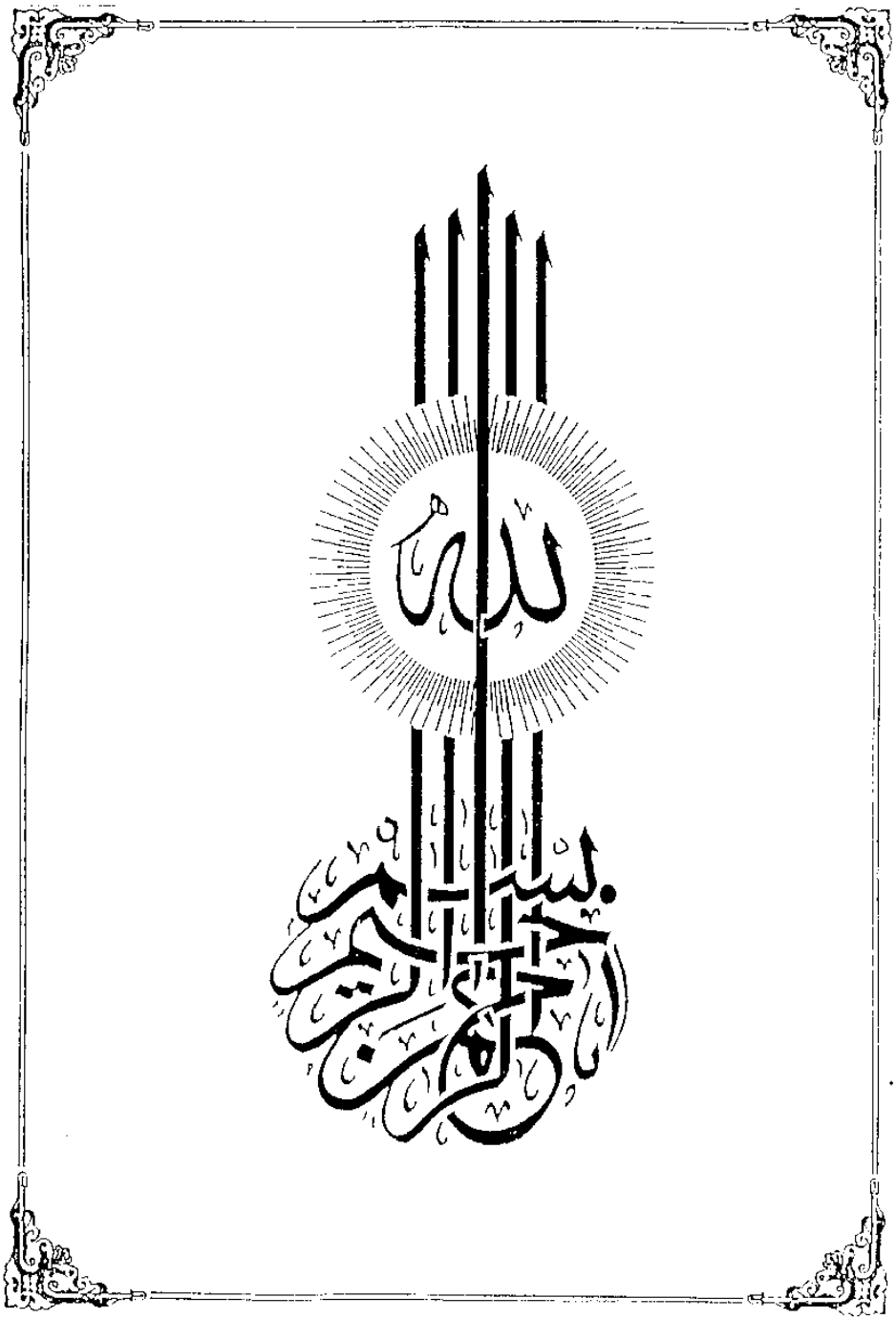

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INTRODUCTION

Shock has been recognised for over 100 years. Decreased tissue perfusion has long been regarded as the basic problem in shock (Shoemaker, 1973).
The major cause of the circulatory collapse is however still debated. By some authors it has been ascribed to a transcapillary loss of fluid and/or a blood stagnation in the systemic vascular beds, reducing venous return to the heart. Others claim that it is primarily a failure of myocardial contractility induced by circulating substances released from ischemic tissues (Haglund, 1978).

In shock vital organs enter in a catabolic phase of metabolism because of either poor tissue perfusion or sepsis. All of the shock states, at least in their severe forms have common pathophysiologic features (Holcroft, 1981).

The corner stone in the management of shock states is to keep the perfusion of different tissues especially the heart and brain.

The aim of this essay is to review the literature about the pathophysiology of shock and the management of its different types.

DEFINITIONS AND CLASSIFICATION

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The earlist definitions of shock were a clinical description of injury. The shock has been defined by Fine (1963) as a species of functional concussion by which the influence of the brain over the organ of circulation is deranged or suspended. Block et al, (1966)defined shock as a progressive vasoconstrictive oligemic anoxia. In 1967, Hardaway et al, defined wound shock broadly as the clinical manifistations of an inadequate volume of circulating blood accompanied by physiologic adjustments of the organism to a progressive discrepancy between the capacity of the arterial tree and the volume of blood available to fill it.

Shock is a breakdown of effective circulation at the cellular level (Trunkey, 1975). It is the inadequate blood flow to vital organs or inability of the body cell mass to metabolise nutrients normally (Maclean, 1977).
Shock of all forms appear to be invariably related to inadequate tissue perfusion. The low flow state in vital organs seem to be the final common denominator in all forms of shock (Shires et al, 1979).

Attempts to classify shock are based on the initial precipitating event (Thal and Kinney, 1967) or on haemodynamic diagnosis (Maclean, 1968).

Etiological Classification of Shock : (Thal & Kinney 1967)

1) Hypovolaemic shock : which may be pure or combined with sepsis or cardiac failure.

2) Cardiogenic shock : as in failure of left ventricular filling or ejection.

3) Septic shock : which may be pure or combined with cardiac failure or hypovolaemia.

4) Neurogenic shock : i.e., loss of vasomotor tone.

Haemodynamic Classification of Shock : (Maclean,1968)

1) Cardiac deficit.
2) Hypovolaemia.
3) Peripheral pooling.

Shires et al (1979) has shown that shock - invariably results from one or more of four but interrelated dysfunctions involving :

1) The pump "heart".
2) The fluid which is pumped "blood".
3) The arteriolar resistance "vessels".
4) The capacity of the venous vessels.

The dysfunctions may be correlated as follows :

1) Neurogenic (Primary, psychogenic, immediate) shock : neurogenic or psychogenic factors e.g. prolonged standing, pain, vasodilator drugs, fright presumably trigger a vaso-vagal reflex as the result of which sudden vasodilatation or inhibition of constriction results in a rapid pooling of blood in the peripheral and splanchnic vessels. This leads to inadequate cardiac output and poor tissue perfusion (Shires et al, 1979).

2) Cardiogenic shock :

Implies failure of the heart as a pump dysfunction from myocardial infarction, serious cardiac arrhythmias or by miscellaneous causes including mechanical restriction of cardiac function or venous obstruction such as occurs in mediastinum, with tension pneumothorax, vena cava obstruction, cardiac tamponade or pulmonary embolism (Braunwald, 1968).

3) Reduction in blood volume :

It may take the form of loss, of the whole blood, of plasma, of extracellular fluid in the extravascular space or a combination of these three. Acute massive haemorrhage is the most direct cause of hypovolaemia, either internal or external. The physiologic essence of the shock syndrome

is disparity between the volume of blood and the volume capacity of the vascular system (Liver, 1977).

An important factor is the rapidity with which the blood is lost. The sudden loss of small quantities of blood taxes the adaptive mechanism to a greater degree than the loss of larger quantities over a longer interval. Exudation of plasma produces shock as a result of a local increase of capillary permeability as in burn and peritonitis or as a result of large quantities of plasma or serum in tissues in severe generalised allergic reations (Anderson, 1967). Dehydration and hyponatraemia are important and direct causes of reduction in blood volume.

Gump et al (1970) believe that within normal limit the loss of one litre of water from the body reduces blood volume by about 75 ml. They also found that the loss of 100 mEq. of sodium result in a blood volume reduction of 115 ml. in an average adult.

Trauma e.g. the crush syndrome produces loss of the whole blood, interstitial fluid and also of plasma.

4) Changes in arterial resistance or venous capacity :

It may result form spinal anaesthesia or from neurogenic reflexes; as in acute pain or may accompany the end stages of hypovolaemia. Septic shock may produce changes in

peripheral arterial resistance and in venous capacity as well as peripheral arteriovenous shunting. Both gram positive and gram negative infection may be responsible and the effects may be attributed to circulating endo-or exotoxin rather than true bacteraemia (Siegel et al, 1971).

Intestinal strangulation, mesenteric vascular occlusion, bile peritonitis, freezing, acute pancreatitis, certain acute pneumonias, irritant war gas poisoning are miscellaneous causes of shock (Harkins, 1970).

EARLY BODY RESPONSES

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The fundamental basis of shock is a sudden decrease in the circulating blood volume. Either due to blood, plasma or fluid loss in hypovolaemic shock, stagnation of blood due to pump flailure in cardiogenic shock, or pooling of blood in the microcirculation in septic and neurogenic shock (Trunkey,1975). This causes reduction in the venous return to the heart and therefore the cardiac output is decreased with resultant hypotension and hence a generalised tissue anoxia (Wilson et al, 1971). As a result, early circulatory, endocrinal and metabolic compensatory mechanisms are brought into play to maintain an adequate circulation to the vital organs.

The Circulatory Responses :

When the cardiac output is reduced, the blood pressure tends to fall as the venous return to the heart decreases. The tonic inhibitory impulses arising from the aortic and carotid sinuses are reduced. Reflex tachycardia and vaso-constriction results. This with increased activity of the vasomotor centre results in an increase in the peripheral resistance and the blood pressure will be maintained (Maclean, 1968). Increasing peripheral resistance by vasoconstriction rapidly becomes generalised and maximal to compensate for the reduced cardiac output and is most marked in the skin,

subcutaneous tissue, skeletal muscles and the splanchnic region but sparing the vessels of the heart and brain.

In hypovolaemic shock the heart may receive 25 % of the total cardiac output as opposed to 5-8 % in the normal condition. The skin becomes pale, clammy and cold. Salivary secretion stops and the mouth becomes dry and thirst results. And concomitantly there's generalised constriction of the veins. It is an important homeostatic mechanism, since about 60-70 % of the total blood volume may be contained within the venous tree. This increases the venous return to the heart and hence the cardiac output (Pardy, 1979).

Transcapillary Refilling :

Another homeostatic mechanism that compensates for hypovolaemia in shock involves mobilization of fluids from the extravascular into the intravascular compartments. Arteriolar spasm results in lowering of capillary hydrostatic pressure and on Starling hypothesis fluid passes into the vessels (Lister et al, 1963). This continues till the reduction in plasma osmotic pressure due to the dilution of the plasma proteins is suffeciently great to offset the forces tending to draw fluid into the blood. As the blood volume is restored, the vasospasm passes off and the capillary pressure rises.

Thus the extracellular fluid passes into the blood till the forces governing the interchange of fluid across the

capillary wall are balanced (Shires et al, 1979).

Both neurogenic and a humoral components are present in the control of the fluid transfer. In the early period reflex activation of the vasomotor fibres contributed significantly to the fluid absorption. The subsequent main part of the fluid gain from the extra-to the intravascular space was due to the action of the blood borne catecholamines. Both the neurogenic and hormonal control was mainly linked to B-adrenergic inhibition of the vascular smooth muscle tone. This control was effected via two mechanisms, viz, by a relatively larger B-adrenergic dilatation of post-than precapillary resistance vessels, leading to adjustment of the ratio of the pre-to postcapillary resistance and thereby to decrease of capillary hydrostatic pressure and via B-adrenergic dilatation of precapillary sphenicters leading to increased capillary surface area available for fluid exchange (Hillman, 1981).

Endocrine Response :

Shock causes endocrinal responses to restore blood volume and maintain flow to vital organs.

1) Catecholamine discharge :

Occurs from adrenal medulla and from the nerve endings throughout the autonomic nerves . Catecholamines produces