PROBLEMS IN SURGICAL SHOCK

THESIS

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M.Ch

By

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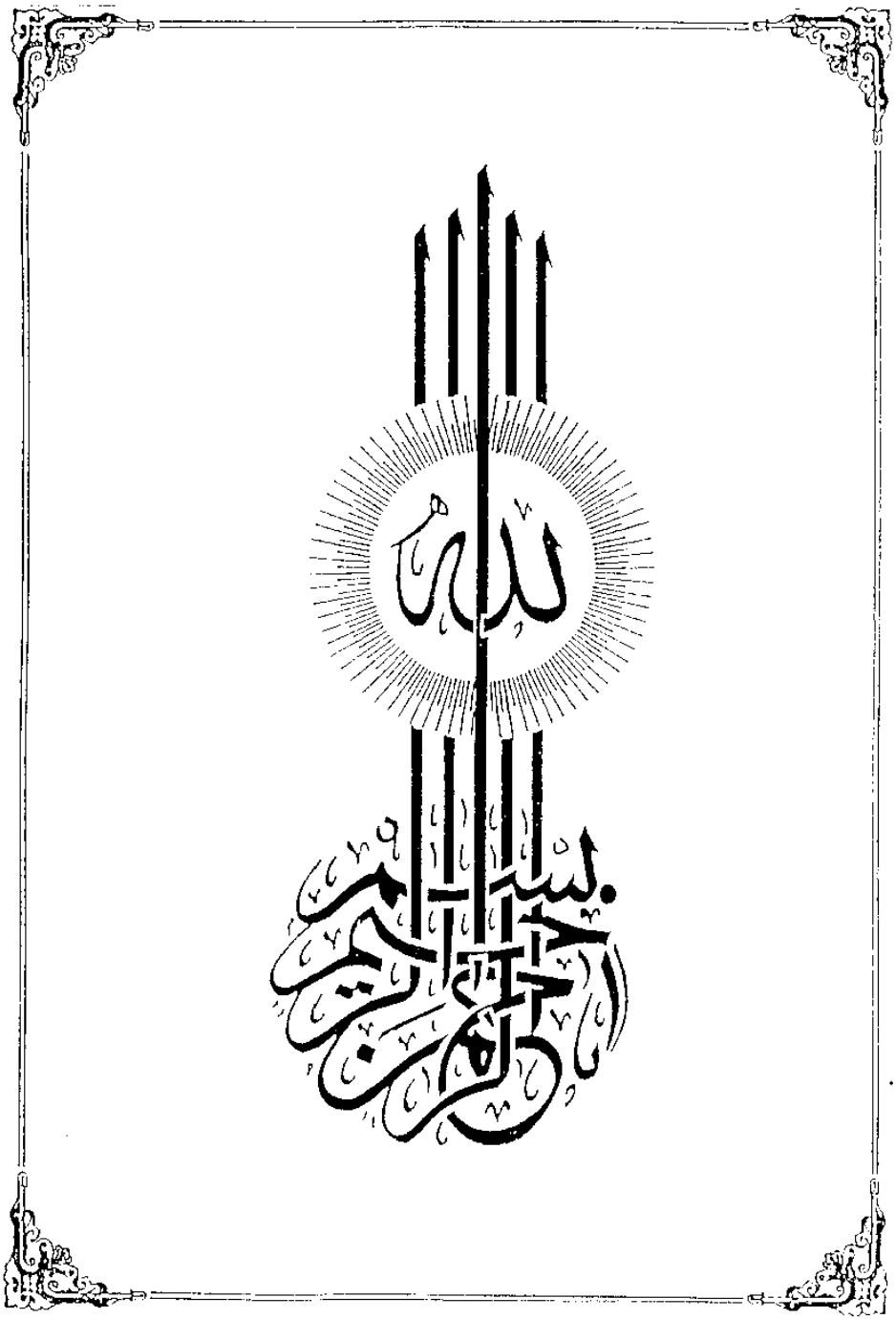
بسم الله الرحمن الرحيم

«وقل رب زدني علما»

صدق الله العظيم



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C O N T E N T S

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INTRODUCTION

I N T R O D U C T I O N

Shock is a descriptive term used by clinicans to denote a syndrome characterized by protracted prostration and hypotension (Weil, 1957). It is usually accompanied by pallor, coldness and moistness of the skin, collapse of superficial veins, alterations of mental status, and suppression of the formation of urine.

Voluminous literature reflects intense disagreement over the meaning and significance of the term "shock". Most of this, however, results from the use of a single name to describe a condition produced by many unrelated causes. Occasion for disagreement disappears if the term "shock" is used within the limits of its definition namely , to indicate the clinical state of the patient without specific implication as to the underlying cause.

Circulatory collapse and peripheral vascular failure are frequently employed by authors to circumvent the dispute aroused by use of the word "shock". These terms, however, are subject to the same misunderstandings, and they add hemodynamic implications that may be misleading. Shock simply refers to the presenting signs of the patient, and within this limit the term is valid and meaningful.

The clinical manifestations of shock often overshadow the primary disease, yet full appreciation of the underlying cause is prerequisite to proper understaning of pathophysiology and treatment of the individual patient. For this reason, use of the term "shock" in a generic sense without reference to the underlying cause has been challenged. Indeed, some competent authorities have urged that patients would be better served if the term were discarded because there is no such thing as treatment of "shock" but only the treatment of the patient in shock in relation to a primary disease. On the other hand, the concept of shock as a syndrome is well established, the term is widely used and, if no more, it communicates the dire clinical state of the patient common to a number of diseases. In fact, it represents the common denominator of a far-advanced defect of vital function, regardless of the initial cause, and as such is the harbinger of death.

HISTORICAL REVIEW

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The word "shock" first used medically by the unamed English translator or Henri Francois Le Dran (1743). A treatise of reflections drawn from experience with gun shot wounds, has been used for more than two centuries in all clinical discriplines to describe a progressive but gradual collapse of vital organs functions after injury or surgery. The initiating injuries seemed disproportionately small when compared with the effect on vital processes. Pathologic findings failed to supply a reasonable cause of death.

At the end of the nineteenth century, surgeons recognized shock when they began to deal with it. The definition at that time was descriptive and usually referred to the post-traumatic or secondary shock. The observations of John Collins Warren (1895) Professor of Surgery, Harvard University are representative.

In 1899, George W. Crile published the first extensive experimental study denoted principally to the solution of the problems of shock. These pioneering experiments were the bases for much of our understanding of traumatic and**hypovolaemic** shock today. He noted that

animal in shock responded to intra-venous adminstration of warm saline. He condented this favourable response with an increases venous pressure, which in turn filled the heart. The heart beat more vigorously and pumped larger quantities of blood, which, he reasoned, nourished the exhausted and starving vital centres, and improved oxygenation within the lungs. Crile (1899) should be credited with observing for the first time the most important characteristic of hypovolaemic shock, i.e. failure of venous return, low central venous pressure and the favourable response of the venous pressure to infusion, accompanied in turn by an increase in cardiac output. He ascribed failure of this treatment, which occured in many of these animals, to exhaution of the vasomotor centre.

During the first two decades of this century, the principal theory to explain the phenomenon of shock was vasomotor which was believed to cause pooling of blood in the great veins with failure of blood flow to vital organs). In treating war casualties, surgeons had noted pallor of tissues of patients in shock, the disappearance, of peripheral pulses at a time when the femoral and carotid pulses were palpable and the constriction of peripheral veins, which supported the latter concept.

It was known at that time that the blood volume was reduced in both clinical and experimental shock, without any indication of external haemorrhage. This observed fact raised the question, where in the body is the blood which is out of currency".

The shock team at Bethume in warld War I noted a high capillary red cell count in patients in shock from trauma without notable haemorrhage. It was not certain at that time whether the increased concentration of red cells was due to transudation of plasma or to slowing of the circulation from a diminished arterial blood pressure.

Members of these early shock teams recognised a difference between blood flow and blood pressure in shock. In 1917 Archibald and Mc Lean noted that while a low blood pressure is one of the most constant signs of shock, it is not the essential thing, let alone the cause of it.

Cannon (1917) believed that the problem of shock still required the demonstration of the same factors, naturally related to the onset of shock, which might so operate in the body that when haemorrhage and infection were ruled out, the persistent low blood pressure characteristic of shock state would become gradually

established. While working with Bayliss in London Cannon (1918) investigated the question of whether the acid known to be developed in injuried tissues might be a contributing factor in the production of shock seen in man. They first showed that acid production was not an essential in lower animals similar to that giving rise to shock in man, the thigh muscles in the anaesthetised cat were repeatedly struk with a blunt wedge shaped hummer, or crushed by compression. The trauma usually failed to break the skin, so that infection from without was unlikely. The course or events was followed by observations of the pulse, respiration, alkali reserve and corpuscular volume. After about 20 minutes the blood pressure began to fall, and a condition resembling post-traumatic shock in man enssued. It was known at the time that Dale and Laidlaw (1910) had been able to induce hypotension by the injection of extremely minute amounts (1 to 2 mg. per kg.) in the anaethetised animal of histamine. The view was taken that secondary shock was due to the action of toxins arising in injuried tissues and histamine was considered the specific offender.

The birth of the Leam approach for investigation and treatment of patient in shock occured during World War I, when American and British physiologists as well as clinicians, led by Bayliss and Cannon (1919) provided the opportunity to called clinical descriptions by line medical officer, and to measure physiologic and biochemical phenomenae in patients with shock.

The final conclusion that a toxaemia, possibly involving a histamine-like substance, was produced in the wound was misleading. The observations of Keith and of Roberston and Bock (1919), which related the presence or absence and the severity of shock to deficits in circulating blood volume, were recognised but unfrotunately believed at that time to be of secondary importance.

A most important observation by Cannon (1919) was the correlation between low blood pressure and arterial blood acidosis. He postulated that the fall in alkali reserve was due to the accumulation of fixed acids, such as lactic acid, as a result of impaired oxygen transport. He noted improvement in shock after the administration of sodium bicarbonate.

Blalock (1930) separately Phemister and Handy (1927) challenged the theory of toxaemia as a mechanism of traumatic shock, and utilizing a model similar to that of Cannon and Bayliss, (1923), demonstrated that the shock observed after trauma to a dogs leg was due clearly to the accummulation of blood and plasma in and around the wound and throughout the tissue spaces far beyond the area of local injury. It was found that trauma of sufficient severity to cause low blood pressure produced extravasation of blood not only into the tissues of the groin and flank, therefore amputation of the upper part of the thigh to demonstrate the difference in weight between the injuried and uninjuried legs as had been done by Cannon and Bayliss (1923) was shown to be inadequate. A wider dissection that permitted weighing the two halves of the lower body demonstrated that the blood and plasma lost into the traumatised side could easly explain the shock observed.

Blalock (1930) noticed that transufsion of blood from one dog in which a low blood pressure had been produced by trauma to an extremity, to another dog, in which a low blood pressure had been produced by loss of blood either outside the body or into the tissue of the