The vestibular system and cardiovascular responses to altered gravity

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CONSIDERABLE EVIDENCE from experiments conducted on animals (e.g., Refs. 1, 5, 6, 10) and human subjects (e.g., Refs. 4, 7–9, 11) demonstrates that inputs from otolith receptors in the inner ear participate in regulating blood pressure during movement and changes in posture. Otolith receptors detect linear acceleration of the head, including variations in head position with respect to gravity. Thus modifications in the gravitational forces acting on the head would be expected to result in altered cardiovascular responses as an animal or human undergoes movement. However, this premise has not been tested before experimentally. The study by Gotoh et al. (3) in this issue of the American Journal of Physiology-Regulatory, Integrative and Comparative Physiology demonstrated that removal of vestibular inputs alters the cardiovascular responses that occur when an animal is exposed to hypergravity. Transient exposure to hypergravity occurs during spaceflight, both during liftoff and landing; the findings of this manuscript are thus relevant to astronauts, who apparently require intact vestibular influences on the sympathetic nervous system to appropriately compensate for gravitational stress. In particular, the findings may be germane to cardiovascular deficiencies that occur during landing, as the vestibular system is highly plastic and adapts to the microgravity environment experienced during spaceflight (see Ref. 12 for review). It has previously been speculated that plastic alterations within the vestibular system during spaceflight may be partly responsible for the transient cardiovascular problems that many astronauts suffer on return to the Earth’s 1-G environment (12); the Gotoh et al. study provides further evidence that this conjecture is correct.

Prior experiments have also shown that the vestibulosympathetic reflex and the baroreceptor reflex interact (6, 7) and that common neuronal circuitry mediates both responses (13, 14). On the basis of this evidence, a logical hypothesis is that animals lacking both baroreceptor and vestibular inputs would have more difficulty in maintaining stable blood pressure than vestibular-intact animals that are baroreceptor denervated or baroreceptor-intact animals with vestibular lesions. However, this hypothesis has not been directly examined in the past. The Gotoh et al. study compared the cardiovascular responses to gravitational stress in sensory-intact animals with those lacking vestibular, baroreceptor, and both inputs. Interestingly, although animals with vestibular lesions (including those with neither baroreceptor nor vestibular inputs) exhibited significantly altered cardiovascular responses to gravitational stress, the most striking effects were in vestibular-intact animals lacking baroreceptor inputs; the latter animals displayed a large increase in blood pressure and renal sympathetic nerve activity during hypergravity exposure. The conclusion from these findings was that the vestibulosympathetic reflex acts to increase blood pressure during conditions that may produce orthostatic intolerance but is prevented from producing hypertension through the interactive effects of the baroreceptor reflex. This finding has implications for astronauts, who may experience alterations in the gain of both vestibulosympathetic and baroreceptor responses (2) during spaceflight. The combined effects of spaceflight on both reflexes may explain the significant alterations in orthostatic responses noted in some individuals immediately on return to Earth.

REFERENCES