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Space Flight and Lunar Dust Hypertension

William J. Rowe*

Former Assistant Clinical Professor of Medicine, Medical University of Ohio, Toledo, USA

***Corresponding author:** *William J Rowe, MD FBIS FACN, Former Assistant Clinical Professor of Medicine, Medical University of Ohio, Toledo, 1485 Bremerton La. Keswick, VA 22947, USA, Tel: 434-9840079, E-mail: rowefemsinspace@gmail.com*

As a member of a North American Fitness Delegation to China in 1988, with an invitation to present a paper in Guangzhou, I chose as a role model Sy Mah, holder of the Guinness Book of Records for having completed 524 marathons. Mah, a professor of physical education at the University of Toledo, was on a sabbatical in Boston and drove all night for his stress test in the morning; it was very abnormal with evidence of severe ischemia. Because of time constraints, I took him to the Medical College at the University of Toledo later in the afternoon for a repeat stress test in conjunction with a maximum oxygen uptake; to my surprise, the stress test was now perfectly normal; this was consistent with coronary vasospasm possibly triggered by a calcium overload, secondary to a reduction of magnesium (Mg) [1].

Ten months later, Sy Mah died of a lymphoma. Because of my concern that, with his having completed as many as 40 marathons a year, over a 20 year period, I sent his heart to Dr William Roberts, arguably one of the world leaders in cardiovascular pathology; I asked Professor Roberts whether there was anything to suggest a catecholamine -producing tumor i.e. pheochromocytoma. His response was that there was nothing specific to suggest this, but there were a couple of areas of focal fibrosis of the papillary muscles, consistent with spasm of blood vessels secondary to high catecholamine's (C).

Since at age 62, his coronary blood vessels were normal, I developed the hypothesis that extraordinary unremitting endurance exercise can injure a normal heart. I stressed the fact that with C elevations there would be, in turn, reductions of Mg ions and the potential for vicious cycles between the two, as well as well as with ischemia [2]. Ithen postulated that since C elevations have been shown in experimental animals after relatively brief Space missions with destructive changes in the mitochondria and various microcirculatory disorders leading to serious myocardial pathology [3], this mechanism might occur as well, in astronauts subjected to progressively longer missions.

When I presented my first space-related paper at the 11th Man in Space Symposium, in Toulouse, France in 1995, there were considerable objections by investigators both from the U.S. and Russia. Their opposition was based on their studies of humans after relatively short missions lasting up to several months, showing no evidence that C were elevated. The moderator concluded with the statement that perhaps by 20 years, we will learn whether there are C elevations in Space.

It certainly didn't take that long; in 2001, it was shown that plasma Norepinephrine was approximately twice the value of the supine position on Earth [4]. I believe that the underlying mechanisms triggering these C elevations are related to the combination of significant Mg deficits as shown in 196space shuttle crew members ($p < .0001$) [5] and invariable dehydration [6]. Significant Mg deficits can be instigated by the combination of malabsorption through the gastrointestinal tract [7] and the loss of storage sites for Mg, with 60 % stored in bones and the remainder in skeletal muscles and soft tissues [8-10]. Because of invariable decreased thirst and endothelial injuries shown in experimental animals, there would be leaking through post-capillary venules with in turn elevations of angiotensin and C. In addition, with loss of skeletal muscles, for example a 40% loss of some leg muscles after just 5 months in Space-- an aging process accelerated by a factor of 10-- there would be a considerable loss of water storage sites in skeletal muscles [6,10,11]. With an invariable loss of Mg, the endothelium is vulnerable to injuries because of oxidative stress with deficiencies of transferrin shown with space flight [8]. Mg keeps iron - conducive to oxidative stress --- out of harm's way by binding iron tightly to transferrin [6,9,12]. In addition to the potential for hypertension by the combination of elevations of Cand angiotensin, and reductions of Mg required for the synthesis of transferrin, there is insulin resistance with insulin required for transferrin's synthesis as well [3,6,8]. It is of considerable interest that the difference between Neil Armstrong's stress test diastolic blood pressure after his historic lunar mission, in comparison to his resting diastolic blood pressure before Apollo 11, showed a marked difference of 50 mm Hg- far above the cutoff level of 15 mm Hg--- this is consistent with an abnormal stress test with a 100 % specificity [13]. Armstrong's lunar heart failure [11], is an example of C cardiomyopathy which is defined as acute temporary heart failure. With C levels twice the levels as those on Earth, Armstrong's case may be the first of many in the future-unless some way is found to prevent the invariable Mg deficits which I believe are responsible for most of the vascular problems I have described.

At this time, there is no subcutaneous replenishable device to administer Mg, Ca, other minerals as well as pharmaceuticals; the latter presents an additional problem --- in treating hypertension for example-because wit invariable complications triggered by endothelial dysfunction, there would be eventually, impairment in renal and hepatic function in order to excrete or metabolize pharmaceuticals.

Furthermore, some pharmaceuticals have been shown to deteriorate which may be instigated by radiation. In addition, there was a life-threatening exposure to highly toxic iron laden dust during the Apollo missions, brought into the lunar habitats on the space suits

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and inhaled while on the lunar surface and during the 3 day journey back to splashdown; note "historical documents" (http://www.femsinspace.com) Whereas Armstrong had only a 2 and a half hour walk on the moon, James Irwin had 3 lunar excursions in a buggy of 5-7 hours in duration. In addition, his space suit supply of water didn't function during his 3 EVA's and he had used an inadequate air conditioning setting with noon temperatures of 250 degrees F. With sweating there is a Mg loss, intensified by a renal loss. Also, his toxic dust inhalation was prolonged since ---after lift-off, his mission orbited the moon for 48 hours, before the 3 day journey home.

After Irwin's return to Earth, a bicycle stress test, showed a blood pressure of 275/125 after only 3 minutes of exercise. As an internist and cardiologist, in practice for 34 years, I have never seen blood pressures even approaching these levels- based on my experience of having supervised over 5000 symptom-limited, hospital-based maximum treadmill stress tests [14].

In addition, information I obtained a few years ago, through the Freedom of Information Act, showed that Irwin had, after his lunar return, stress test-cyanosis of the finger nail beds; similarly, I have never observed this. (See website historical documents) This trapping of Irwin's venous blood peripherally, supports my "Apollo 15 Space Syndrome" [15] manifested by severe space walk- fingertip pain; I postulated that this could be due to endothelial dysfunction with plasma fluid trapped distally; possibly intensified by this degree of hypertension, precipitated by the combination of space flight and highly toxic iron-laden dust. Irwin had a myocardial infarction, 21 months after his lunar mission. A contributing factor could well have been a single elevated blood pressure of 145/110 a month prior to his mission. Irwin suffered 3 more myocardial infarctions and died in 1991, 20 years after Apollo 15.

Using Irwin's case as an example, it begs the question as to whether we can apply this limited information regarding highly toxic lunar dust in formulating concepts as to the role of dust-laden urban pollution in contributing to hypertension [13,14]. What role does an iron content play on Earth? With space tourism, in the not too distant future, I am concerned that tourists will not inform those in charge, that they have hypertension or vascular disease. Even for a mere two hour flight to experience weightlessness at 63 miles, there is a risk during Earth reentry. In Irwin's autobiography [16] he describes his severe "shortness of breath" during reentry at 7 G forces; his dyspnea was of such severity that, while engaged in attempting to make a recording during the brief radio blackout, he was too dyspneic, to say one word. Although with space tourism, the reentry G forces would be predictably lower, perhaps a reentry force of 2-3, particularly with middle aged unfit tourists, there could be a fatality.

Since the thirst mechanism is impaired in Space, tourists would be at risk of significant dehydration, depending upon the duration, with in turn a greater risk of hypertension [6]. However in my "Neil Armstrong Syndrome" [17], Armstrong was so thirsty, he corrected his apparent dehydration during his 3 day journey home and thereby reduced his postulated very high levels of catecholamines.

The summary figure shows not only the vicious cycles between reduced Mg ion levels and high catecholamines. But how a Mg deficit can shorten the life span. Even though a serum Mg lacks sensitivity-with 99% intracellular-it was reduced significantly ($p < .0001$) in 196 space shuttle crewmembers [5]; similarly at the 11th. Man in Space Symposium, Toulouse, France in 143 cosmonauts. Mg is both a strong antioxidant and calcium blocker. Atrial natriuretic peptiderequires mg for its release and it can be reduced by 40% in only 7-12 days [5].

Finally, since there is no exposure to sun and with invariable mal absorption [7] there is a vitamin D deficiency [8]. But apparently the jury is still out as to whether correction of a vitamin D deficit will offset hypertension [18,19] and its effect in Space with the extraordinary vascular hazards. Despite invariable dehydration [6] there is a risk of hypertension particularly secondary to potential renal disease precipitated by angiotensin, C, endothelin elevations and vicious cycles with Mg ion deficits and ischemia. [4,17,20,21] Because of malabsorption [7], some method of administering vitamin D will be required.

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