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Haemodynamic and baroreflex responses to whole-body tilting in exercising men before and after 6 weeks of bedrest

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Abstract We sought to determine whether the cardiovascular deconditioning that occurs in exercising men after prolonged (42 days) bedrest in the head-down tilt (HDT) position is primarily related to mechanical changes in the heart or to an impaired arterial-cardiac-chronotropic baroreflex. Seven subjects were studied before (C, control) and repeatedly after HDT with rapid tilting between the upright and supine positions during steady-state 50-W dynamic leg exercise. Ventricular interdependence was assumed to be an index of cardiac size; it was assessed on the basis of the initial dip of arterial pulse pressure (PP) induced by a sudden tilt from the upright to the supine position (down-tilt). Arterial-cardiac-chronotropic baroreflex sensitivity (ABS) was assessed as the ratio between tilt-induced heart rate transients and the preceding (and reciprocal) transient in arterial pressure. On the first day of recovery, the initial PP dip was –4 (2) mmHg (where 1 mmHg is 0.13 kPa), less than half of the control value; on subsequent recovery days, the initial PP dip was not significantly different from the control value. When tilting from the upright to the supine position, mean ABS ranged from 1.02 to 1.06 bpm/mmHg during three separate control sessions. Tilts in the opposite direction gave lower ABS values because of the more sluggish HR response and ranged from 0.43 to 0.45 bpm/mmHg in the control situations. ABS did not change after HDT. Our results indicate that impairments of the cardiovascular system after long-term bedrest are of haemodynamic rather than baroreflex origin.

Key words Heart function · Hypokinesia · Orthostasis · Simulated weightlessness · Tilting

Introduction

It is well established that most people who have been exposed to bedrest or spaceflight develop orthostatic intolerance, characterized among other things by a relative tachycardia in stand tests (Blomqvist and Stone 1983). Furthermore, it has been frequently observed that, for a given submaximal workload, cardiac output decreases and heart rate (HR) increases (Ferretti et al. 1998; Greenleaf et al. 1996; Saltin et al. 1968; Sundblad et al. 2000b). These findings suggest that the tachycardia is compensating for the reduced stroke volume caused by haemodynamic impairment, and appear to contradict the often-proposed notion that an impairment of the arterial-cardiac baroreflex contributes significantly to the orthostatic intolerance and cardiovascular deconditioning observed after bedrest and spaceflight. However, so far almost all of the studies of arterial baroreflex function after bedrest or spaceflight have been conducted with the subjects lying down and resting (Convertino et al. 1990, 1992; Crandall et al. 1994; Fritsch et al. 1992; Fritsch-Yelle et al. 1994; Hughson et al. 1994). The one exception is the study by Haruna et al. (1997), who investigated the vagal carotid-cardiac baroreflex and concluded that reflex sensitivity is reduced during rest while supine, but not during rest while sitting or during dynamic exercise. From a functional standpoint and in keeping with the approach of Haruna et al. (1997), it would seem more appropriate to study the arterial baroreflex in situations when it is actually needed, i.e., during physiologically stressful situations such as orthostatic stress and exercise.

In the present study, we sought to determine indices of both cardiac pumping function and arterial-cardiac baroreflex control by using methods that allow serial measurements to be made from exercising humans. We found no impairment of arterial-cardiac baroreflex function. On the other hand, signs of a reduced degree of ventricular interdependence on the first day of recovery after HDT suggest that the size of the heart had decreased.
Methods

Seven healthy male volunteers completed this study. Their mean (SE) age, mass, height, and maximal oxygen consumption were 28 (1) years, 73 (4) kg, 1.78 (0.01) m, and 2.83 (0.2) l·min⁻¹, respectively. The study was a part of a long-term bedrest study at the Institut de Médecine et de Physiologie Spatiales (MEDES) in Toulouse, France, organized by the European Space Agency and Centre National d’Études Spatiales. The experiments were approved by the local ethics committee (Comité Consultatif de Protection des Personnes dans la Recherche Biomédical, Toulouse, France).

Originally, eight subjects entered the study, but one withdrew because of persistent back pain. All subjects had a negative history of cardiovascular disease and were normal with respect to clinical examination, hemoglobin (Hb), arterial blood pressure, and the electrocardiogram (ECG).

The 42-day bedrest period was performed in a 6° head-down tilt (HDT) position. No deviations from the HDT position were permitted (video surveillance), and neither exercise nor muscle contraction tests were carried out during this period. Subjects were sedentary and confined to the hospital area for the 15 days immediately prior to, and the initial 13 days after, the HDT period, with an occasional brief stroll permitted. The experiments were conducted in conjunction with a recently published study (Sundblad et al. 2000b).

Protocol

Pre-HDT control experiments were performed 14 (C1), 11 (C2), and 6 (C3) days before the HDT period. No measurements were made during the HDT bedrest period. Experiments after the HDT period were conducted in the afternoon of the same day that the HDT period ended (R + 0; 8 h after rising from the bed). A medical examination, including a stand test and a lower body negative pressure (LBNP) test, was conducted during this 8-h period. Further recovery experiments were conducted 2 (R + 2), 4 (R + 4), 8 (R + 8), 12 (R + 12), and 32 (R + 32) days after the HDT period.

Experimental procedures

The subjects exercised on a tilt-board on which an ergometer was mounted with the crank axis at the head level when the subjects were supine (Fig. 1). The tilt-board could be tilted rapidly in 2 s from an 80° upright position to the horizontal (0°) position, or the other way. The exercise workload was 50 W. Subjects started in the supine position and, after a warm-up period of 6 min, each subject was tilted without prior notice to the upright position for 3 min and then back to being supine for 3 min. This procedure was repeated three times. Throughout this report, a tilt from the upright to the supine position is designated a “down-tilt” and a tilt from the supine to the upright position is termed an “up-tilt”.

Cardiovascular measurements

Heart rate (HR) was calculated beat-by-beat with an analogue HR meter (ECG: Biolchamp amplifier, model 20-4615-65, Gould, Valley View, Ohio, USA), and the arterial blood pressure (AP) in a finger was recorded with a plethysmographic device (Finapres, Type 2300, Ohmeda, Englewood, Colo., USA). AP readings were compensated for the hydrostatic pressure difference between the finger and the carotid sinus level by measuring the pressure in a water-filled catheter attached to the neck and the Finapres cuff. The AP at heart level was calculated sample-by-sample using an algorithm whose input variables are tilt angle, and the distances between the Finapres cuff, the heart, and the carotid sinus level.

Continuous recordings of HR, ECG, AP, and hydrostatic compensation pressure were recorded at a sampling rate of 100 Hz per channel by a digital data recording system (ASYST, Keithley, Taunton, Mass., USA). Offline data reduction included several steps using the same system. The on-line analogue beat-by-beat HR signal consisted of a series of levels, with each level being triggered by the R-peak in the ECG. The level represented the inverse of the duration of the previous R–R interval. Off-line, each beat-by-beat HR level was relocated back to the R–R interval from which it derived. For each heart beat, the systolic maximum and the diastolic minimum pressures were identified. The level of systolic arterial pressure (SAP) was stored for the duration of the heart beat, i.e., between the times of successive diastolic minima. Diastolic arterial pressure (DAP) curves were created by linear interpolation between the above-mentioned diastolic minima. Pulse pressure (PP) was defined as SAP minus DAP. Mean arterial pressure (MAP) was computed as the time-average of the arterial pulse wave between successive R-peaks.

Absolute PP values cannot be determined accurately from arteries in the periphery because of pulse wave amplification (Rowell et al. 1968). On the other hand, the Finapres gives reasonable measures of MAP and DAP (Idema et al. 1989). Our rationale for evaluating PP by estimating it peripherally is that we sought to analyze only the changes in pressure that were induced by rapidly tilting a single individual. The MAP at each instant at a hypothetical baroreceptor level (arterial receptor pressure, ARP) was computed as the arithmetic mean of the MAP at the heart and carotid levels. In this calculation, it is assumed that the arterial baroreflex inputs from aortic and carotid sites are equally important.

To reduce random noise and to enhance responses to tilting, recordings from repeated, identical sequences were averaged sample-by-sample. To make sure that the different data sets were aligned properly, they were matched at the time when the subjects were positioned at 30° tilt. The parameters for each subject were derived from the average of three recordings of identical sequences (Fig. 2). In general, down-tilts induce transient peaks in the pressure parameters, whereas up-tilts induce transient nadirs. These fluctuations in arterial pressure induce reciprocal changes in HR, which are mediated via the baroreflex. The mechanisms behind these fluctuations are discussed in detail elsewhere (Sundblad et al. 2000a).

Steady-state values were taken as the mean of a 30-s segment recorded 120 s following the tilt. The amplitudes of the peaks and

Fig. 1 Schematic of the tilt-board, which was tilted manually every 3 min between the upright (80°) and supine (0°) positions. Each tilt was completed within 2 s and was conducted during steady-state dynamic leg exercise.