Arterial pressure in humans during weightlessness induced by parabolic flights

BETTINA PUMP, REGITZE VIDEBAEK, ANDERS GABRIELSEN, AND PETER NORSK

Pump, Bettina, Regitze Videbæk, Anders Gabrielsen, and Peter Norsk. Arterial pressure in humans during weightlessness induced by parabolic flights. J. Appl. Physiol. 87(3): 928–932, 1999.—Results from our laboratory have indicated that, compared with those of the 1-G supine (Sup) position, left atrial diameter (LAD) and transmural central venous pressure increase in humans during weightlessness (0 G) induced by parabolic flights (R. Videbæk and P. Norsk. J. Appl. Physiol. 83: 1862–1866, 1997). Therefore, because cardiopulmonary low-pressure receptors are stimulated during 0 G, the hypothesis was tested that mean arterial pressure (MAP) in humans decreases during 0 G to values below those of the 1-G Sup condition. When the subjects were Sup, 0 G induced a decrease in MAP from 93 ± 4 to 88 ± 4 mmHg (P < 0.001), and LAD increased from 30 ± 1 to 33 ± 1 mm (P < 0.001). In the seated position, MAP also decreased from 93 ± 6 to 87 ± 5 mmHg (P < 0.01) and LAD increased from 28 ± 1 to 32 ± 1 mm (P < 0.001). During 1-G conditions with subjects in the horizontal left lateral position, LAD increased compared with that of Sup (P < 0.001) with no further effects of 0 G. In conclusion, MAP decreases during short-term weightlessness to below that of 1-G Sup simultaneously with an increase in LAD. Therefore, distension of the heart and associated central vessels during 0 G might induce the hypotensive effects through peripheral vasodilatation. Furthermore, the left lateral position in humans could constitute a simulation model of weightlessness.

ON THE BASIS OF RESULTS of ground-based simulation studies and of spaceflights, it is generally accepted that central blood volume increases and the heart is distended during the initial phase of weightlessness (1, 4, 8). This notion has recently been confirmed by us during parabolic flights, where left atrial diameter (LAD) and transmural central venous pressure increased during weightlessness in humans to values even above those of the 1-G supine position (12). Simultaneously, intrathoracic pressure decreased (12). Thus the decrease in intrathoracic pressure during weightlessness apparently augments the filling of the heart, resulting in left atrial distension (12, 13).

The increased transmural filling pressures of the heart during weightlessness should result in stimulation of cardiopulmonary low-pressure receptors. If this is the case, it would be expected that peripheral vasodilatation is initiated to maintain or decrease mean arterial pressure (MAP). Because intrathoracic pressure also decreases, this could per se also lead to an increase in central transmural (e.g., aortic/cardiac) pressures, resulting in additional vasodilatation and a decrease in peripheral MAP.

We tested the hypothesis that MAP is decreased by short-term weightlessness to values below those in a 1-G supine position. Furthermore, we compared the effects of weightlessness with the effects of the 1-G horizontal, left lateral position, because we have previously observed that the latter position increases LAD (personal observations). Therefore, the 1-G horizontal, left lateral position might simulate the immediate effects of weightlessness on cardiovascular variables in humans.

MATERIALS AND METHODS

After giving their informed consent, 6 men and 2 women (mean age 35 yr [range: 26–53 yr], height 176 cm [range: 165–189 cm], and weight 72 kg [range: 62–85 kg]) participated in the experiment. Two additional subjects entered the study, but their participation was cancelled, because it was technically impossible to obtain an adequate blood pressure signal (see below). All were healthy, as indicated by a physical examination, hematocrit, electrocardiogram, chest X-ray, and arterial pressures (<140/90 mmHg). The protocol was approved by the Medical Board of the European Space Agency and considered acceptable and in compliance with the declaration of Helsinki by the Ethics Committee of Copenhagen. No complications occurred.

The study was carried out at Bordeaux airport (France) in connection with a European Space Agency parabolic flight campaign in October 1998. The parabolic maneuvers were performed in a specially modified Airbus (A300) on 3 consecutive days with 30 parabolas and 3–4 subjects investigated each day. A parabola consisted of three phases, each lasting 20 s: pull-up, with the G load gradually increased to 1.8 (1.8-Gpre), followed by a sudden onset of weightlessness (0 G), and finally pull-out, with the G load up to 1.8 (1.8-Gpost). Subsequently, the G would return to 1 (1-Gpost). During weightlessness, the G level was 0.00 ± 0.02, except during five parabolas with fluctuations up to ±0.07 G. Because the subjects were free of the floor during 0 G, the effects of these fluctuations were negligible. During the 1-G periods, the maximum variation was ±0.1 G, but usually ~0.05 G.

The subjects ingested a light meal (no coffee) some 2.5 h or more before the flight and were thereafter fasting. They were not allowed to take any medication against motion sickness. Five of the subjects had previously participated in parabolic flights, and none of the subjects exhibited symptoms of motion sickness such as paleness, nausea, or sweating at the time of investigation except for one, at the end of a parabolic maneuver. Data from this maneuver are therefore not presented, and the experiment was discontinued in this subject.

The subjects were investigated during six consecutive parabolas in three different postures: supine (Sup), seated with legs horizontal (Seat), and the horizontal, left lateral
position (Lat). During Seat, the subject was supported by a vertical board. Straps around the waist and legs prevented the subject from floating in an uncontrolled manner. The sequence of Seat and Sup was randomized among the subjects in a balanced fashion, whereas measurements with the subjects placed in Lat always were conducted thereafter. The measurements were performed during 20 s of 1 G immediately before the parabolic maneuver, throughout the three phases, and finally for 20 s thereafter, where the G level had returned to 1 (Fig. 1).

MAP and heart rate (HR) were recorded continuously by an infrared photoplethysmograph (Finapres, 2300 Finapres, Ohmeda) in the right index finger, with the pressure signal stored in a computer (Labview) for later analysis. Occasionally, it was not possible to detect an arterial pressure signal in some of the subjects. Therefore, n < 8 with regard to some of the data. MAP was determined as the electronic mean, and all values are presented as means of 17–20 s of stable G within each parabolic phase. Special care was taken to ensure that the cuff around the index finger was kept level with the fourth intercostal space when the subjects were Seat by keeping the hand in an adjustable sling, and at level with the midaxillary line and midsternum, respectively, when they were Sup and Lat.

LAD was estimated for 20-s periods before, during, and after the parabolas by echocardiography (Aloka SSD 500, Simonsen & Weel) by using the parasternal long-axis view. The position of the transducer was adjusted by watching two-dimensional and M-mode pictures on a screen. Pictures were stored on tape by a video recorder (Sony SVO-9500 MDP), and LAD was determined according to the criteria of Feigenbaum (3) as an average of measurements from three M-mode printouts. All the measurements were performed in a blinded fashion by the same observer.

Ambient temperature was kept between 20.1 and 25.5°C, humidity between 19 and 58%, and pressure between 630 and 700 mmHg. Pressure varied \( \pm 1.5 \) mmHg during the parabolic maneuvers.

Data are presented as means \( \pm SE \) and as means of two parabolic maneuvers with the subject in the same position for each subject. ANOVA (Statgraphics plus for Windows, version 3.0) for repeated measures, with the variable as main variate and time and subject as factors, was used to evaluate the effects of the G load on a variable within each series of experiment. Differences between mean values were evaluated by a post hoc multiple-range test (Newman-Keuls). \( P < 0.05 \) was chosen as the level of significance.

**RESULTS**

With the subjects in Sup, MAP decreased during 0 G compared with that of 1 G from 93 \( \pm 4 \) to 88 \( \pm 4 \) mmHg (\( P < 0.001 \), Fig. 2). When the subjects were Seat, MAP decreased during 0 G from 93 \( \pm 6 \) to 87 \( \pm 5 \) mmHg (\( P < 0.01 \)). MAP was unaffected by the 1.8 G before 0 G. There were no effects of changes in G load when the subjects were placed in Lat (Fig. 2). No significant differences could be detected comparing MAP values of Seat, Sup, and Lat under 1- and 0-G conditions, respectively. During 1.8-Gpost when the subjects were Seat, however, MAP increased compared with that of Lat (\( P < 0.05 \), Fig. 2).

HR increased from 77 \( \pm 5 \) to 92 \( \pm 7 \) beats/min during 1.8-Gpre of Seat and decreased again to 76 \( \pm 8 \) beats/min during 0 G (\( P < 0.005 \)). During 1.8-Gpost, HR tended to increase although insignificantly. In contrast to this, HR was unchanged during any of the G levels when the subjects were Sup or Lat (Fig. 2). During 1 and 0 G, respectively, HR of Seat, Sup, and Lat subjects did not differ significantly (Fig. 2).

During Seat and Sup, LAD was very sensitive to the changes in G (\( P < 0.001 \), Fig. 2). LAD decreased during Sup from 30 \( \pm 1 \) to 28 \( \pm 1 \) mm during 1.8 G and increased to 33 \( \pm 1 \) mm during 0 G. The same pattern was observed during Seat, with values 2–3 mm lower.
There were no significant effects of changes in G stress on LAD when the subjects attained Lat. Lat per se induced a 4 to 6-mm increase in LAD compared with those for Sup during 1 and 1.8 G (P < 0.001). It is noteworthy that LAD attained similar values during 0 G in all three of the positions (Fig. 2).

**DISCUSSION**

The results of this study demonstrate that MAP in humans is decreased by 20 s of weightlessness induced by the parabolic flights to below 1-G Sup. Simultaneously, the left atrium is distended and HR is unchanged. Therefore, the decrease in MAP in supine subjects during 0 G of parabolic flights could have been caused by peripheral vasodilatation induced by stimulation of cardiopulmonary low-pressure receptors. Because we have previously observed that intrathoracic (esophageal) pressure also decreases during 0 G when subjects were Sup (12), it is likely that a combination of increased venous return and decrease in intrathoracic pressure accounts for stimulation of the cardiopulmonary low-pressure receptors. It is possible that the arterial baroreceptors were also stimulated during 0 G compared with 1-G Sup, caused by the decrease in intrathoracic pressure. This possibility is uncertain, however, because HR was unchanged throughout the parabolic maneuver, when the subjects were Sup.

It is noteworthy that LAD during 0 G increased to a value above that of 1-G Sup and similar to that of 1-G Lat. Furthermore, there were no effects on MAP in Lat. Therefore, Lat might constitute a 1-G simulation model to mimic the immediate effects of weightlessness on cardiovascular variables in humans.

Like other investigators (6, 11), we observed a decrease in MAP in seated subjects during weightlessness induced by the parabolic maneuvers. The mechanisms of the hypotensive effects of 0 G with the subjects Seat, probably constituted stimulation of cardiopulmonary and carotid baroreceptors. Because LAD increased during 0 G, and because we have previously observed an increased transmural central venous pressure during weightlessness (9, 12), we are convinced that cardiopulmonary low-pressure receptors were stimulated. Furthermore, the carotid receptors must have been stimulated, because the hydrostatic pressure gradient between the heart and the carotid sinus of a seated subject is abruptly eliminated on entrance into 0 G. Therefore, we think that the mechanism of the decrease in MAP in 1-G Seat compared with 0 G is very similar to that of a decrease induced by a simple 1-G posture change from upright seated to supine (10). This notion, however, is still speculative and needs to be experimentally verified.

The mechanisms of the decrease in MAP during 0 G when subjects were Sup probably involved a combination of cardiopulmonary low-pressure stimulation and the mechanical effects of a decrease in intrathoracic (intrapleural) pressure. Stimulation of low-pressure reflexes could have induced peripheral vasodilatation, with a resultant decrease in MAP. In addition, previous results from parabolic flights have shown that intrathoracic pressure decreases during 0 G compared with 1-G Sup (12). Results in a study by Ikeda et al. (5), who used continuous negative pressure breathing in supine subjects, indicate that a negative pressure in the thorax induces a decrease in MAP. Thus a decrease in intrathoracic pressure could probably mechanically induce a decrease in MAP. Therefore, the decrease in MAP when subjects were Sup could have been caused by a combination of a decrease in intrathoracic pressure per se and cardiopulmonary low-pressure-receptor stimulation.

Fig. 2. MAP (A), left atrial diameter (LAD; B), and heart rate (HR; C) during 20 s of 1, 1.8, 0, 1.8, and 1 G. Subjects were investigated in Seat (○), Sup (●), and Lat (■). Values are means ± SE of n = 8 for LAD in Seat and Sup, n = 7 for MAP and HR in Seat and Sup, and n = 6 for Lat. *Significantly different compared with 1-G values before parabolic maneuver (P < 0.05). ** Significant difference between indicated sessions (P < 0.05).
Because of the augmented hydrostatic pressure differences during the 1.8-G periods, causing an unloading of baroreceptors in subjects during Seat, the effects of hyper-G on HR were more pronounced in this posture, where HR increased, than during Sup, where HR was unchanged. This is in accordance with results from Lathers et al. (7). During the 0-G phase, when the subjects were in Seat, HR decreased 15 beats/min back to the 1-G level and did not decrease further, probably because the period of 20 s was too short. It is very likely that the G-induced changes in HR were mainly elicited by changes in stimulation of carotid baroreceptors.

It is noteworthy that because HR in subjects in Sup was unchanged during 0 G, whereas MAP decreased, the hypotensive effects of 0 G must primarily have been induced by peripheral vasodilatation. Therefore, it is likely that efferent sympathetic nervous activity during 0 G was decreased to below the level for 1 G, inducing the peripheral vasodilatation. This notion needs to be further evaluated, e.g., by microneurographic recordings in supine subjects during parabolic flights.

The present results confirm that the heart is distended during 0 G (12). During Seat and Sup, LAD decreased during 1.8 G and increased to the level above that for 1 G during weightlessness. However, LAD did not change during parabolic maneuvers, when subjects were in Lat. Furthermore, MAP and HR were unchanged by the changes in G stress in this posture. Lat may therefore constitute a model for simulating the immediate effects of weightlessness on cardiovascular variables.

It is noteworthy from a methodological point of view that LAD measured in one plane by echocardiography did not change during 1.8 and 0 G, respectively, when the subjects were in Lat. This observation indicates that the increase in LAD when the subjects were in 1-G Lat, compared with 1-G Sup, is not caused by an asymmetrical change in the shape of the left atrium.

Limitations in interpreting the results. With parabolic flights, it is only possible to investigate the immediate effects of changes in G, because each phase only lasts 20 s. It is not known whether longer periods of 0 G would reveal a more pronounced decrease in MAP or whether MAP gradually would return to the level in 1 G. Furthermore, when the effects of 1 G are compared with those of weightlessness, it is important to consider the effects of hyper-G in between.

When measuring arterial pressures, we made the assumption that 1) the fourth intercostal space in Seat, 2) the midaxillary line in Sup, and 3) the midsternal level in Lat would reflect the same anatomic reference points in relation to the heart and central vessels. This assumption might have caused minor errors in comparing MAP of the different postures during 1 and 1.8 G, respectively. Furthermore, it could be argued that the changes in MAP from 1 to 0 G were purely hydrostatically induced, because, i.e., during 1-G Sup, the finger with the Finapres cuff could have been placed below the correct reference point. This is very unlikely, however, because no changes in MAP occurred during 1.8 G, where the hydrostatic pressure differences, if present, would have induced an increase in MAP.

Besides high- and low-pressure receptors, vestibular stimulation might also theoretically have affected the regulation of MAP during the parabolic maneuvers. Cui et al. (2) observed that caloric vestibular stimulation can affect muscle sympathetic nervous activity. These authors, however, did not observe concomitant changes in arterial pressures. Thus it remains to be determined whether vestibular stimulation during parabolic maneuvers participates in the cardiovascular responses to 0 G.

In conclusion, MAP decreases during short-term weightlessness induced by parabolic flights to below that of 1-G Sup, and LAD increases simultaneously. It is likely that distension of the heart chambers and associated central vessels during 0 G through stimulation of cardiopulmonary reflexes induces a decrease in MAP by peripheral vasodilatation. Stimulation of the cardiopulmonary receptors during 0 G is probably caused by 1) a decrease in intrathoracic (intrapleural) pressure, with a resultant increase in transmural central venous pressure (12); and 2) an increased venous return due to less compression of the peripheral tissues and of organs on the central veins. Furthermore, the size of the left atrium in the 1-G Lat position is similar to that in weightlessness. Thus Lat might constitute a 1-G simulation model of the immediate effects of weightlessness on cardiovascular variables in humans.

Perspectives. The results of this study demonstrate that the transverse gravitational stress (+1 Gx) in humans affects central cardiovascular variables, because MAP decreases and LAD increases in subjects while Sup when G load is changed from 1 to 0. The effect of +1 Gx on cardiac filling pressures and MAP could be due to compression of the heart by its own weight and by the weight of the lungs and thoracic cage. This compression might limit venous return and result in a diminished cardiac output. In addition, it is noteworthy that LAD and MAP were unaffected by the change in gravitational stress when the subjects were placed in Lat, whereas LAD was elevated compared with when they were Sup. Therefore, we suggest that these observations be taken into account in the positioning of patients with heart failure, for example.

Furthermore, we observed that Lat mimics the effects of weightlessness on MAP, HR, and LAD. This position might, therefore, be a better way of simulating the effects of weightlessness than, for example, the supine or head-down bed-rest position. Therefore, in the future, a comparison between the cardiovascular effects of Lat and bed rest should be performed to optimize the 1-G simulations of the effects of weightlessness on cardiovascular variables in humans.

The support of the European Space Agency and of the French company Novespace, in particular Thierry Gharib, is acknowledged with gratitude. Furthermore, the technical assistance of Paul Bruun and Brian Støvhase Nielsen are gratefully acknowledged.

This experiment was supported by Danish Research Councils Grant 9602455.
REFERENCES


