The following is the abstract of the article discussed in the subsequent letter:

Perhonen, Merja A., Fatima Franco, Lynda D. Lane, Jay C. Buckey, C. Gunnar Blomqvist, Joseph E. Zerwekh, Ronald M. Peshock, Paul T. Weatherall, and Benjamin D. Levine. Cardiac atrophy after bed rest and spaceflight. J Appl Physiol 91: 645–653, 2001.—Cardiac muscle adapts well to changes in loading conditions. For example, left ventricular (LV) hypertrophy may be induced physiologically (via exercise training) or pathologically (via hypertension or valvular heart disease). If hypertrophy is treated, LV hypertrophy regresses, suggesting a sensitivity of using nocturnal lower body positive pressure (LBPP) as a countermeasure against microgravity-induced cardiac atrophy. Reduced myocardial mass in chronic microgravity, then replacement via LBPP of this nightly cardiac distension may help prevent such myocardial atrophy.

Previous work demonstrates that central volume expansion at night does not elicit diuresis to the extent that similar volume expansion provokes during the day (e.g., Ref. 5). Therefore, nocturnal LBPP should not exacerbate microgravity-induced hypovolemia if nighttime antidiuresis operates during spaceflight similar to Earth-bound circadian conditions.

In regard to microgravity-induced hypovolemia, from the discussion of Perhonen et al. (4) on diuresis during the first 24 h of bed rest, a reader could reasonably conclude that a similar renal response occurs during spaceflight. However, diuresis is not seen in flight; in fact, existing data suggest acute and chronic antidiuresis early in spaceflight (2, 3, 6). I respect that Perhonen et al. did not state that “spaceflight causes diuresis.” However, because this is a widespread misconception, these differences between bed rest and spaceflight need to be explicitly recognized.

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To the Editor: Dr. Watenpaugh presents an interesting hypothesis, namely, that providing a positive pressure to the lower part of the body at night, also called LBPP, could stretch the heart and minimize cardiac atrophy during exposure to microgravity. Pilot work in our laboratory could not confirm a sustained increase in either central venous pressure or stroke volume from LBPP, possibly due to stress-relaxation of the venous capacitance vessels or even translocation of blood past the heart into the head and neck. Therefore, we have not been enthusiastic about applying this stimulus as a tool to expand the heart during our experiments. However, the idea certainly is intriguing, and we look forward to seeing such experiments conducted by Dr. Watenpaugh or others.

Dr. Watenpaugh also rightly points out that a diuresis has not been observed during early spaceflight, and we were careful not to state so explicitly. However, as noted by Dr. Watenpaugh himself (2), much of this failure may be due to the specific circumstances of flight experiments and the specific body position to which the comparison is being made. In space, the first measurements are usually not made until after the astronauts have been lying for many hours with their feet up in the air in the “prelaunch” position. Thus much of the short-term diuresis and natriuresis that would normally occur with the transition from standing upright in 1 G to microgravity has already occurred before launch. Subsequent measurements are then affected by limited fluid intake during the first few hours in space, in part because of the development of space motion sickness and also because of reduced access to fluids. Ultimately, it has been well documented that plasma volume is reduced early during exposure to spaceflight (1), although it is not certain whether this reduction occurs in the kidney or in the extravascular space. We agree with Dr. Watenpaugh’s speculation (2) that if an astronaut could be transported immediately from the upright position on Earth to space then a diuresis would likely be observed, as has been noted in virtually all ground-based models.

REFERENCES


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