Virtual conductance, real hypotension: what happens when we stand up too fast?

CARDIOVASCULAR PHYSIOLOGISTS HAVE long been curious about the mechanisms of fainting, and the goal of understanding why people faint under a variety of conditions has taken researchers to the ends of the Earth, and beyond. Some of these investigations have focused on adaptation to extreme environments such as microgravity, and the deleterious effects on orthostatic tolerance that are seen upon return to earth (1). At another end of the spectrum is work that has focused on the various pathophysiology that predispose “Earthbound” individuals to repeated syncopal attacks, and finding the means to alleviate these problems (5). Perhaps at the center of our collective interests lies the intrigue of studying a system, generally very robust and reliable, right at the moment that it happens to fail. Add to that, it is often those we believe to be most fit (6), or having the “right stuff,” who collapse from something as innocuous as “getting up too fast.” In our everyday experience, we are largely unaware of the extreme variation in gravitational force vectors and their pull on blood in compliant vessels, or the multitude of local vascular and reflex responses that together minimize the impact of these forces on the delivery of oxygenated blood to our brains to support our consciousness. When these responses fail, and blood pressure is compromised, the results can range from disconcerting signs and symptoms of cerebral hypoperfusion to dangerous loss of consciousness.

A particular set of circumstances that brings together many of these interesting ideas is what is known among pilots as the “push-pull effect,” a term that has its origins in the series of control-stick maneuvers that produce a steep dive and headward gravitational forces followed by a steep climb and footward forces, potentially leading to loss of consciousness. The study by Sheriff et al. (9) in the Journal of Applied Physiology provides an exquisite beat-by-beat picture of how these gravitational forces play out in the intact human volunteer when push precedes pull and provides insight into why the system sometimes fails when we stand up quickly.

Of fundamental importance to many such investigations is the tenet that the cardiovascular system plays by well-defined rules of fluid dynamics that are often boiled down to the hydraulic resistance equation: pressure equals the product of flow and conductance. Along these lines, Sheriff and colleagues (9) demonstrate that the marked fall in perfusion pressure to the brain that occurs when upright posture is preceded by a brief period of modest head-down tilt is due to an exaggerated rise in the rate of blood flowing into the legs. Recognizing that the hydraulic resistance equation was developed for steady-state conditions, the authors calculated a “virtual vascular conductance,” borrowing a concept from the muscle pump literature that they helped establish (10). Thus it becomes apparent from these new findings that a short-term headward gravitational force will modify the subsequent vascular response in the legs during the transition to footward forces. The nature of this exaggerated rise in virtual vascular conductance is unclear at present. It could be evidence of a myogenic vasodilation (i.e., a “real” change in conductance), activated by falling transmural pressure in leg vessels during head-down tilt, or it could be a rapid refilling of leg veins that were emptied during the “push” phase, leading to accelerated arterial inflow (i.e., a “virtual” change in conductance analogous to some muscle pump theory) (8). Whether the conductance is virtual or real, it leads to real hypotension at the level of the brain. Future studies will need to partition the active, or real, part of this vascular conductance from the purely mechanical, or virtual, component.

It is noteworthy that when humans move from supine to upright, there is a rapid rise in arterial inflow to the dependent limbs that is not initially matched by the volume of venous blood leaving the limbs. Because of this, the legs remain a key site of intervention via anti-shock/anti-G suits (3) and with physical maneuvers such as leg-crossing and muscle tensing (5) in the prevention of fainting responses. Put in this greater context, it is clear that changes in venous compliance of the legs with exercise training (7), microgravity (2), and other conditions may predispose some individuals to being more susceptible to the push-pull effect and related phenomenon, although demonstrating this connection has often been elusive (4).

The work by Sheriff and co-workers (9) also makes a compelling case that the pulmonary circulation plays a protective role when humans are exposed to the series of forces involved in the push-pull effect, such that cardiac output increases more in the upright position when the lungs have been “prefilled” with blood by slight head-down tilt. This highlights the fact that both the systemic and pulmonary circulations are compliant and, as a result, do not strictly adhere to a simple hydraulic resistance equation on a beat-by-beat basis. It also brings to mind the classic studies at the Mayo Clinic during World War II, in which Drs. Edward J. Baldes and Earl H. Wood developed a grunting maneuver for pilots that capitalized on this pulmonary reservoir to boost arterial pressure during high-G turns. This stands as yet another example of how forces external to the cardiovascular system can be used to produce virtual conductances with real impact.

REFERENCES


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