Both Central and Peripheral Mechanisms Contribute to Post-spaceflight Orthostatic Hypotension

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This work was undertaken to determine the mechanisms of the low vascular resistance, both preflight and postflight, in astronauts who suffer from orthostatic hypotension and presyncope after short-duration spaceflight. METHODS: Twenty-two astronauts, nine of whom became presyncopal on landing day, were studied before flight, on landing day, and three days after landing. Hemodynamic measurements were recorded before and after intravenous injections of phenylephrine; hemodynamic measurements and plasma norepinephrine levels were measured before and after intravenous tyramine injections. Then tilt tests were performed to determine orthostatic tolerance, during which hemodynamic measurements, plasma catecholamines, and arginine vasopressin levels were measured. RESULTS: Before flight increases in systolic pressure and total peripheral resistance after phenylephrine injections were significantly greater in astronauts who would not become presyncopal on landing day than in those who would (P = 0.02 for systolic, P = 0.03 for resistance). On landing day, responses to phenylephrine were similar to those before flight in both groups, showing no effects of spaceflight. Before flight, norepinephrine release in response to tyramine injections was not different between groups. On landing day, norepinephrine release in response to tyramine was significantly greater than preflight in the presyncopal group (P < 0.01), but was not different in the non-presyncopal group. However, during upright tilt, norepinephrine release was not different from preflight in the presyncopal group, but was significantly greater than it had been preflight in the non-presyncopal group (P < 0.001). On landing day, upright plasma epinephrine levels were significantly higher in the presyncopal group than their own before flight (P < 0.001) and than those of the non-presyncopal group (P < 0.001). Similarly upright arginine vasopressin levels in the presyncopal group were significantly higher than their own preflight (P < 0.01) and than those of the non-presyncopal group (P < 0.001). CONCLUSIONS: Astronauts who are susceptible to postflight orthostatic hypotension and presyncope have a preflight vascular resistance that is lower than that of astronauts who are not susceptible to orthostatic hypotension. This low resistance is explained by lower α1-adrenergic receptor responsiveness. Furthermore, their low norepinephrine release on landing day is not related to decreased synthesis or storage of norepinephrine. In addition, release of both epinephrine and vasopressin are markedly increased in these astronauts on landing day. We suggest that central integration of baroreceptor input is disrupted by spaceflight in these subjects.