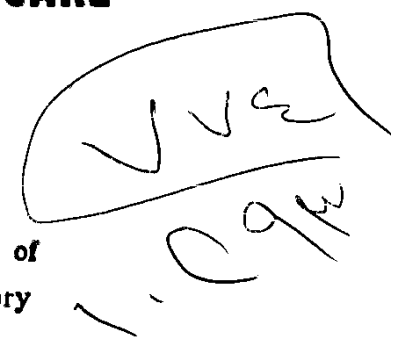
SURGICAL INTENSIVE CARE



AN ESSAY

Submitted for Partial Fulfilment of
Master Degree in General Surgery

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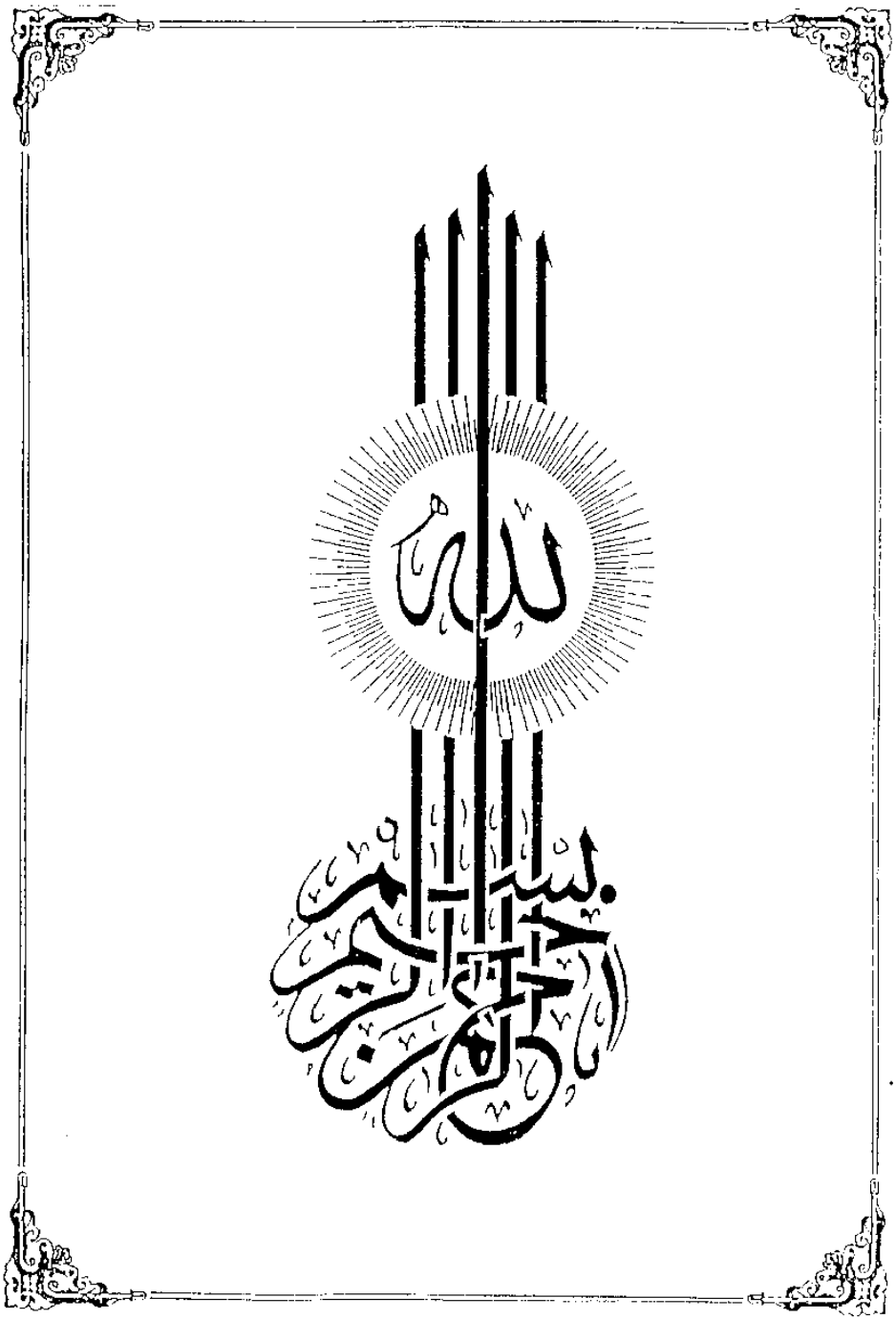

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CHAPTER I

INTRODUCTION

The objective of the surgical intensive care unit is to provide maximal care for the patient in the most effective and efficient manner possible for optimal results.

This requires consideration of several essential components, including the physical location of the unit and its proximity to diagnostic and therapeutic modalities such as the operating room, recovery room, X-ray unit, and laboratories as well as to well-trained personnel and adequate equipment.

The increasing complexity in management of the critically ill has resulted in a sphere of activity that is beginning to evolve as a subspeciality.

In most major hospitals, surigcal special care, critical care, and intensive care units are now physically and administratively separate from medical units.

The development of the area of critical care of the surgical patient has been accompanied by the designation of directors of surgical special care units.

Whereas formerly most directors were anaesthesiologists, today they are more often surgeons who consider this an area of special surgical challenge.

The intensive care unit is an area of polypharmacology. A multitude of interventions to treat and/or prevent established conditions may or may not be appropriate.

This essay attempts a global approach to aspecific area.

It is hoped that it may provide a ready and effective reference source for the individual who is in the front line, who faces problems requiring immediate solution.

So, here, we try to give an approach to the normalities and abnormalities of the body physiology and how to face these abnormalities and how to care with patients in the intensive care units.

HOMEOSTASIS

CHAPTER II

Homeostasis

Bodily changes in trauma and surgery

Changes in biochemistry, metabolism and visceral function :

1. LOSS OF BODY CELL MASS :

The basic transaction between the body cell mass and the extracellular fluid after injury is the lysis of cellular protoplasm and the conservation of extracellular fluid, some compounds are converted to glucose and burned through the carbohydrate-oxidative pathway, and most of the nitrogen is excreted in the urine as urea.

The tissue most prominently involved is skeletal muscle, this is demonstrated by the increased amount of creatine and creatinine in blood and urine and by the evident rapid decrease in the bulk of palpable muscles.

Transient immobilization and starvation add to this impost on the skeletal muscle mass.

This release of the products of cellular protoplasm to the extracellular fluid accounts for several events : the negative nitrogen balance, the appearance of new

glucose from noncarbohydrate sources, the relative inability of the body to synthesize new muscle protein immediattely after injury, and the loss of intracellular electrolytes (particularly potassium, phosphate and sulfate) into the extracellular fluid and thence to the urine via the kidneys as well as the appearance in the urine of increased amounts of xanthines found in muscles (creatine, creatinine, uric acid).

The normal draft on muscle cells after medium-grade injury in the adult amounts to about 30 gm. of nitrogen, corresponding to about 220 gm. of protein, and approximately I kg. of lean tissue. This is about 5 percent of the fat-free body mass, Moore, (1981).

II. THE METABOLIC RESPONSE TO INJURY :

The metabolic response to injury may be divided into three phases :

(1) Early (or "ebb") Phase, lasting 12-24 hours after injury.

(2) Catabolic (or "flow") Phase, lasting several days to severalweeks.

(3) Recovery (or anabolic) Phase.

During the early phase body temperature and oxygen consumption are reduced. Insulin secretion is suppressed and levels of circulating catecholamines are very high.

The catabolic phase has been the subject of intensive investigation.

The characteristics of this phase are :

(a) Increased resting metabolic expenditure (RME).
(b) Increased O_2 consumption.
(c) Sodium and water retention (usually with a fall in the serum sodium concentration.
(d) Increased lipolysis and gluconeogenesis.
(e) Increased catecholamine secretion (though not to the extent seen in the early phase).
(f) Increased levels of glucagon, cortisol and growth hormone.
(g) Normal or raised insulin secretion, but with insulin antagonism at cellular level.
(h) Negative nitrogen balance.

During the recovery phase the losses sustained during the catabolic phase are restored. Nitrogen balance becomes positive, weight returns to its previous value and the patient resumes normal activities.

The ability to work a full day without undue fatigue shows that recovery is complete. The change from catabolic to recovery phase is accompanied by an improvement in morale and a return of appetite. The speed with which this change can occur has been compared to turning a metabolic switch. Seeley, (1982).

III. CONSERVATION OF EXTRACELLULAR FLUID :

While the body mass is being reduced by lysis of protoplasm, the organism is devoting several active mechanisms to the conservation of extracellular fluid and thus to the maintenance of plasma and blood volume.

The tendency to conserve extracellular fluid after trauma is manifested by a decreased absolute sodium excretion rate, a decreased sodium concentration in the urine, an inability to excrete sodium bicarbonate, a tendency toward aciduria, and a decreased sodium content of saliva and sweat. There is some evidence that the distal small bowel also participates in this regulation with increased reabsorption of sodium from the distal ileum.

The volume-conserving mechanisms consist primarily in the reduction of sodium loss in urine, saliva, and

reduction in water loss in all these body fluids, including gastrointestinal juice.

In many post-traumatic states, there is a trade-off or ion pair effect between sodium and either potassium or hydrogen ion.

When sodium is retained, potassium and hydrogen ions are lost or move into cells, and there is a tendency toward extracellular alkalosis. By contrast, when sodium is lost, potassium and hydrogen ions are retained (or move out of the cell), and there is a tendency toward extracellular acidosis and hyperkalemia.

The plasma concentrations of sodium and potassium characteristically move in opposite directions.

The administration of large loads of sodium-containing fluids after operation or injury will give the illusion of overcoming a tendency toward sodium conservation because sodium excretion is increased thereby. But retention still occurs, and the ease with which positive sodium balances are achieved after trauma, demonstrates that renal salt conservation is still present.

Retention of the sodium ion in the body tends to maintain the volume of extracellular fluid.

After injury, another fluid-conserving mechanism also comes into play in the form of a sharp restriction in the excretion of free water, or antidiuresis. The tendency to conserve body water is demonstrated by a rising urine osmolality due to distal tubular water reabsorption. With the urine sodium content markedly reduced, the increased urine osmolality must be due to the presence of other substances at increased concentrations. These are chiefly phosphate, potassium, and urea.

Large water loads given to postoperative patients will increase water excretion and thus (as with the case of sodium) give the superficial appearance to the uncritical observer of overcoming the water-conserving mechanism, Moore, (1981).

IV. HEMATOLOGIC EFFECTS :

The most predictable post operative hematologic changes are the platelet tide, an early thrombocytopenia and a later thrombocytosis.

The early fall in platelets amounts to between 30 and 50 percent below the preoperative level and

starts, as does the eosinopenia, shortly after the beginning of the operative experience. It lasts longer, however, than does the eosinopenia, namely, up to the fifth day. The thrombocytosis that follows is of a magnitude of between 50 to 100 percent above the preoperative level.

The degree of the response depends on the magnitude of the operation. If the bone marrow is examined at the times of the greatest alteration in the level of blood platelets, characteristic changes in megakaryocytes are observed. In the early phase an excess of those forms that are loaded with platelets, the D-forms of de la Fuenta is seen, Williams et al., (1955).

A corresponding decrease occurs in the late phase. These observations suggest a suppression followed by a release of platelets by the megakaryocytes.

Decreased labile factor (Factor V-proaccelerin) occurs during the first week. The serum prothrombin also decreases.

Fibrinogen is increased postoperatively. This becomes manifest on the third postoperative day and