Effects of gravity on lung diffusing capacity and cardiac output in prone and supine humans

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Submitted 16 December 2002; accepted in final form 7 March 2003

Rohdin, M., J. Petersson, P. Sundblad, M. Mure, R. W. Glenny, S. G. E. Lindahl, and D. Linnarsson. Effects of gravity on lung diffusing capacity and cardiac output in prone and supine humans. J Appl Physiol 95: 3–10, 2003; 10.1152/japplphysiol.01154.2002.—Both in normal subjects exposed to hypergravity and in patients with acute respiratory distress syndrome (ARDS), there are increased hydrostatic pressure gradients down the lung. Also, both conditions show an impaired arterial oxygenation, which is less severe in the prone than in the supine posture. The aim of this study was to use hypergravity to further investigate the mechanisms behind the differences in arterial oxygenation between the prone and the supine posture. Ten healthy subjects were studied in a human centrifuge while exposed to 1 and 5 times normal gravity (1 G, 5 G) in the anterioposterior (supine) and posterioanterior (prone) direction. They performed one rebreathing maneuver after ~5 min at each G level and posture. Lung diffusing capacity decreased in hypergravity compared with 1 G (ANOVA, P = 0.002); it decreased by 46% in the supine posture compared with 25% in the prone (P = 0.01 for supine vs. prone). At the same time, functional residual capacity decreased by 33 and 23%, respectively (P < 0.001 for supine vs. prone), and cardiac output by 40 and 31% (P = 0.007 for supine vs. prone), despite an increase in heart rate of 16 and 28% (P < 0.001 for supine vs. prone), respectively. The finding of a more impaired diffusing capacity in the supine posture compared with the prone at 5 G supports our previous observations of more severe arterial hypoxemia in the supine posture during hypergravity. A reduced pulmonary-capillary blood flow and a reduced estimated alveolar volume can explain most of the reduction in diffusing capacity when supine.

LUNG ELASTICITY RENDERS THE distributions of gas, blood, and tissue influenced by gravitational forces. Thus the increased weight due to tissue and alveolar edema in acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) causes the lung to collapse under its own weight (the “sponge model”) (7, 14, 29). These patients show a better arterial oxygenation and less impaired alveolar-to-arterial O2 transfer in the prone (face-down) than in the supine (face-up) posture (9–11, 16, 22, 24, 30). The exact mechanisms by which this improvement of arterial oxygenation occurs have not yet been determined (23). We recently showed that the improved gas exchange effect of prone positioning also exists in healthy subjects using a model of acute lung insufficiency with gravity (G)-induced ventilation-perfusion mismatch (35). Common denominators between the patients and the hypergravity condition are an increased hydrostatic pressure gradient down the lung (14, 37, 38) and an increased weight of the heart (18, 46). The aim of the present study was to complement our previous data on arterial oxygenation with data on lung blood flow, lung volume, and lung diffusing capacity (DLCO).

Even though the recumbent position improves overall tolerance to hypergravity in terms of signs of cerebral hypoperfusion (45), we reasoned that, not only lung function, but also central circulation would be severely affected by hypergravity in the horizontal posture, even though there is a somewhat lower hydrostatic distance from the dependent to the nondependent parts of the thorax in the horizontal compared with upright posture. Furthermore, we hypothesized that the hypergravity-induced decrease in DLCO over the alveolar membrane would be more pronounced in the supine than in the prone posture, which is analogous to our laboratory’s previous findings with alveolar-to-arterial O2 transfer (35). Finally, we hypothesized that, if DLCO were to be more impaired by hypergravity in the supine than in the prone posture, this would be accompanied by impairments of its major determinants, such as alveolar volume (VA) (36, 43) and homogeneity of pulmonary-capillary blood volume distribution (31).

MATERIALS AND METHODS

Subjects. Eight men and two women were studied. Their ages, heights, and body masses ranged from 21 to 29 yr, 165

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to 191 cm, and 55 to 92 kg, respectively. They had no history of cardiopulmonary disease and were not taking medications at the time. They were also instructed not to drink coffee or use nicotine-containing products on the day of the experiment. The subjects received written information about the procedure, and informed verbal consent was obtained. The experimental protocol used in the present study was approved by the Ethics Committee of Karolinska Institutet. In a previous paper, our laboratory (35) has reported data on alveoloarterial oxygen differences in prone and supine subjects exposed to hypergravity. There was an overlap of 6 out of 10 subjects compared with the present study, and the experiments were performed on separate occasions. The limited overlap does not permit direct statistical comparison between the two studies, and, therefore, the results are reported separately.

Equipment and measurements. The experiments were conducted in the human centrifuge at Karolinska Institutet, Stockholm, Sweden. The centrifuge has two arms: one with a gondola, and one with a platform. A support structure was mounted on this platform. The subject was placed on a padded support surface that could be adjusted to be perpendicular to the resultant of the normal-G and the centrifugal-G vectors. The subject was secured on the surface by a five-point safety belt. The head and torso of the subject were fixed-G vectors. The subject was secured on the platform of the centrifuge and breathed air through the mouthpiece. Approximately 4.5 min after reaching the desired G level, the subject performed one rebreathing maneuver: after an expiration to functional residual capacity (FRC), the subject rebreathed the full bag volume eight times at a rate corresponding to 3 s/breath. The gas mixture used for rebreathing contained 35% oxygen (O₂), 5% argon (Ar), 3% sulfur hexafluoride (SF₆), 5% helium (He), 0.63% acetylene (C₂H₂), 0.5% carbon monoxide (CO), and balance of nitrogen (N₂). CO with the stable isotope ¹⁸O (molecular weight 30) was chosen to permit analysis of CO in the presence of N₂ (molecular weight 28). The rebreathing gas volume varied from 1 to 2 liters, depending on the stature and the preference of the subject. The subject was instructed to just empty the bag on each inspiration. The rebreathing maneuver was separated by at least 10 min to permit elimination of test gases. At least 8 of these 10 min were at normal G. Each rebreathing maneuver ended with a slow (~0.5 l/s) exhalation to residual volume, where gas tracings were analyzed for phase IV phenomena, which, if present, would indicate poor intrapulmonary gas mixing at the end of the rebreathing (34).

Data analysis. Offline data analysis was performed with an Acknowledge 3.2 Biopac digital data-handling system (Biopac). Offline computations included algorithms for total dry pressure correction (42) and computation of calibrated values for all dry-gas fractional concentrations. Also, concentration readings were corrected for the response latency of the mass spectrometer system (5), and gas volumes and flows were converted to STPD (standard temperature and pressure, dry) or FToS (body temperature, ambient pressure, saturated with water vapor), when appropriate.

During the rebreathing maneuver, Dₗcₒ₂ was calculated from the rate of uptake of C¹⁸O, and the cardiac output (Qₑ) was calculated as being proportional to the uptake of C₂H₂ (13, 39–41). Because Dₗcₒ₂ depends on the Vₐ at which the determination is made (36, 43, 44), and because FRC and thereby Vₐ differed between conditions, a method had to be devised by which Dₗcₒ₂ data obtained with different Vₐ values could be compared. Clearly, the mean Vₐ during a rebreathing maneuver must be somewhere between FRC (the volume when connected to the bag) and FRC plus the bag volume, with the latter being the lung volume after inhalation of the bag content. Thus Dₗcₒ₂ was corrected for differences in Vₐ between conditions, as described by Montmerle et al. (20); algorithms from Stam et al. (44) were used to recalculate Dₗcₒ₂ if the Vₐ was 50% of total lung capacity in the supine posture at 1 G. Vₐ was estimated as FRC + ½ rebreathing bag volume, and total lung capacity as vital capacity + residual volume, which was computed from anthropometric data, according to Quanjer et al. (32). The residual volume appears to be unaffected by the size and the axis of acceleration (15).

The intercept of the CO regression line with the initially inspired CO level was used to define the instant when the inhaled gas mixture reached the alveoli, i.e., the onset of alveolo-capillary exchange of the inhaled foreign gases or time₀ (40). Lung tissue volume was estimated as described by Sackner et al. (40) from the intercept of the back-extrapolated C₂H₂ disappearance curve with the ordinate at time₀. FRC was...
calculated from the dilution of the insoluble gas Ar in the lung-bag system volume, where all Ar readings were offset by the Ar concentration in atmospheric air. Oxygen uptake was calculated from the linear slope of end-tidal O2 values during rebreathing and the lung-bag system volume (6).

Statistical techniques. ANOVA (STATISTICA 5.0, Statsoft, Tulsa, OK) with repeated-measures design with two dependent factors [G (in the anterior-posterior or the posterior-anterior direction) and posture] was used to test for differences between changing G levels and posture and interaction between these parameters. Results were considered statistically significant if P < 0.05, and all tests were two-sided. Data are presented as means ± SE, unless otherwise stated.

RESULTS

All 10 subjects completed the experiments. However, subjects generally experienced recumbent exposure to 5 G very stressful, and some had difficulty following the required even rebreathing pattern exactly. Therefore, the number of data points is <10 for some of the parameters (Table 1). This is because some parameters were more sensitive to deviations from the ideal rebreathing pattern than others. FRC was relatively easy to define with confidence, because 1–5% Ar had the most favorable signal-to-noise ratio of the tracer gases, and identifying an equilibrium level would have been simple, even with a noisy signal. In contrast, C18O and C2H2 were detected at maximal levels of 0.3 and 0.6%, respectively, with proportionally less favorable signal-to-noise ratios compared with those of Ar. Moreover, computations of DLCO and Q1 included the fitting of a linear slope to end-tidal points on a log-linear scale, where the constancy of the breathing pattern is much more critical. One way to avoid such problems would have been to make repetitions of the rebreathing maneuver at each G level and posture, but one 5-G exposure in each posture was enough for the majority of the subjects. The present experimental design did not permit determination of reproducibility of rebreathing test parameters. In a previous study with sitting subjects (34), duplicate determinations of DLCO had a coefficient of variation of 9% at 3 G. The corresponding value for Q1 was 8%.

No end-expiratory concentration deviations (phase IV phenomena) were identified in any of the subjects. Cardiopulmonary variables are shown in Table 1. Generally, there were marked effects of G and/or posture for most of the variables, but we found no interactions between G and posture for any of the variables, except for arterial oxygen saturation (Table 1).

Lung function. Hypergravity reduced DLCO compared with normogravity in both postures (P = 0.002), and DLCO was lower in the supine posture than in the prone (P = 0.01). At 5 G, DLCO was significantly lower in supine than in prone (Fig. 1). There was a tendency for interaction between posture and G for DLCO (P = 0.07), i.e., a more manifest hypergravity-induced decrease in DLCO in supine posture compared with prone.

Arterial oxygen saturation at hypergravity decreased by 5.5 ± 0.5% units (mean ± SE) in the prone posture compared with 1 G and by 10.0 ± 1.4% units in the supine. The hypergravity-induced impairments of arterial saturation were significantly larger in the supine posture (P = 0.002). FRC decreased in hypergravity (P < 0.001) but was significantly larger in the prone posture compared with supine at both G levels (P <
Differences between postures (P < 0.001): 8% larger at 1 G and 26% at 5 G, respectively. Estimated VA decreased in hypergravity (P < 0.001), but was significantly larger in the prone posture compared with supine at both G levels (P < 0.001). There was a tendency for a hypergravity-induced difference in oxygen uptake (P = 0.09 for the difference between G levels), with a tendency to increase in the prone but not in the supine posture (P = 0.05 for prone vs. supine). Oxygen uptake was 17% higher in the prone posture than in supine at 5 G, and there was a tendency for interaction between posture and G (P = 0.07). There was no difference in lung tissue volume between postures (P = 0.2) and no difference between G levels (P = 1.0). A surrogate for arteriovenous O2 difference, computed as O2 uptake-to-Ql ratio, did not differ between postures at any G level (P = 0.8), but in both postures together it was 80% larger at 5 G compared with 1-G control (P < 0.001).

Central circulation. Ql decreased with increasing G level (P = 0.001) and was lower in the supine than in the prone posture (P = 0.007). At 5 G, Ql was significantly lower in supine than in prone (Fig. 2). Heart rate (HR) was significantly higher in the prone posture at both G levels (P < 0.001). In both postures, HR increased in hypergravity (P = 0.002), and there was a tendency for interaction between posture and G (P = 0.07). Stroke volume (SV) was 87.1 ml/heartbeat in normal G (mean value of prone and supine) and decreased to 44.6 ml in hypergravity (P < 0.001), with no difference between postures (P = 0.9).

DISCUSSION

The highlight of this investigation was that DLCO and pulmonary blood flow were larger when prone than when supine in normal humans exposed to hypergravity. However, both variables were markedly reduced in hypergravity compared with normal G.

Diffusing capacity. We have previously shown in sitting subjects that hypergravity induces a gas-exchange impairment and, therefore, a reduction in DLCO (34). This is most likely a consequence of a less homogenous distribution of VA with respect to pulmonary-capillary blood volume (34). Although we are not aware of any previous determinations of DLCO in humans lying in hypergravity, we expected a hypergravity-induced reduction in DLCO, as has been previously demonstrated in sitting subjects (34). Accordingly, there was a marked reduction in DLCO at 5 G in both postures, even though DLCO was lower in the supine posture than in the prone. This finding is in concordance with our laboratory's previous finding of a more marked hypergravity-induced arterial hypoxemia in the supine posture than in the prone due to a more severe mismatch between the distribution of ventilation and perfusion in the supine posture (35). In the same study, we also reasoned that interstitial pulmonary edema and alveolar hypoventilation are not likely to have influenced our results in hypergravity (35). It has been shown that even mild exercise will open all or nearly all pulmonary vessels, including capillaries (33), due to an increase in Ql. Also, DLCO increases progressively with increasing pulmonary blood flow (33) because of a more homogenous distribution of pulmonary-capillary blood volume in relation to VA (12). By analogy, therefore, we expect the higher Ql in the prone posture at 5 G to be accompanied by a larger number of pulmonary capillaries recruited in the prone posture and, therefore, a more homogenous perfusion distribution in that posture.

We, therefore, estimate that the combination of more homogenous pulmonary blood distribution and larger alveolar gas volume (larger FRC) in the prone posture is the cause of the improved DLCO and arterial oxygenation.

As in our laboratory's previous study (35), arterial oxygen saturation was significantly more reduced by hypergravity in the supine compared with prone posture (Table 1). Our findings of less impaired arterial oxygenation, together with higher Ql in the prone posture compared with supine at 5 G, are in apparent contrast to the data obtained by Barr (4) in sitting subjects at 5–6 G. Barr (4) found that the arterial desaturation became much more pronounced when the subjects used an anti-G suit and ascribed this difference to a better maintained Ql and an associated higher degree of shunting through poorly ventilated parts of the lung. The inflation of the G suit, however, is associated not only with a centralization of blood but also with abdominal compression and a headward displacement of the diaphragm, most likely resulting in a decrease in VA. Together, the data of Barr (4) and the present data support the notion that a better maintained Ql at 5 G should be accompanied by an increased VA, in order not to result in more severe desaturation.

FRC. In the supine posture at normal G, there is a positional shift of the diaphragm caused by the weight of the abdomen pushing the diaphragm headward that results in a smaller FRC compared with that in the prone posture (21). This results in a larger VA in the prone posture, which will affect the lung function in a positive manner because, generally, there is a positive relationship between DLCO and VA (43). This phenomenon has been postulated as a possible mechanism for the beneficial effect on arterial oxygenation when
prone. However, several studies in patients with severe ALI have failed to show a positive correlation between the FRC and arterial Po2 (8, 25, 27).

The mean values of Table 1 show an almost linear relationship between VA and DLCO, but this is not to say that VA is a principal determinant of DLCO. Instead, a more detailed analysis shows a weak, if any, correlation between these parameters: R² values were 0.4–0.1 when data for VA and DLCO were correlated within 5-G prone and 5-G supine posture experiments. In addition, the volume-corrected DLCO did not differ largely from the noncorrected DLCO, as shown in Table 1, and the difference between postures at hypergravity was still present. Taken together, our results support the conclusion that the positive effect of prone positioning may only be due, to a limited degree, to a larger VA.

Because, generally, intrathoracic and pleural pressures decrease with increasing lung volume, it can be postulated that the lower FRC values in the supine vs. prone posture at both levels of G should be associated with higher pleural pressures at comparable hydrostatic levels. At the same time, the pleural pressure is the extracardiac pressure and, therefore, one important determinant of the transmural pressure in the heart during diastole. As a consequence, the larger FRC in the prone posture may be one mechanism contributing to a more favorable venous return to the heart in the prone posture.

QL, HR, and SV. From a hemodynamic point of view, it is generally agreed that there is an advantage to being recumbent compared with sitting when exposed to hypergravity, for example, during take-off of a spacecraft, due to a smaller hydrostatic pressure gradient between the heart and the head, compared with that in the sitting posture (45). However, our results demonstrate that the cardiovascular function is also severely affected while recumbent during hypergravity, as illustrated by the dramatic reduction in QL. Only a few previous measurements of QL have been made in human subjects during exposure to forward acceleration (17, 19). Nolan et al. (26) performed experiments with supine subjects, where two out of four volunteers experienced grayout at 5.8 G. In contrast, such phenomena did not occur in subjects who were studied in the launch position (17, 19), i.e., with the legs elevated and hips and knee joints in 90–100° angle. Under such conditions, no systematic change in QL was obtained at G levels up to 5 G (17, 19). Together, these results point to the importance of a hydrostatic blood column above the heart for the maintenance of venous return in hypergravity.

Wood and coworkers (37, 38, 47) studied the topographic relationships of the heart and the lungs in dogs exposed to 1 and 6–7 G in prone and supine postures, measured from biplane roentgenograms. There was a marked dorsal displacement of the heart in the supine posture in hypergravity, compared with a stable position of the heart against the anterior chest wall in the prone posture (37, 38, 47). Thus the displacement of the heart in the supine dogs must have resulted in a compression of underlying lung tissues and a stretching of the tissues between the heart and the anterior chest wall. Even though the ventral-dorsal dimensions of the dogs in these studies (37, 38, 47) were similar to those of humans, the size of the human heart is much larger than that of the dog heart in relation to the ventral-dorsal dimensions. Malbouisson et al. (18) performed a thorough analysis of computed tomography scans of the chest in normal subjects and in patients with ARDS. In normal supine subjects, the contour of the heart was shown to occupy most of the distance between the anterior chest wall and the spinal column, and, in ARDS patients, all of this distance. Therefore, there is much less margin for G-induced displacement of the heart in humans compared with the dogs studied by Wood and coworkers (37, 38, 47). Thus the negative hydrostatic gradient for venous return in the supine posture is likely to have increased in proportion to G, as would the corresponding positive gradient in the prone posture.

The higher FRC in prone (see above) may also have contributed to more favorable conditions for diastolic filling. It must be noted, however, that the increased intravascular vertical hydrostatic pressure gradients in hypergravity are associated with proportional increases in the corresponding gradients in extravascular tissues. Also, the pericardial sac will limit the extent to which the diastolic heart can expand in response to an increased diastolic filling pressure. In a thorough analysis of these factors in dogs exposed to 1 and 6 G, Wood (47) showed that the transmural (right atrial minus pericardial) pressure did not differ between prone and supine postures at 1 G, and at 6 G the transmural pressure was only 2 cmH₂O higher in prone than in the supine posture.

In summary, therefore, we propose that an improved preload in the prone posture compared with supine in hypergravity accounts for the better preservation of QL.

In the present experiments, SV shows no difference between postures, but there is a significantly higher HR in the prone posture at both gravitational conditions, in keeping with our previous observations (35). There are two reasons why we estimate that this difference in HR is not due to a difference in reaction to emotional stress with an associated increased sympathetic nerve activity. First, there is a difference in HR between postures at normal G, but most likely no difference in the level of stress. Second, almost all subjects complained about it being more difficult to breathe in the supine posture at 5 G because of substernal pain. The substernal pain and larger degree of arterial desaturation when supine (35) may have produced secondary alterations of the autonomic outflow, but, if so, they were associated with lower rather than a higher HR in the supine posture.

A second possibility is that this difference in HR might be due to a reflex response to a lower arterial blood pressure in the prone posture. However, in a previous study, our laboratory (35) showed that, although the mean arterial pressure (MAP) increased in hypergravity in lying subjects, there was no difference...
between the supine and the prone posture. Our previous finding of an increase in MAP of 21% in supine subjects during hypergravity compared with normal G is in agreement with the 17% increase observed by Wood et al. (48). In our MAP measurements, we did not take into account that hypergravity could have affected the position of the heart in the thorax (46) and the thoracic shape, and it probably also compressed the padding on which the subject was lying. However, we attempted to assess the potential impact of these confounding factors, and we concluded that any small differences in MAP between postures, if measured with more precise hydrostatic references, probably would be even less marked at hypergravity. In short, therefore, we do not consider it likely that baroreflex inputs from MAP at heart level influenced the HR difference between postures.

A third possible mechanism could be a tachycardic effect from stimulation of cardiopulmonary mechanoreceptors. The presence of such a reflex was suggested by Bainbridge (2) on the basis of acute atrial distension in an animal model. Recent studies have revived the notion of a functionally important Bainbridge reflex in humans (3, 28). Put together, the present results, data from transmural atrial pressures in prone and supine dogs (47), and the recent human studies in favor of a Bainbridge reflex (3, 28) may explain the relative tachycardia in the prone posture at hypergravity, but not readily at 1 G where transmural right atrial pressures may not differ between the prone and supine posture.

It is difficult to interpret the similarity of SVs between the two postures at 5 G. One possibility would be that there were similar limitations of SV in the two postures due to hypergravity-induced cardiac deformation and/or vascular engorgement of surrounding lung tissue. Another possibility is that there were primary differences in venous return, which determined Qt, and that SV was set secondarily by HR. We consider the latter alternative more likely, because the position of the heart relative to the inferior caval vein differs widely between the two postures. However, topographical data from humans in hypergravity will be required for a better understanding of factors determining Qt and SV under the present hypergravity conditions.

Hypergravity as a model of acute lung insufficiency. Experimental data clearly demonstrate the presence of increased hydrostatic pressure differences down the lung, both in ARDS (14) and in hypergravity (37, 38). Another commonality is the effect of increased cardiac weight on underlying lung tissue. Malbouisson et al. (18), studying supine ARDS patients with computed tomography scans, found a marked increase in cardiac mass compared with that in normal supine subjects, and an associated increase in the pressure exerted by the heart on the right and left lower lobes of the lung. They also estimated the fractional content of gas in the lung tissue below the heart and found that it was more reduced in patients than in normal subjects compared with lung tissue located outside the heart limits on both sides. The observation by Alexander et al. (1) that supine patients with cardiomegaly have a reduction in ventilation in the left lower lobe, which is reversed when changing to the prone posture, lends further support to the notion that an increased weight of the heart compresses lung tissue in the supine but not in the prone posture.

The finding that the negative impact of these increased pressure gradients is ameliorated by prone posture compared with supine, both in patients with ARDS and in normal subjects in hypergravity, however, does not necessarily mean that hypergravity models all aspects of ARDS. An important difference between the hypergravity model and the patients is the distribution of fluid between intra- and extravascular compartments in the lung. In the present hypergravity model, there was no evidence of lung tissue or alveolar edema, and it must, therefore, be assumed that the increased radiological density always observed in dependent parts of the lungs in hypergravity (15) is entirely caused by vascular engorgement. The question arises whether an intravascular expansion at the expense of the alveolar gas volume is less of an impediment to gas exchange between the gas and the blood than if the space-occupying fluid accumulation is in the interstitial tissue and within the alveolar space, as has been shown in ARDS (14). Data from previous hypergravity experiments (34) suggest that there is sequestration of blood in dependent lung parts but that this increased amount of lung-capillary blood, nevertheless, is associated with a reduced diffusing capacity. Thus, apart from being rapidly reversible, the sequestration of blood in the dependent vascular compartments during hypergravity appears to play a role similar to that of extravascular fluid accumulation in terms of impeding gas exchange. However, determinations of the topographical distributions of blood and gas in the lungs during hypergravity are required for more complete understanding of the value of hypergravity as a model of ARDS and ALI.

In summary, we found lower DLCO values in the supine posture compared with prone in resting humans during hypergravity, which supports our previous observations of larger alveoloarterial O2 differences in the supine posture under identical conditions. We speculate that this difference in cardiopulmonary function between postures is of little consequence in healthy subjects at normal G but appears to make a difference when the function of the lung as a gas exchanger is impaired, as in patients with acute lung insufficiency or when exposed to hypergravity.

We acknowledge the dedication of the subjects and the crew at the human centrifuge, Karolinska Institutet, Sweden, in particular the excellent technical support by Barbro Bergström and Bertil Lindborg.

This study was supported by the Swedish National Space Board, Fraenckel’s Fund for Medical Research, The Laerdahl Foundation for Acute Medicine, and the Swedish Research Council (project numbers 05020 and 10401).

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