" POTASSIUM AND ANESTHESIA

Essay

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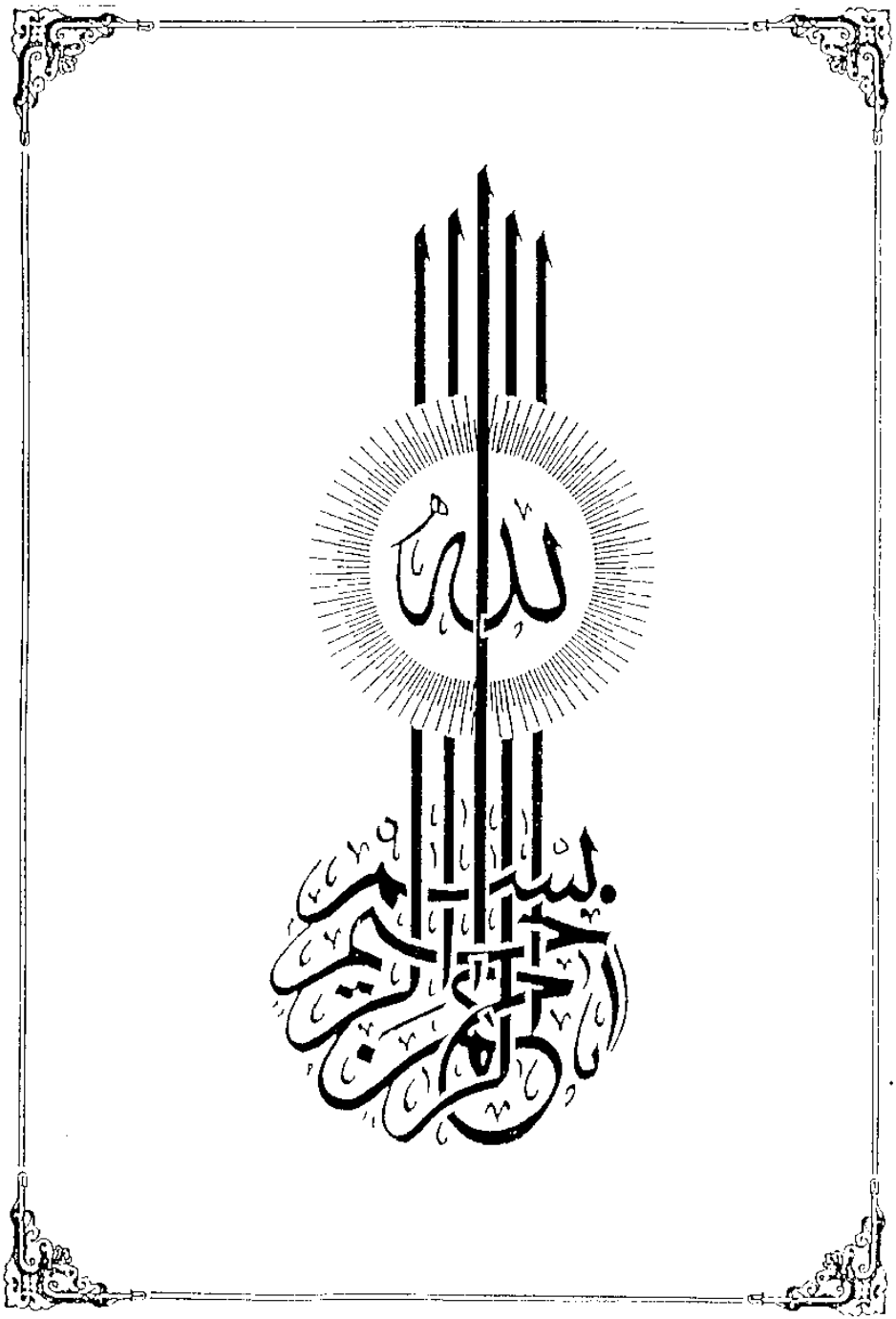
بسم الله الرحمن الرحيم

" قالوا سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم "

(صدق الله العظيم)



To My Family

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INTRODUCTION

Potassium is the second common cation in the body and the principle intracellular cation.

Potassium plays a major role in the electrophysiology of cell membranes as well as carbohydrate and protein synthesis. The excitability of the cells is proportional to the ratio of intracellular to extracellular potassium concentrations. Consequently , any variations in this ratio will affect cellular function , result in dysfunction of excitable cell membranes and cause the signs and symptoms that are associated with imbalances of potassium concentration (***De Wardener, 1985***).

Disturbances in potassium homeostasis are not surprisingly , connected to a wide variety of pathological conditions such as arrhythmia , acid - base disturbance , digitalis toxicity and myocardial infarction (***Tetzlaff et al. , 1993***).

The role of potassium therapy in the preoperative period has evolved within the last decade as studies evaluated the risk of morbidity. Earlier work suggested that all patients with hypokalemia were at risk for serious cardiac arrhythmia during general anesthesia. Recent work has failed to detect an increased incidence of serious arrhythmia in the chronically hypokalemic , otherwise healthy patients and even in high - risk patients. As a result , acute potassium therapy is less

common due to its known risks , and cancellation of elective surgery for arbitrary potassium values is less common. This doest not extend to acute potassium disturbances or chronic disturbances in some patients (digitalis therapy , acute cardiac ischemia , etc.) (***Tetzlaff et al. , 1993***).

Severe hyperkalemia poses a potentially life - threatening emergency because of the risk of cardiac arrest. The level of serum potassium and the presence or absence of accompanying electrocardiographic changes should determine the aggre - ssiveness of the therapeutic approach. Therapeutic strategies commonly used are aimed at counteracting cardiac toxicity , shifting potassium into cells , and removing the excessive potassium burden from the body (***Lutarewych and Batlle , 1991***).

" ANATOMY OF TOTAL BODY POTASSIUM CONTENT "

Anatomy of total body potassium content :

Total body potassium is about 50 mmol.Kg^{-1} of body weight or about 3500 mmol in a 70 Kg person (***Brown , 1984***).

As much as 98 % of total body potassium content is confined to intracellular water , the remaining 2 % being located in the extracellular fluid compartment. The bulk of intracellular potassium is contained in skeletal muscle cells , with comparably smaller quantities in liver , bone , skin and red blood cells (***Lutarewych and Batlle , 1991***).

The concentration of potassium in the extracellular fluid is about 4 mmol.L^{-1} and the intracellular concentration is 150 mmol.L^{-1}. Assuming a 20 L extracellular space and a plasma concentration of 4 mmol.L^{-1} , the plasma content of potassium would be only 80 mmol of the 3500 mmol in total body content (***William and Epstein , 1989***).

The serum value of potassium is remarkably constant despite a wide variance of dietary intake and urinary excretion. Body potassium homeostasis is driven to maintain the extracellular concentration at the expense of total body content to maintain membrane function (***Brown , 1984***).

Estimation of total body potassium concentration from serum values is inaccurate because of preponderance of intracellular potassium , although the majority (more than 90 %) is readily exchangeable between compartments (***Tetzlaff et al. , 1993***).

There is a rough correlation between serum potassium and total body potassium excess or deficit. When potassium depletion occurs , the serum concentration reflects the total body deficit in a linear relationship for deficits up to 500 mmol. With each acute decrease of 0.27 $mmol.L^{-1}$ serum potassium being equivalent to 100 mmol of total body deficit. In contrast , relatively minimal increases in total body potassium are accompanied by a sharp increase in plasma potassium (***Lutarewych and Batlle , 1991***).

Even severe degrees of total body potassium deficit , increase or internal distribution have a relatively minor impact on the intracellular potassium concentration. Accordingly , the ratio of internal to external potassium , which determines the membrane potential varies mainly as a consequence of changes in extracellular potassium ; thus , it is important to maintain extracellular potassium within the normal range (***William and Epstein , 1989***).

" POTASSIUM AND EXCITABLE CELLS "

Potassium and excitable cells :

All cells have an electrical potential difference across their membrane that is determined by the concentration of various ions in the intracellular and extracellular space and the permeability of the membrane to each ion (***Wong et al. , 1993***).

The primary extracellular ions are sodium and chloride , whereas te primary intracellular ions are potassium and negatively charged proteins and metabolites (***Wong et al. , 1993***).

The 40 - fold concentration difference of potassium is maintained by the Na^+ / K^+ adenosine triphosphatase (ATPase) pump located in the cell membrane. Extracellular potassium is pumped into the intracellular space , whereas intracellular sodium is pumped into the extracellular space. Transport of these ions occur against a high concentration gradient with energy derived from the hydrolysis of adenosine triphosphate (***Ganong , 1993***).

The cell membrane normally is highly permeable to potassium , which diffuses out of the cell but is nearly impermeable to sodium (***Solomon and Katz , 1986***).

The resting membrane potential is generated largely by the diffusion of potassium out of the cell into the extracellular

fluid down a concentration gradient. Since potassium is positively charged , this movement of potassium makes the interior of the cell electrically negative with respect of the exterior (***Ganong , 1993***).

The resting membrane potential can be expressed mathematically by the Nernst equation , if the cell membrane is permeable only to potassium (***Tetzlaff et al. , 1993***) :

$E_m = - 61 \text{ Log } K_i / K_e$

Where :

E_m = resting membrane potential.

K_i = intracellular potassium.

K_e = extracellular potassium.

$$E_m = - 61 \text{ Log } \frac{160}{4.5}$$

= - 95 mv (Cell interior is negative).

However , the cell membrane has a limited permeability to sodium. As a result , the resting potential is more accurately described by the Goldman equation (***Goldman , 1943***).

$$E_m = - 61 \text{ Log } \frac{K_i + 0.01\ Na_i}{K_e + 0.01\ Na_e}$$

= - 88 mv

Where :

Na_i = intracellular sodium.

Na_e = extracellular sodium.

During excitation , the release of acetylcholine at synapses and motor end plates results in a decrease in the magnitude of the membrane potential to a critical value referred to as the threshold potential (Et). In skeletal muscle , the normal resting potential is approximately - 88 mv and the normal threshold potential is approximately - 65 mv. When the threshold potential is reached , certain sequence of changes in the membrane potential constitutes the action potential. The action potential occurs in two separate stages called membrane depolarization and membrane repolarization. When the permeability of the membrane to sodium ions suddenly increases , many of the sodium ions that are present in very high concentration outside the fiber rush to the inside to cause complete disappearance of the normal negative resting potential , and usually enough charges actually to develop a positive state inside the fiber. This sudden loss of normal negative potential inside the fiber is called depolarization. The positive potential that develops momentarily inside the fiber is called the reversal potential. Almost immediately after depolarization takes place , the pores of the membrane again become almost impermeable to sodium ions at the same time considerably more permeable than normal to potassium ions. Therefore , sodium ions stop moving to the inside of the fiber , and , instead , potassium ions move to the outside because of the high potassium concentration on the inside. Thus , because the potassium ions are positively charged , the excess