Computer Systems Analysis of the Cardiovascular Mechanisms of Reentry Orthostasis in Astronauts

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Abstract

Reentry orthostasis secondary to a prolonged exposure to microgravity is a common problem among astronauts. However, the physiologic mechanisms are poorly understood due to the many control systems involved. In this study an advanced computer model of cardiovascular functioning was employed in a systems analysis approach to clarify the relative importance of some of the adaptive physiologic processes engaged when humans return from space. After simulation of the conditions of zero gravity for one month, the model predicted that the change in capacitance of the lower extremity veins resulting from a loss of external fluid forces in the dehydrated extracellular compartment was the dominant mechanism associated with reentry orthostasis. This condition appears accentuated in women due to their inherent lower center of gravity and proportionately larger mass in the lower extremities
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1. Introduction

The exploration of space is one of the last great frontiers. However, there are still many physiologic barriers that need to be resolved before further advances and longer duration missions can be achieved. Many astronauts experience orthostatic intolerance upon initial reentry into earth's gravitational field [1]. Beyond being simply an anomaly of space travel, this condition could pose a real threat in the event of a necessary emergent return to earth and could impact pilot performance. In addition, a mission to other planets such as Mars could be limited by these physiologic constraints. Our understanding of the mechanisms responsible for the hypotension seen after exposure to microgravity is poorly understood and requires a dissection of many complex control mechanisms. A systems analysis approach using modern computer simulation techniques permits a more detailed examination of this common problem.

2. Methods

A special adaptation of a well-established large integrative computer model of circulatory physiology (QCP www.biosim.com) was constructed using data acquired from shuttle astronauts within hours of landing. The model contains over 4000 equations that describe the detailed interaction of multiple aspects of the circulatory system as regulated by neural, endocrine and metabolic control mechanisms [2]. The integrated relationships are based upon basic physical principles and well-established functional physiologic interactions [3]. Specifics peculiar to exposure to microgravity including baroreceptor deconditioning, compartmental fluid shifts as well as the special anatomic differences in men and women were also incorporated into the structure of the mathematical model. The female gender differences were simulated by a 15% caudal shift in the center of gravity of the model's anatomic structure with a resulting relative increase in the proportional mass in the lower body compartment. However, no gender distinctions were otherwise incorporated in the physiologic functioning of the model. The newly formulated model was then used in a series of computer simulation studies solved on a Windows-based PC using standard numerical methods to develop a working hypothesis concerning the physiologic origins of reentry orthostasis [4].

3. Results

After simulation of the conditions of zero gravity for one month, the model predicted the typical changes in vital signs and hemodynamics observed in astronauts during spaceflight. Also noted were the adaptive compensatory changes produced as fluid shifts from dependent areas and result in a diuresis with loss of plasma volume, resetting of the baroreceptors while maintaining affective central volumes and cardiac output (figure 1).

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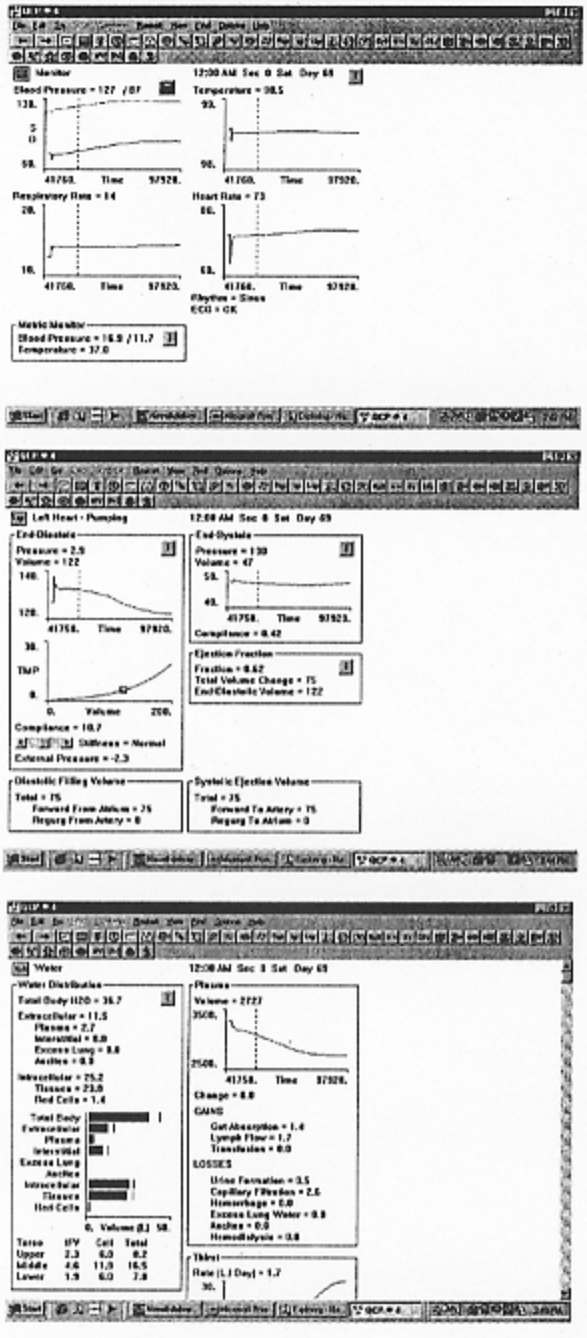

Figure 1. Hemodynamic changes in microgravity.

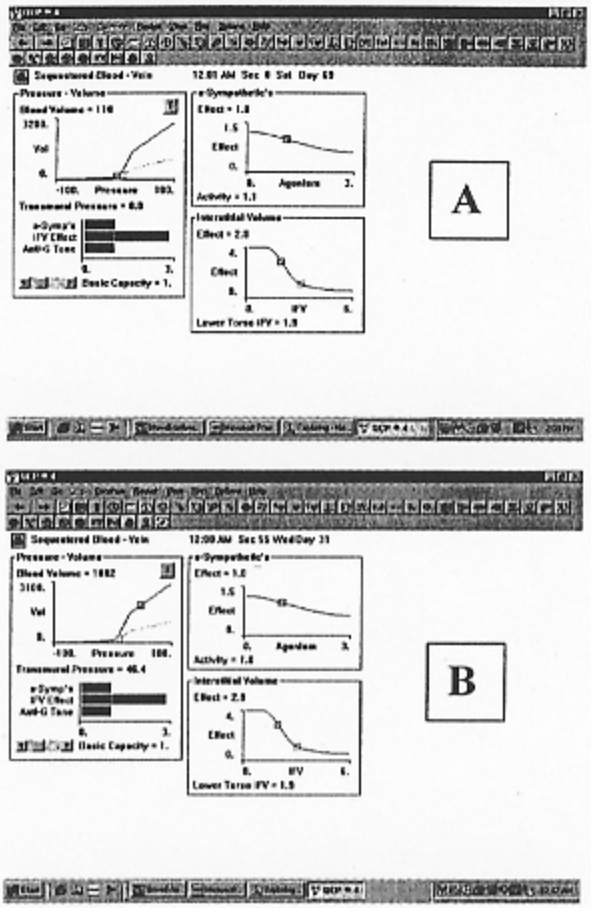

Figure 2. Changes in the leg vein compliance after fluid shifts in microgravity (A) and upon reentry (B).

Of particular interest is the relative contracture of the extracellular fluid compartments and change in capacitance of the veins in the lower extremities secondary to this volume loss. The compliance (pressure-volume relationship) of these veins is determined by:

1. adrenergic tone
2. surrounding muscle tone
3. external compressive forces of the interstitial fluids

Exposure to microgravity shifts this pressure-volume curve secondary to the loss of fluid from the interstitium (figure 2). During spaceflight this compliance change has little impact on hemodynamics due to the low pressure requirements necessary to drive venous return. Upon reentry into earth's gravity, the model predicted a sequestering of blood in these now lower compliance vessels with a resulting orthostatic intolerance occurring when the astronaut stands (figure 3).

Compensatory mechanisms counteract the fall in blood pressure in most individuals and the effects are noted to be transient (figure 3). While varying the cardiac function and baroreceptor sensitivity can potentiate this intolerance, the change in capacitance of the lower extremity veins resulting from a loss of external fluid forces in the dehydrated extracellular compartment was the dominant mechanism associated with reentry orthostasis.

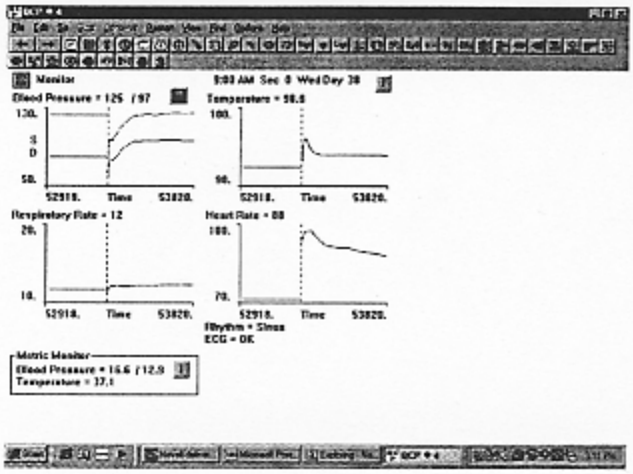

Figure 3. Hemodynamic transients and compensations upon reentry into gravity.

This condition is thought to be accentuated in women due to their inherent lower center of gravity and proportionately larger mass in the lower extremities [6]. When this simple anatomic assumption is incorporated into the simulation, the orthostasis was more pronounced and overwhelmed all the counter-regulatory interactions (figure 4). When the same female model is treated with midodrine (an α agonist) there is a marked improvement in the orthostatic intolerance (figure 5) by reducing the relative importance of the volume depletion on leg vein compliance (figure 6).

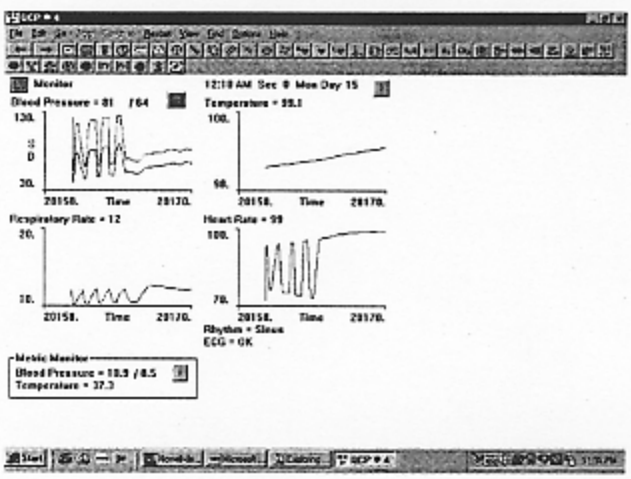

Figure 4. Hemodynamic transients in females upon reentry into gravity.

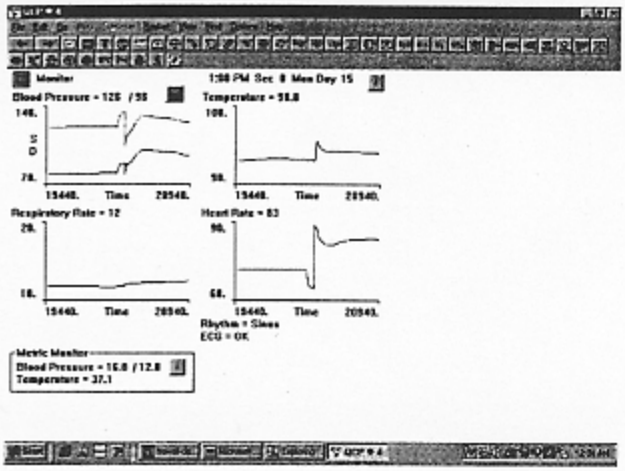

Figure 5. Hemodynamic transients in females upon reentry into gravity after midodrine treatment.

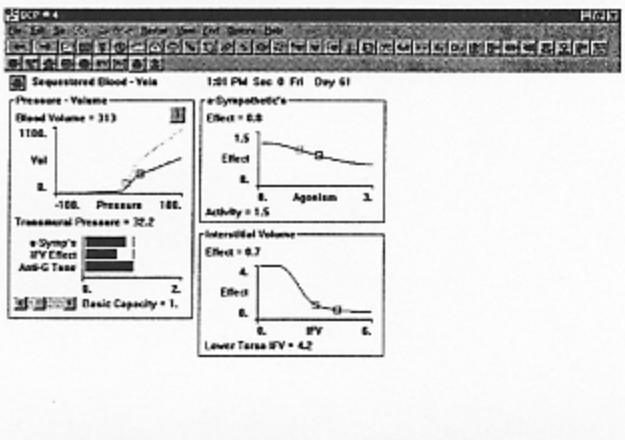

Figure 6. Leg vein compliance in females upon reentry into gravity after midodrine treatment.

4. Conclusions

A systems analysis approach using a computer model of cardiovascular physiology was employed to examine the important mechanisms involved in reentry orthostasis after spaceflight. Changes in capacitance in lower extremity veins due to a volume contracture of the surrounding interstitial spaces and their influence on venous return were found to be causally more important than alterations in nervous and hormonal reflexes effecting peripheral resistance or cardiac function. Alterations in the compliance of the vasculature of these dependent areas results in an increase in sequestered blood volume and appears to be the ***dominant*** mechanism

responsible for reentry orthostasis. Other factors such as attenuation of baroreceptor sensitivity, decreases in peripheral resistance, and dehydration can further exacerbate the orthostasis. Further validation of the model with experimental data and testing of the hypothesis is needed.

Acknowledgments

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