REGULATION OF FLUID AND CELL VOLUME DURING SIMULATED MICROGRAVITY AND SPACE FLIGHT

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INTRODUCTION: Body fluid regulation in space differs from that on earth. A number of experimental models have been employed to simulate the physiologic conditions of microgravity: head-out water immersion (HOWI), head-down bedrest (HDBR), supine position, and parabolic flights. Although in each case there is a shift of fluid cephalad from the lower extremities, recent studies demonstrate that none of the models mimic the plasma volume, hormonal, or renal functional changes seen in space flight.

METHODS: A medline search was performed to obtain recent articles relating to fluid balance and kidney function in space. An emphasis was placed on obtaining data relating to fluid shifts between body compartments and changes in cell volume.

RESULTS: In HOWI, there is a shift of blood volume into the thorax. HOWI causes an increase in hydrostatic pressure reducing the vascular capacitance resulting in peripheral vascular filling and high perfusion flow to tissues. (1) There is an increase in renal plasma flow, but the glomerular filtration rate remains stable, resulting in an increase in filtration fraction. A natriuresis and diuresis ensues. There is a decrease in the release of norepinephrine, renin, and antidiuretic hormone. (2)

In microgravity of space flight, there is also a shift of the blood volume cephalad. Because of the absence of hydrostatic pressure, there is no compression of peripheral vessels. This favors an increase in venous capacitance and lower peripheral vascular filling. This is sensed by baroreceptors resulting in increased release of norepinephrine, renin, and antidiuretic hormone. (3) Renal blood flow remains stable and the GFR is stable or increased, which results in an increase in filtration fraction. (4) Despite the increase in filtration fraction, as opposed to the findings in HOWI, there is no increase in sodium and water excretion seen in space. Recently it was found that there is net sodium retention during long term space flight without apparent water retention. (5)

Total body water has been shown to remain constant during space flight. (6) It was found that both plasma volume and extracellular fluid volume decreased during flight. This provides evidence that there is a shift of fluid from the extracellular to intracellular fluid compartment. Because the plasma volume decrease was small, most of the fluid shift likely came from the interstitial compartment.

The shift of fluid to the intracellular space would result in an increase in cellular volume. It is interesting to speculate on what basis this might occur. Regulation of cell volume is a fundamental process, critical to the function of all living cells. (7) The steady state volume of the cell reflects its content of osmotically active solute. This includes both diffusible solutes (such as electrolytes) and non-diffusible solutes (such as proteins). An increase in cellular volume is due an increase in intracellular osmolality. There are a large number of factors which play a role in cell volume regulation which might be altered in zero gravity. (8) The cell cytoskeleton plays an important role in the maintenance of cell volume and there is evidence from plant studies that the microtubular network may be altered in zero gravity. (9) Alterations in levels of different hormones can affect cellular uptake of solutes. Changes in metabolism may alter the concentrations of intracellular non-diffusible solutes.

The volume of red blood cells (RBCs) has been determined during space flight. (10) There actually was found to be a decrease in mean cell volume of RBCs, which appears to be due to a selective destruction of newly produced RBCs (which have a larger volume than mature erythrocytes).

CONCLUSIONS: There are significant differences in regard to fluid an electrolyte balance between the models which simulate microgravity on Earth and what occurs during space travel. There is an apparent shift in fluid from the extracellular to intracellular compartment in space (6), the mechanistic basis of which is unknown. Further studies to expand findings of Leach et al (6) are needed.

REFERENCES: