Editorials

Orthostatic Hypotension in Earthlings and Space Travelers

HUMANITY'S first victory over the encumbrance of gravity occurred when humans evolved the capacity to maintain an upright posture. This capability was made possible by a major shift in the dominant function of the sympathetic nervous system. In the lower animals, the sympathetic nervous system had certainly been an important regulator of the circulation, but its most critical function was to mediate the "fight or flight" phenomenon. With the development of the upright posture, the sympathetic nervous system assumed a new burden, one that entailed a fundamental transformation of function. Henceforth, it would be inextricably linked to standing upright. In the comfortable life of modern humans, more norepinephrine is synthesized and released each day to maintain the upright posture than for any other purpose.

Humanity's second victory over gravity came with space travel, but it was only a partial victory. Of the many early concerns about the effects of weightlessness on human health, only a few have proved to be genuinely problematic. Orthostatic hypotension on return to Earth is operationally one of the most important of these. Although it resolves within days of returning to gravity, it can be a serious problem during shuttle reentry when gravitational force in a head-to-toe direction is transiently greater than 1*g*; loss of consciousness could occur under such circumstances, with serious consequences.

Orthostatic hypotension occurs when autonomic nervous system control over the heart and vasculature is overwhelmed or becomes dysfunctional.[1] In this issue of the journal, Alan S. Hollister, MD, PhD, provides a valuable guide to the many clinical causes of orthostatic hypotension.[2] Several points need to be kept in mind by physicians examining patients with symptoms that might be caused by orthostatic hypotension.

First, the diagnosis requires clinical vigilance and a willingness to take the extra time to obtain a blood pressure in the standing position as well as the supine or seated position. Because many patients with the Bradbury-Eggleston syndrome and the Shy-Drager syndrome have supine hypertension,[3] the erroneous—and, for these patients, dangerous—misdiagnosis of essential hypertension may be made. Obviously these patients will be harmed if they are given antihypertensive agents.

Second, the vasodepressor effect of food[4] can be used to help in diagnosing orthostatic hypotension; the diagnosis may be missed unless supine and upright blood pressures are measured in the hour following a high-carbohydrate meal. Before meals, many affected patients may not have a demonstrable fall in pressure with standing.

Third, medications are still the most common cause of symptomatic orthostatic hypotension.[3] The most insidious offender is amitriptyline, but many less commonly used tricyclic antidepressant agents have this effect. They are insidious because the orthostatic hypotension may not be noted in the first few days of therapy and therefore may not be recognized by the patient or physician as the actual cause of the clinical problem.

Finally, orthostatic hypotension is not synonymous with autonomic failure.[5] In an earlier era, adrenal failure was the most commonly recognized cause of orthostatic hypotension, and in the pivotal initial clinical description of autonomic failure, the critical issue of making this differential point occupied much of the attention of Bradbury and Eggleston.[6] Adrenal failure is a comparatively rare cause of orthostatic hypotension today. In mastocytosis, the mitral valve prolapse syndrome, and astronauts returning from space, however, it is unlikely that autonomic degeneration plays any actual role.[7]

The diversity of causes of orthostatic hypotension makes the physician's task difficult, but not impossible. By applying the approaches outlined by Hollister, the cause of most patients' orthostatic hypotension can be determined. Correct diagnosis often is the key to successful treatment.

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Has Our 'Healthy' Life-style Generated Eating Disorders?

AS ELLEN HALLER, MD, points out in her timely review article in this issue of the journal,[1] our young women, particularly those from reasonably well-to-do white families, have found themselves in the midst of an epidemic of eating disorders. In these patients we witness the collision of the cultural pressures on women, psychological vulnerabilities, and biologic factors that contribute to psychopathology and ill health. Research has documented social pressures on today's women to be thinner and to exercise more than was the standard in the 1940s and 1950s.[2] Although some attention to these matters is good, as with many other things, too much is not. As a result, surveys repeatedly show that about three quarters of women whose weights are fully in the normal range feel too fat and wish to lose weight; indeed, some studies have shown that they desire on average to weigh only slightly more than the weights in the anorexia nervosa range.[3] In only 1% to 3% do clinically significant anorexia nervosa and bulimia nervosa, develop, however. We do not understand why this small but far from insignificant subgroup has overt eating disorders, but current evidence suggests that anorexia nervosa or bulimia nervosa is more likely to develop in women with premorbid personalities and temperaments who tend to be particularly timid and avoidant, obsessional, or sensitive and moody and that women who have a greater tendency to be overweight may be more prone to have bulimia