Exposure to LBNP results in body fluid shift to lower extremities similarly as under influence of orthostatic stress. In susceptible persons it leads to syncope. For better understanding why certain individuals are more susceptible to orthostatic challenges it seemed necessary to collect more data on hemodynamic and neuroendocrine adjustments occurring before onset of presyncopal symptoms. Accordingly, in this study heart rate (HR), blood pressure (BP), stroke volume (SV), cardiac output (CO), hematocrit, plasma catecholamines, adrenomedullin, ACTH and plasma renin activity (PRA) were measured in 24 healthy men during graded LBNP (-15, -30 and -50 mmHg). Thirteen subjects completed the test (HT group) whereas 11 had presyncope signs or symptoms at -30 mmHg or at the beginning of -50 mmHg (LT group). Comparison of these groups showed that LT subjects had lower baseline total peripheral resistance and higher plasma adrenomedullin. During LBNP plasma catecholamine and PRA increases were even greater in LT than in HT group while plasma adrenomedullin elevations were similar in both groups. Plasma ACTH increased only in LT group following presyncope symptoms. Low tolerant group showed more rapid decline of SV and CO than HT subjects from the beginning of LBNP. It is suggested that measurements of SV at the level of LBNP which did not evoke any adverse symptoms may be of predictive value for lower orthostatic tolerance.

Key words: orthostatic tolerance, hemodynamic response to LBNP, catecholamines, adrenomedullin, plasma renin activity, ACTH
INTRODUCTION

Application of lower body negative pressure (LBNP) causes a shift of fluid from the upper body to lower extremities resulting in central hypovolemia that eventually may lead to reduction of cerebral blood flow and syncope. Since the first study describing this procedure (1) LBNP has been applied for evaluation of the compensatory ability of the cardiovascular control systems. This research tool has been used in space and aviation medicine to study orthostatic intolerance after space flight and effects of microgravity induced by bed rest or vertical acceleration in aircrafts (2). Recently Cooke et al. (3) suggested that LBNP may be also used to identify persons who will progress to shock under condition of hemorrhagic trauma.

Several studies demonstrated that certain healthy individuals well tolerate central hypovolemia induced by LBNP or orthostatic stress while others quickly develop presyncope symptoms (4 - 9). The mechanisms protecting against circulatory shock include multiple interacting systems of cardiovascular control. Intolerance of LBNP may be connected with abnormally large decrease in central blood volume due to high compliance of capacitance blood vessels in lower extremities, inadequate reflex and mechanical nonautonomic mechanisms regulating stroke volume (SV) and heart rate (HR) as well as inadequate function of neuroendocrine mechanisms responsible for peripheral vasoconstriction. It was shown that LBNP tolerance is lower in tall than in short persons (9), in women than in men (6) and in endurance athletes than in untrained subjects (10). The authors suggested that in women and athletes orthostatic intolerance is associated with decreased cardiac filling rather than reduced responsiveness of vascular resistance. According to them gender and fitness related differences in LBNP tolerance may depend on differences in cardiac mechanics and Frank-Starling relationship.

Inverse relationship between SV decrease and muscle sympathetic nerve activity (MSNA) during LBNP was reported by Convertino et al. (4). It supported the hypothesis that information about reduction of cardiac chamber size caused by decreased cardiac filling is transmitted to and then integrated in the central nervous system, resulting in peripheral sympathetic activation (11). Reduced plasma norepinephrine response to standing in astronauts exhibiting hemodynamic collapse suggested that attenuated sympathetic activity may be the cause of orthostatic intolerance (12). Convertino et al. (4) who submitted healthy male volunteers to LBNP reported that in the one subject who experienced collapse there was an abrupt complete withdrawal of MSNA. However, in this individual and in other subjects before the presyncope symptoms MSNA increased even more than in those who were able to complete the test without incidents. In spite of hyperadrenergic response in the subjects with low LBNP tolerance peripheral resistance was lower than in high tolerant group suggesting that in the former vascular responsiveness to norepinephrine might be reduced.
Insufficient increase in peripheral resistance may be also related to inadequate release of some other vasoactive substances. It was reported that orthostatic stress induced increases in plasma concentrations of vasopressin (5, 7) angiotensin II (7), ACTH (8), adrenomedullin (13), plasma renin activity (5, 7, 8) and a decrease in atrial natriuretic peptide (8).

Greenleaf et al. (7) comparing healthy men with high and low LBNP tolerance demonstrated that the latter had lower baseline plasma renin activity (PRA) with attenuated PRA and plasma angiotensin II responses to LBNP. In the subjects with low LBNP tolerance the increase of plasma vasopressin was more pronounced while elevation of plasma catecholamines was similar to that found in subjects with high tolerance. In either group LBNP did not change plasma concentration of endothelin-1. Since angiotensin II is one of the most powerful vasoconstrictor, the data may indicate that low tolerance of LBNP is associated with inadequate activation of the renin-angiotensin system. The authors suggested that baseline PRA may have predictive value for orthostatic intolerance. Similar results concerning PRA and catecholamines were obtained by Convertino and Sather (5). However, these authors found lower vasopressin response to LBNP in the subjects who developed presyncope than in those who did not. On the other hand, in the earlier study Harrison et al. (14) demonstrated higher increases of PRA and vasopressin in subjects who exhibited low tolerance of orthostatic stress.

Because of several discrepancies and limited number of subjects in the studies reported so far, pathophysiology of orthostatic intolerance in young apparently healthy subjects is not fully recognized. In order to better understand why certain individuals are more susceptible to orthostatic challenges it seemed necessary to collect more data on hemodynamic and neuroendocrine adjustment occurring before onset of presyncopal symptoms. Thus, in the present study the relatively homogenous group of 24 young male subjects were submitted to LBNP test. It allowed to identify a representative number of 11 individuals with low LBNP tolerance. Their cardiovascular and endocrine changes induced by LBNP were compared with those in 13 subjects who did not show the presyncopal symptoms or signs. Among cardiovascular indices heart rate (HR), blood pressure (BP) and stroke volume (SV), preejection period (PEP) and ejection time (ET) were determined. The neuroendocrine variables included plasma concentrations of norepinephrine (NE), epinephrine (E), ACTH, adrenomedullin (ADM) and plasma renin activity (PRA).

**MATERIALS AND METHODS**

**Subjects.**

Twenty four healthy male students of the Military Academy (age: 20.8± (SD) 0.9 yr, body mass: 74.2±(SD)7.1 kg, height: 176.9±(SD)4.3 cm, maximal oxygen uptake: 47 ± (SD)4 ml·kg⁻¹·min⁻¹)
volunteered to this study after giving informed consent. All lived in the Students' Hostel, had similar daily activities and the same controlled diet. The study protocol was approved by the Ethical Committee of the Medical Academy in Poznan, Poland.

**Study protocol.**

Three days before starting the LBNP testing the subjects were submitted to the incremental exercise test performed on bicycle ergometer until volitional exhaustion in order to determine their maximal oxygen uptake (VO$_2$\text{max}). In the evening before LBNP test the subjects reported to the laboratory where they spent the night. The tests were performed in the morning, after an overnight fast. To avoid any orthostatic effects the subjects were carried on the stretcher in the supine position and laid down on the LBNP table. Then, the LBNP chamber was sealed at the level of iliac crest. During the whole LBNP test the subjects remained supine. Thirty min after inserting catheter to the antecubital vein and instrumentation two baseline circulatory measurements were made and blood sample for hormone and hematocrit (HTC) determinations was taken at ambient pressure. Then, the subjects were submitted to serial LBNP: 10 min at -15 mmHg, 10 min at -30 mmHg and 10 min at -50 mmHg or until onset of presyncopal signs or symptoms, and 10 min of the recovery period at ambient pressure. The presyncope symptoms and signs include: lightheadedness, nausea, sweating, narrowing of vision and rapid drop of systolic blood pressure by more than 20 mmHg or bradycardia. Before and every 3 min during LBNP and the recovery period blood pressure (BP), heart rate (HR), stroke volume (SV), cardiac output (CO), ejection (ET) and pre-ejection time (PEP) were measured. Blood samples for epinephrine (E), norepinephrine (NE) and ACTH concentrations and plasma renin activity (PRA) were taken at the end of the second (-30mmHg), the third (-50mmHg) stage of LBNP or immediately after onset of presyncope symptoms as well as at the end of the recovery period. Hematocrit and adrenomedullin (ADM) concentration were determined only before and at the end of final LBNP stage.

**Methods.**

For LBNP the chamber with the pressure control system was used (ITAM, Zabrze, Poland). It allows the changes in LBNP within approx. 15 s. The integrated HR was monitored and recorded by the Sport Tester (PE 3000, Polar Electro, Finland). Blood pressure (BP) was measured on brachial artery by the automatic oscillometric sphignomanometer. Stroke volume (SV) and cardiac output (CO) were measured by impedance cardiography (ICG) using a monitoring device designed in the Medical Research Centre, Polish Academy of Sciences by Cybulski et al. (15). The measurement is based on the tetrapolar technique: the sinusoidal alternating current (95 kHz) is applied via the pair of electrodes placed on the chest, the voltage signal is collected from other electrodes and demodulated. The ECG and the first derivate of the impedance signal are sampled at the rate of 200 Hz. The system allows for the off-line, beat-to-beat automatic evaluation of SV, HR, ET and PEP. Cardiac output (CO) was calculated as a product of SV and HR. Validity of SV measurements was determined using echocardiography (r=0.90, n=21, p<0.001) (16). Mean blood pressure was calculated as diastolic BP plus 0.33 of the difference between systolic and diastolic BP. The total peripheral resistance (TPR) was calculated dividing CO by mean BP.

The blood samples were taken to the chilled polyethylene tubes. For catecholamine determination the tubes contained EGTA and reduced glutathione while for other hormones the tubes with EDTA and aprotinin (Trasylol, 500 KIU/ml blood) were used. All samples were centrifuged within 30 min at 3000 rpm at 4°C, and stored at -70°C until further processed.

Plasma [E] and [NE] were measured using high pressure liquid chromatography. Other hormones were determined by radioimmunoassay using CIS bio International (France) kits for
plasma [ACTH] and [ANP], Phoenix Laboratories (Belmont, CA, USA) reagent set for [ADM] and Immunotech, Angiotensin I kit (Prague, Czech Republic) for PRA.

Statistics.

The data are presented as means with standard errors (SEM) unless otherwise stated. Two way analysis of variance for repeated measures was used for statistical evaluation of the results. The two factors were the subject groups with high and low tolerance of LBNP and repeated measures of plasma hormones and circulatory indices. When significant F value was obtained a paired Student’s t test was used to evaluate the effect of LBNP. A comparison between groups was made using a nonparametric Whitney-Mann test. P<0.05 was accepted as the level of significance.

RESULTS

LBNP tolerance.

Thirteen subjects completed LBNP test while 11 subjects exhibited presyncopal symptoms at -30 (6 subjects) or -50 mmHg (5 subjects). Basing on this the subjects were assigned to two groups: those who tolerated 10 min of LBNP at -50 (HT) and those with lower LBNP tolerance (LT). There was no significant differences between HT and LT groups in body mass, height, body mass index (BMI) and VO$_2$max (Table 1). Baseline HTC was similar in LT and HT subjects (36.3 ± 1.3 vs 37.0 ± 1.1%). It increased during LBNP to 39.3 ± 1.4% (p<0.05) in LT group and to 39.0 ± 1.5% (p<0.01). The calculated plasma volume decreases were 7.4 ± 2.9 and 7.3± 1.8%, respectively.

Cardiovascular responses to LBNP (Fig1-3).

Analysis of variance of baseline values revealed that LT subjects had higher HR, systolic BP, SV (p< 0.05), CO with lower TPR, and shorter duration of PEP. Baseline diastolic and mean BP and duration of ET did not differ between the groups.

Table 1. Basic anthropometric characteristics and maximal oxygen uptake (VO$_2$max) of subjects with high- and low tolerance of LBNP (values are means ± SD)

<table>
<thead>
<tr>
<th></th>
<th>High tolerance</th>
<th>Low tolerance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>Age (years)</td>
<td>20.6 ± 0.7</td>
<td>21.1± 1.1</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>74.5 ± 7.2</td>
<td>73.8 ± 7.3</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>177.5 ± 4.9</td>
<td>176.1 ± 3.6</td>
</tr>
<tr>
<td>BMI (kg·m$^{-2}$)</td>
<td>23.6 ± 1.5</td>
<td>23.8 ± 2.3</td>
</tr>
<tr>
<td>VO$_2$max (L·min$^{-1}$·kg$^{-1}$)</td>
<td>46 ± 4</td>
<td>48 ± 3</td>
</tr>
</tbody>
</table>
As it is shown on Fig 1, HR increased significantly during LBNP in both groups. In the recovery period it declined towards initial values. In LT group at the end of the recovery HR was even lower than before LBNP. ANOVA showed significantly higher HR values in LT than in HT group and significant interaction of the LBNP effects and group affiliation. Post hoc comparisons revealed significant differences between groups starting from the last minute of the first stage of LBNP (-15 mmHg). Systolic BP in LT group decreased throughout the
whole period of LBNP. At termination of the test systolic BP in this group was 95 ± 6 mmHg and was lower than pre-LBNP value by 35 ± 6 mmHg. In HT group the decline of systolic BP reached significance at -50 mmHg. At termination of the test systolic BP in this group was 110 ± 2 mmHg and was lower than the pre-LBNP value by 10 ± 3 mmHg. The diastolic BP was not affected by LBNP in HT group while in LT there was a decrease when presyncopal symptoms occurred. The mean value at this time was 59 ± 4 mmHg and it was lower from the initial
level by 14 ± 3 mmHg. During the recovery period both systolic and diastolic BP returned to initial values or even exceed them in HT group. ANOVA did not show differences between groups either in systolic or in diastolic BP values but the time courses of BP changes during LBNP assessed by interactions of two factors were significantly different.

Analysis of basic impedance cardiography signal (Z0) showed that Z0 values increased progressively during LBNP in both groups (p<0.001) but there were no significant differences between the groups either in Z0 values or the time courses in their changes. Stroke volume decreased during LBNP in both groups (Fig.2). In LT group this decrease was significant from the beginning of the LBNP test while in HT subjects significant decreases were found starting from -30 mmHg. During recovery SV increased but did reach the initial value in either group. ANOVA did not show differences in SV values between groups but the interaction between the group affiliation and LBNP effects was significant. Cardiac output (CO) decreased in both groups. During the recovery period CO increased but did not attain the pre-LBNP values. ANOVA showed significantly higher CO values in LT than in HT group and highly significant interaction
between the group affiliation and LBNP effects. Post hoc comparisons of CO values between groups showed significant differences only at -15 mmHg and during the recovery period.

Total peripheral resistance (TPR) increased significantly during LBNP in both groups, however, in HT subjects significant differences were found only at -50 mmHg while in LT group increases reached statistical significance already at the first stage of LBNP (Fig.2). In both groups during the recovery TPR decreased but was still higher than before LBNP. ANOVA revealed significant difference between groups without interaction between the effects of group affiliation and LBNP. The post hoc analysis showed significant differences in control period, at -15 mmHg and during the recovery period (Fig.2).

Changes in the duration of systolic time intervals are presented in Fig.3. LBNP caused a significant progressive increase in PEP duration and opposite change in ET in both groups. During the recovery period ET attained the initial levels while the duration of PEP was still longer than before LBNP. ANOVA did not show significant differences between groups in the duration of both time intervals, however the time courses of changes in PEP and ET during LBNP assessed by interaction between the two factors were steeper in LT than in ET group (p<0.001 for PEP and p<0.01 for ET).

**Neurohormonal responses to LBNP (Fig 4-5).**

There were no significant differences between groups in the baseline values of plasma catecholamine and ACTH concentrations and plasma rennin activity (PRA) while plasma level of adrenomedullin (ADM) was significantly higher in LT than in HT group.

During LBNP both norepinephrine (NE) and epinephrine (E) concentrations increased significantly in both groups (Fig.4). During the recovery period plasma concentrations of NE returned to initial values, whereas plasma E was still elevated. Comparisons between groups showed that at -30 and -50 mmHg plasma NE was significantly higher in LT than in HT group. Similar tendency was noted in plasma E. An increase in plasma NE during the whole period of LBNP tended to be greater in LT than in HT group (2.15 ± 0.60 nmol/L vs. 0.87 ± 0.31 nmol/L, p=0.060). The differences between groups in plasma E increases were insignificant (0.48 ± 0.19 nmol/L and 0.24 ± 0.09 nmol/L in LT and HT group, respectively, p=0.250).

Plasma rennin activity rose significantly during LBNP and remained still elevated after 10 min of recovery in both groups (Fig.4). At -30 mmHg PRA was significantly higher in LT than in HT group. The increases in PRA during the whole period of LBNP were similar in HT and LT groups.

Plasma ACTH level did not change significantly during the test in HT group while in LT group it tended to increase during LBNP and it was significantly elevated at the end of recovery period (Fig.4). In spite of reduced number of
Fig. 4. Changes in plasma norepinephrine, epinephrine, plasma renin activity and ACTH during LBNP test in the subjects with low (LT) and high (HT) LBNP tolerance. Asterisks denote significant differences between groups: *p<0.05, **p<0.01, ***p<0.001; crosses denote significant differences from baseline values: †p<0.05, ‡p<0.01, §p<0.001
subjects from LT group who withstood at least a few min at -50mmHg the difference in ACTH concentration between groups was significant at this stage of LBNP. After 10 min of recovery the difference between groups became highly significant.

Plasma ADM concentration increased significantly during LBNP in both groups (Fig.5). The values obtained at the end of LBNP and the LBNP-induced increases did not differ between LT and HT groups.

DISCUSSION

Comparison of baseline cardiovascular indices and hemodynamic responses to LBNP in the subjects with low and high LBNP tolerance

The subjects from LT group had greater baseline values of HR with shorter duration of PEP, greater SV, CO and systolic BP than those in HT subjects but their TPR was lower. None of these indices seemed to have a predictive value for orthostatic intolerance except perhaps lower peripheral resistance. The initial values of HTC and a decrease in plasma volume during LBNP were similar in both groups.

The most pronounced differences between HT and LT groups were in the time-course of hemodynamic changes induced by LBNP. Lower tolerance was associated with rapid decreases in SV and CO which were significant already at -15 mmHg when in the subjects with high tolerance these variables were not altered significantly. This is in agreement with the suggestion of Levine et al. (10) and Fu et al. (6) that inadequate regulation of SV can be responsible for LBNP intolerance. However, it should be noted that at -30 or -50 mmHg SV and CO values in LT subjects did not differ from those in HT group. In parallel with the changes in SV there was a decrease in the ejection time which is in line with the data reported by Turski et al. (17). Heart rate at -15 mmHg was similar in both

![Fig.5. Changes in plasma adrenomedullin before and after LBNP in the subjects with low (LT) and high (HT) LBNP tolerance. Asterisks denote significant differences between groups: *p<0.05; crosses denote significant differences from baseline values: ++p<0.01, +++p<0.001.](image)
groups but at -30 mmHg it reached significantly higher value in LT than in HT group. This was probably due to greater arterial baroreceptor activation caused by reduction of SV.

Total peripheral resistance in LT group at -15 mmHg increased slightly but it was still lower than in HT subjects who showed a significant increase in TPR only at -50 mmHg. In the intolerant subjects at -30 mmHg there was further increase in TPR but shortly before the presyncope a tendency towards lowering of resistance was noted. Although systolic blood pressure declined from the beginning to the end of LBNP in LT subjects the differences between groups in systolic BP were insignificant until occurring of presyncope signs or symptoms.

Analysis of cardiovascular alterations induced by early stage of LBNP which was well tolerated by all subjects suggests that the high rate of decline of SV and CO with relatively low TPR may be considered as predictors of LBNP intolerance in healthy young men. Heart rate and blood pressure during initial LBNP did not discriminate the subjects with higher and lower tolerance which remains in agreement with the study of Greenleaf et al. (7) and Simonson et al. (18).

Comparison of baseline neuroendocrine indices and their responses to LBNP in the subjects with low and high LBNP tolerance

The new finding of the present investigation is that the subjects with low LBNP tolerance had significantly higher initial plasma adrenomedullin (ADM) concentration. This peptide, secreted by adrenal medulla, vascular endothelial and smooth muscle cells, cardiac myocytes and kidneys is considered to be a potent vasodilation factor (for rev. see 19). It seems likely, therefore, that the higher baseline plasma ADM concentration in the subjects from LT group may contribute to their lower peripheral resistance.

Initial plasma concentrations of catecholamines and ACTH as well plasma renin activity (PRA) did not differ between the subjects with lower and higher LBNP tolerance. The present results are in agreement with the previous data showing that baseline plasma catecholamines (5, 7, 8) , ACTH (8) and PRA (5) do not discriminate subjects with low and high orthostatic tolerance. However, our data on basal PRA did not confirm the finding of Greenleaf et al. (7) who demonstrated that low baseline PRA is associated with LBNP intolerance in young healthy men. In should be noted that in their study dietary NaCl intake during three days before LBNP was the same in high and low tolerant subjects while in our study the sodium intake was not controlled although the subjects consumed similar diet in the canteen. Some possible differences in sodium intake among our subjects might be a cause of discrepancy between the present study and that of Greenleaf et al. (7). Besides, there are differences in the study protocol. Our subjects remained in the supine position for approx. 8 hours before LBNP whilst those examined by Greenleaf et al. were supine for 1h , then sitting
for 1 h and again supine for 45 min before the test. This procedure modified plasma volume and presumably PRA already before LBNP.

During LBNP significant increases were found in plasma catecholamines, renin activity and adrenomedullin in both groups. Plasma norepinephrine (NE) concentration was significantly higher in LT group than in HT group at -30 and -50 mmHg. Thus, the low LBNP tolerance cannot be attributed to inadequate sympathetic activation before onset of syncope. This is consistent with the results obtained by other authors who did not find lower plasma NE responses to LBNP (5,7) or head-up tilt (8) in presyncopal subjects. Moreover, Convertino et al. (4) demonstrated greater increases of MSNA at each level of LBNP before cardiovascular collapse in low than in high tolerant subjects.

Comparison of plasma epinephrine (E) concentrations between LT and HT groups did not show significant differences similarly as in the previous studies (7, 8). It should be noted, however, that in LT subjects a tendency towards greater E values than in HT group was noted already at -30 mmHg.

In opposite to the results obtained by Greenleaf et al. (7) and Convertino and Sather (5), we did not find attenuated response of PRA to LBNP in the subjects who exhibited presyncopal symptoms or signs. Moreover, at -30 mmHg in these subjects PRA was slightly but significantly higher from that in the HT group. Thus, it seems unlikely that in our subjects depressed renin-angiotensin system activity was responsible for low LBNP tolerance.

The marked difference between HT and LT groups was demonstrated in plasma ACTH response. The increase in this hormone concentration occurred only in LT group at -50 mmHg and during the recovery period. Similar results were obtained previously by Jardine et al. (8) who compared ACTH responses to head-up tilt in the patients with low and high orthostatic tolerance. Since in the HT subjects there were no changes in ACTH level and in the LT group the increase occurred at the end of the LBNP test and during the recovery period it seems likely that the activation of hypothalamic pituitary system is related to the impending cardiovascular collapse.

The LBNP-induced increases in ADM were highly significant and similar in both groups. The role of this hormone in the cardiovascular adjustment to central hypovolemia may include stimulation of cardiac contractility and counteracting the vasoconstrictory influences of catecholamines and angiotensin. Thus, ADM may contribute to the fine control of peripheral resistance. In the available literature only three papers addressed to the effect of orthostatic stress on plasma ADM were found. Rössler et al. (13) demonstrated a rapid increase whereas Mallamaci et al. (20) and Nishikimi et al. (21) did not find any changes in ADM level during head-up tiling in healthy men.

In summary, the present study showed that low LBNP tolerance in young healthy men was associated with relatively low total peripheral vascular resistance under basal conditions and a rapid decline of SV, CO and ET already at the level of LBNP which does not evoke any adverse symptoms. This was
accompanied by even greater increase in plasma catecholamine concentrations and plasma renin activity than in the subjects with high tolerance. The impending cardiovascular collapse was followed by a marked increase in ACTH release. The subjects with low LBNP tolerance had slightly higher initial adrenomedullin concentration then those from high tolerance group. However, the increases in this peptide concentration during LBNP were similar in these two groups.

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**REFERENCES**


17. Turski BK, Gembicka-Kuzak DM, Debiński WB. Compensatory reactions during lower body negative pressure (LBNP), head-up tilt (HUT) and +Gz tolerance. *J Gravitational Physiol* 1996; 3; 97 - 98.


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